

# Brain Dynamics

by Justo Gonzalo  
Volume 2

Edited and translated from Spanish by Isabel Gonzalo Fonrodona

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# BRAIN DYNAMICS

The brain activity according to the dynamic conditions  
of nervous excitability

by Justo Gonzalo

Volume 2

Edited and translated by Isabel Gonzalo Fonrodona

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*This edition is dedicated to the memory of  
my mother Ana María Fonrodona Masuet*

## Preface

This volume is the English translation of Volume 2 of the book *Dinámica Cerebral* written in Spanish by Justo Gonzalo Rodríguez-Leal (Barcelona 1910 – Madrid 1986), first published in 1950. The volume, devoted to tactile functions, is the continuation of Volume 1 (on general aspects and visual functions)<sup>1</sup> to which it refers continuously.

The interest of the research described here lies, as in Volume 1, in the fact that it is surprisingly of current interest, apart from its undoubted historical interest. Some aspects were ahead of discoveries that were made later. It is remarkable that some of the phenomena exposed are still unknown and that the proposed functional dynamic unit of the cortex is closely related to current trends in the study of the brain.

The author, after specialization in neurology and brain pathology in Austria and Germany (1933-35) developed a research on the human cerebral cortex. The research here presented (as that of Volume 1) was supported by the Cajal Institute at the Spanish National Research Council in Madrid. More data on the author's research are easily accessible in an open digital support<sup>2</sup>.

This volume deals with tactile functions and elaborates on concepts introduced in Volume 1. The tactile phenomenology in cases of *central syndrome* is described. This syndrome, already studied in Volume 1, is the result of a unilateral lesion in an association area in the left parieto-occipital cortex, equidistant from the visual, tactile and auditory primary areas. It consists of a multisensory alteration (visual, tactile, auditory) although the lesion does not involve specific areas, all functions being affected, from simple excitability to more complex functions, bilaterally and symmetrically. In particular, the striking phenomenon in which the visual image is tilted or nearly inverted (see Vol. 1), is now extended to the phenomenon of localization of stimuli in the tactile system. Inversion in tactile space is studied in detail in cases of central syndrome, being generalized to all sensory systems of a spatial nature, once confirmed in the auditory system.

Similarly to what happens in vision, the tactile phenomena in the mentioned syndrome have a dynamic character since the disorders vary with the intensity of the stimulus and with the facilitation by other stimulus. The phenomenon of facilitation by muscular effort is particularly noticeable. The greater the deficit in brain excitability (due to the lesion), the greater the effect of facilitation. In the process of tactile localization of a stimulus, up to five phases are distinguished, from simple sensation to specific localization (passing through inversion), as stimulation increases. This process is described as a spiral development of the organization of the sensory field (tactile and also visual). As in vision, a continuity is found between the various functions that appear according to physiological requirements. Likewise, a continuity is established between sensory functions and gnosis, both being based on the same physiological laws. The

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<sup>1</sup> The English translation of Vol. 1 is available in Open Access at: <https://eprints.ucm.es/id/eprint/63730/>

<sup>2</sup> [https://en.wikipedia.org/wiki/Justo\\_Gonzalo](https://en.wikipedia.org/wiki/Justo_Gonzalo)

schema function is studied in detail and considered in varying degrees according to the somatic model, postural model and praxis model.

A reference case in this research is the famous Schneider case of Goldstein and Gelb studied in 1918 and 1919, which deserves publications even at present, and which the author interpreted under the central syndrome. This syndrome is also related to Gerstmann's syndrome. In subsequent research, the author found 35 cases that also fit the central syndrome. As the author pointed out, the scarcity of cases of central syndrome is not due to their exceptionality, but rather to the fact that they remain hidden due to the difficulty in the examination of these types of patients.

In a later publication<sup>3</sup> the author exposed a model based on functional gradients through the cortex, according to which, its specificity is distributed in a continuous gradation, and in agreement with a continuous transition between the central syndrome and other cortical syndromes. The author continued to develop a functional brain model applying the principle of similarity of a dynamic system to the central syndrome, the latter being understood as a change of scale in nervous excitability with respect to a normal individual. This concept is already introduced in Vol. 1 as well as in this Vol. 2. Thus, allometric relationships between different sensory qualities were found. These last aspects are briefly presented in the Spanish facsimile edition (Supplement II), also exposed more recently by other authors<sup>4</sup> placing them in a current context and including new developments involving power laws of perception based on clinical data from this book.

This research was later echoed in the field of cybernetics and artificial intelligence.<sup>5</sup> It is in this context that the 'Red Temática en Tecnologías de Computación Artificial/Natural' (RTNAC) together with the University of Santiago de Compostela, published in 2010 a Spanish facsimile edition (whose free online version maintains a significant rate of visits since its publication.)

The present English translation aims to expand the dissemination of this pioneering research, in the hope that the inquiring minds of scientists will appreciate the originality and profoundness of this research. Obviously, the text and references should be understood in the context of the time this volume was written, particularly the expressions of a temporal character. The text parts in small print, as in the original, include experimental details and supplementary data. The text in the figures has been translated while maintaining the original figures even if some of them are not of high quality.

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<sup>3</sup> Gonzalo J. (1952), *Trabajos del Instituto Cajal de Investigaciones Biológicas*, Vol. **XLIV**: 95-157. Included as Supplement I in the Spanish facsimile edition "Dinámica Cerebral" of 2010. English translation Open Access in the repository of the Complutense University of Madrid: <https://eprints.ucm.es/30931/>

<sup>4</sup> Gonzalo Fonrodona I., Porrás M. A. (2014), Nervous excitability dynamics in a multisensory syndrome and its similitude with normals. Scaling Laws, in: Costa A., Villalba E. (Eds.) *Horizons in Neuroscience*, Vol. **13**: Chap.10: 161–189. Open Access at: <https://doi.org/10.48550/arXiv.2006.01666> and also at: [https://novapublishers.com/wp-content/uploads/2019/06/978-1-62948-426-6\\_ch10.pdf](https://novapublishers.com/wp-content/uploads/2019/06/978-1-62948-426-6_ch10.pdf)

<sup>5</sup> For example: Delgado A.E. (1978), "Modelos neurocibernéticos de Dinámica Cerebral", PhD thesis, Polytechnic University of Madrid. Mira J., Delgado A.E., Moreno-Díaz R. (1987), The fuzzy paradigm for knowledge representation in cerebral dynamics, *Fuzzy Sets and Systems*, **23**: 315–30. Mira J., Delgado A.E. (2003), Neural modeling in cerebral dynamics, *Biosystems*, **71**: 133–44.

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## *Sensory dynamics. Tactile functions*

The significance of touch is understandable since, for many authors and since ancient times, it has been the primary and fundamental sense.

The similarity between tactile and visual functions is very pronounced; not in vain vision has been referred to as touch at a distance. Thus, we shall find a striking parallelism between the physiological organization of touch and vision in issues such as general excitability, sensation, space and forms, orientation and finally schema.

However, despite the apparent uniformity of the tactile constituent elements, there is a functional complexity perhaps absent in the case of vision. Especially tactile space is a broad field of investigation, and any function developed to some extent must ultimately refer to it, from rudimentary localization to the body schema, going through the remarkable phenomena of spatial inversion.

Tactile functions have always been studied by neurologists given their importance in neurological diagnosis, and recently, after some partial attempts at renewal, these functions seemed to be an exhausted field for research. However, in the case of touch, the dynamic analysis has once again revealed a multitude of unknown phenomena in all kinds of functions, phenomena that can easily be interpreted from a strictly physiological point of view. We therefore believe that this means a profound transformation, in data and theory, in the knowledge of touch sense.

# General excitability

## 17 Electrical excitability

### 17.1 Strength-duration curve. Hoorweg's law

The determination of tactile electrical excitability has been carried out in identical technical conditions as for the retina, and we refer back to it for methodology. The results are likewise of the same type as for the retina in our two patients M and T.

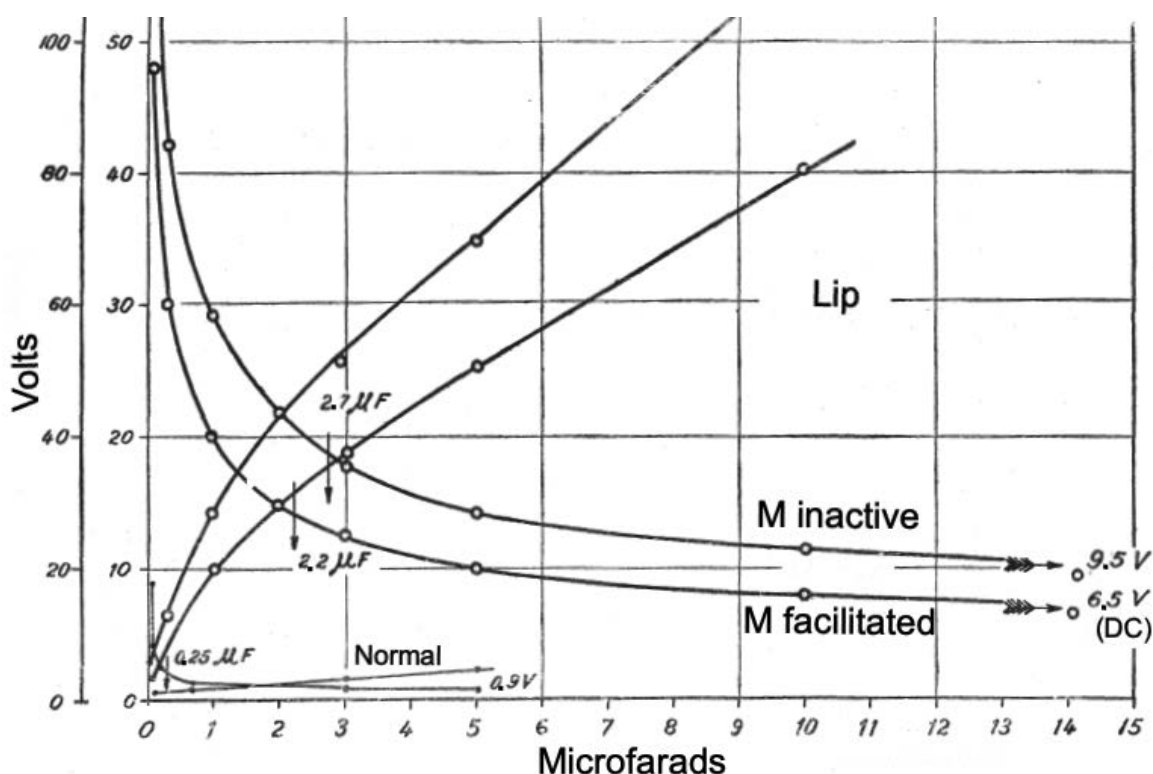
Due to the special characteristics of excitability in these subjects, it is necessary, as in the case of the retina, to avoid fatigue, every action of facilitation, as well as latent addition of stimuli if determinations are made close in time. In this way we obtain the strength-duration (voltage-capacitance) curve for single stimulation, which corresponds to the true tactile excitability state, that is, in the inactive state free of any summative influence. For the curve of facilitation by muscular effort, we request the subject to contract his entire musculature with the maximum strength possible every time a stimulus is applied.

Disturbances of tactile sensitivity are spread through the entire surface of the body according to the characteristics of the central syndrome. The curves shown in Fig.82 refer to the electrical stimulation of the lip. This zone offers an unusually remarkably lower rheobase threshold, which allows to take better advantage of the voltage scale in this elemental excitability curve, and above all, in other curves corresponding to more complex functional levels, as we shall see later.

Figure 82 shows the two curves for subject M, in the inactive state and under facilitation by maximum muscular effort, as well as the curve for a normal subject, all obtained under the same technical conditions, the cathode stimulating the mucosa of the middle part of the lower lip. It should be warned that the sensations obtained are not the same in all cases although we are dealing with the minimal tactile sensation perceptible to electrical stimulation. In the normal subject, a sensation of perfectly localized and circumscribed minimal skin contact is achieved, whereas in the pathological cases, a diffusely spread pressure, like a gust of wind, without any spatial localization is felt, so that the subject feels stimulated on some undetermined part of his body. This occurs both in subject M and subject T. The sensation of minimal intensity that in a normal case goes together with spatial localization (without irradiation), in our cases is devoid of any kind

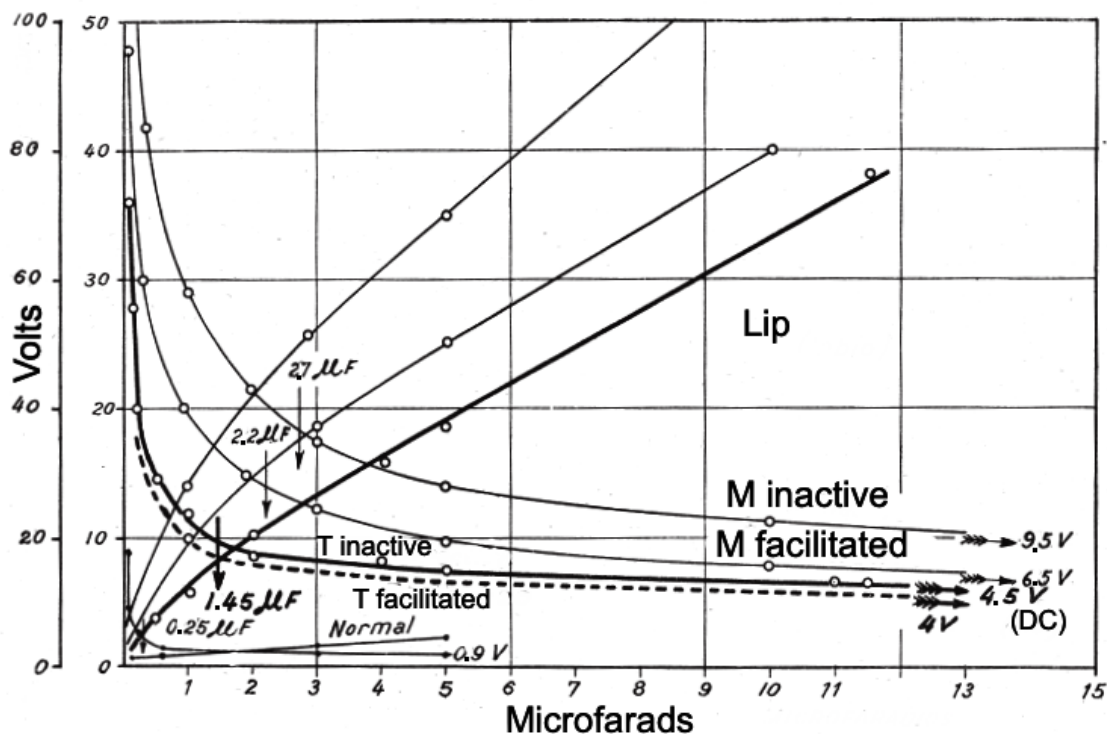
of spatial organization due to asynchrony. This will be studied in depth in the chapter on tactile space.

As seen in the curves, the rheobase and the chronaxic capacity of the inactive state (the true excitability state) in subject M, are about ten times the normal value. Under facilitation, these parameters are significantly lowered, but the decrease is no more than one third. As can be observed, the behavior of tactile excitability in subject M is of the same quantitative order in all aspects as the retinal excitability already studied. The slower reaction time is reflected in the lower curvature of the voltage-capacitance curves compared to the normal subject. It is also seen in the slight concavity towards the origin of the electricity quantity lines for the inactive and facilitated states, whereas in the normal subject it is a straight line.



**Figure 82.** Strength-duration curves according to Hoorweg' law (volts vs. microfarads) for the M case with cathode on the midline of the lower lip, in direct current (DC). Note the different curves for the inactive state and the state under facilitation by strong muscular effort, with rheobase and chronaxic values much higher than in a normal subject. Also compare the respective electricity quantity lines (left scale) for the inactive and the facilitated state.

For the T case, the mentioned quantitative parallelism with vision is also valid, so that in touch, in the inactive state, we found the same increase of four times the normal rheobase and chronaxic values (Fig. 83), as in vision. As for facilitation by muscular effort, the decrease in values is very small but sufficiently pronounced to be evident. In Fig. 83, the two extreme states of subject T can be compared with the analogous states of subject M and with the normal subject.



**Figure 83.** Strength-duration curves according to Hoorweg' law (volts vs. microfarads) with cathode on the midline of the lower lip, in direct current (DC), for subject T (thick curves), compared with subject M (thin curves). Note the different degrees of facilitation in these subjects as well as their different rheobase and chronaxie values.

As we know, the different M and T curves correspond to different losses of brain matter destroyed by the lesion. The values obtained are shown in Table 19.

**Table 19.** Electrical excitability (midline of lower slip).

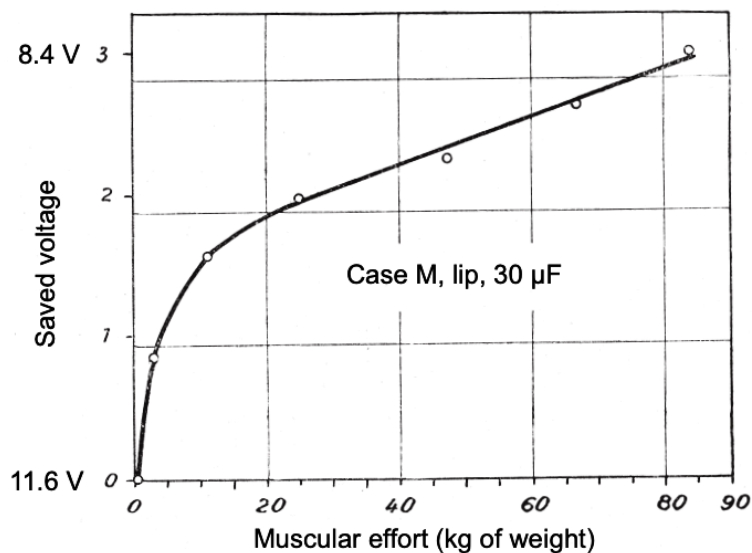
	<i>Rheobase (V)</i>	<i>Chronaxie (<math>\mu F</math>)</i>
M, inactive state	9.5	2.7
M, under facilitation	6.5	2.2 - 2
T, inactive state	4.5	1.45
T, under facilitation	4	1.25
Normal	0.9	0.25 - 0.3

If this table is compared with the analogous table on electrical excitability of the retina in these M and T cases (Table 2 in Vol.1), it can be seen that the excitability disorder in touch is quantitatively of the same order. Therefore, the excitability curves for the retina (Fig. 9 in Vol. 1) and touch (Fig. 83) show an identical arrangement in the set of excitability levels studied. It is important to highlight this similarity because it shows that the central syndrome is revealed in a very pure form, since the excitability deficit is the same quantitatively for both vision and touch. The values obtained in the inactive subject M are about 10 times those of the normal subject, and in subject T they are about four to

five times those of the normal subject. We also see that both subjects present in touch the same permeability to facilitation by muscular effort as in the retina.

## 17.2 The facilitation phenomenon

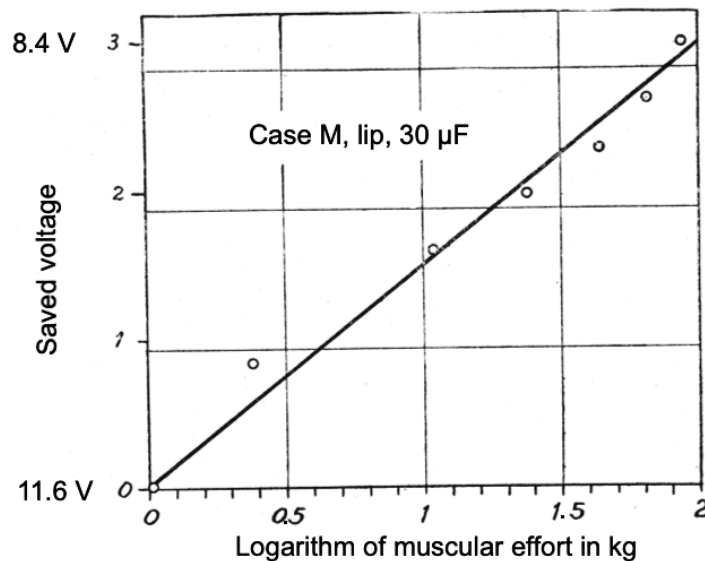
The figures and table above show the different facilitation action in the two cases M and T, i.e., the different *permeability to summative effect* on the nervous centers. This facilitation, by lowering the elementary excitability curve, saves energy of the stimulus. For example, the saving at the rheobase threshold is approximately one third in the M case and one tenth in the T case, with respect to their respective inactive states. Permeability to the summative effect is the same for touch as for vision. It was already said that the impact of the central syndrome is the same on the different receptors.



**Figure 84.** Voltage saved as a function of intensity of facilitation by muscular effort (kg of weight held by subject M). Transition from the inactive state to the facilitated state, for a fixed stimulus duration (30  $\mu$ F).

To know how this saving varies as a function of the facilitation applied, it is sufficient to measure the decrease in voltage (with fixed capacitance close to that of the rheobase threshold) when subject M holds different weights that give the measure of the muscular effort made, analogously to what was done in the case of the retina. Figure 84 shows the curve of this voltage saving. Starting from a voltage of 11.6 volts (corresponding to the inactive state), increasing muscle efforts are applied and the voltage for each new minimum stimulation is determined. At the beginning the saving is fast, as shown by the sharp rise of the curve, becoming slower and slower thereafter. The limit is reached at around 8.4 volts, i.e. after having saved about 3 volts, corresponding to the effort of holding the high load of 80 kg weight. It is notable that low-intensity facilitation already exerts a very favorable action on excitability, as shown in the graph. In fact, small muscular actions, that go unnoticed in ordinary life, act as a not negligible facilitation.

Taking the logarithm of the facilitation, i.e., of the held weight, the relationships between saving and logarithm of facilitation approximates a straight line (Fig. 85), so it can be stated that *the level of excitability is directly proportional to the logarithm of the applied facilitation*. This is a law of great importance for the dynamics of nervous centers. The relationships obtained are of the same kind as those corresponding to this type of experience in vision, and all the considerations made then with respect to vision, are equally valid now.



**Figure 85.** The same experiment as in Fig. 84 but taking the logarithm of the weights held by subject M. The voltage saved (increase in excitability) grows proportionally to the logarithm of muscular effort.

The same behavior is found for other non-muscular or cross-modal sensory facilitations, although their action is much weaker than muscular effort, and can only be clearly evidenced when applied in pathological phenomena with a wide margin of variation, for example diverse alterations of tactile localization, which corresponds to other chapters.

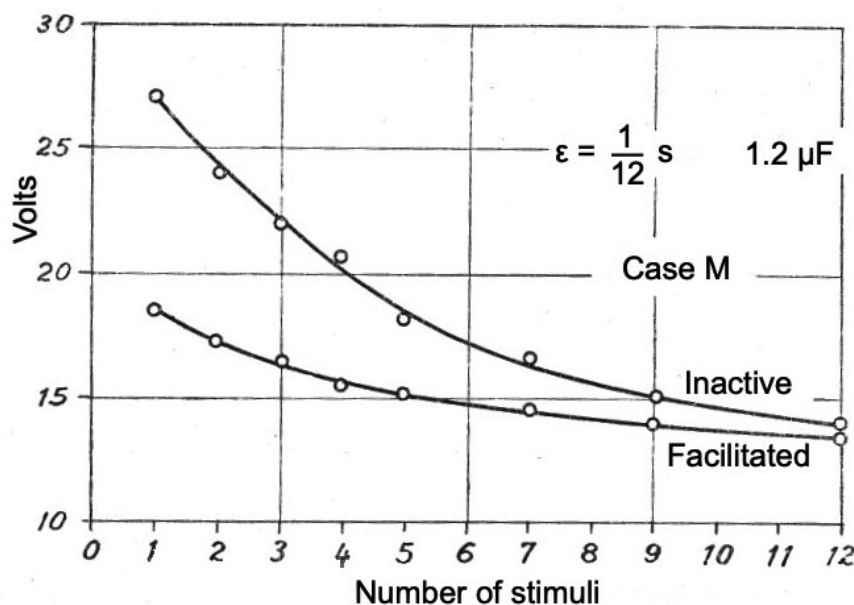
### 17.3 Iterative excitability

Tactile behavior in iterative excitability is of the same kind as that described in the case of vision. Taking into account the previous study of this issue (Sec. 4.3 in Vol. 1), brief indications will suffice.

First of all, it should be noted that, unlike the normal retina, normal cutaneous sensitivity has a certain tendency to iteration (or latent addition) of successive stimuli, although it is small. Several authors such as [Altenburger \(1933\)](#), [Schriever \(1933\)](#), etc., found an iterative effect only for tactile functions such as pain or temperature, but not at all for pressure or mere contact sensation. However, we have found a certain iteration for

mere contact by stimulating the lower lip of normal subjects in order to compare the values with those of our patients. Also, Piéron and Segal (1938) defend the existence of iteration for mere contact by stimulation with capacitors. Considering the values obtained by the different authors, some agreement among them would be possible since the iterative capability for pressure or contact is notably lower than that for pain and temperature. The low iteration for mere contact compared to the other functions is probably the reason why it has gone unnoticed.

We shall now deal only with iterative excitability for contact (or pressure) in our two cases. It is clear that, as in vision, the iterative disorder is very pronounced, the iteration in the normal subject being almost negligible compared to these cases. By examining subject M (with greater disorder) at the midline of his lower lip, as in the previous tests, we can determine the curves for the *law of numbers* and the *law of intervals* in the extreme inactive and facilitated states, as shown in Figs. 86 and 87.



**Figure 86.** Law of numbers in the M case, lower lip midline. Note the different slopes of the curves for the inactive state and the facilitated state. Time interval  $\epsilon = 1/12$  s, capacitance  $1.2 \mu\text{F}$  (half the chronaxie capacitance for the inactive state). The voltage saved is 50% in the inactive state and 30% in the facilitated state.

In Fig. 86 it can be seen that the succession of a number of stimuli causes a marked decrease in the voltage necessary to awaken the tactile sensation which, as mentioned above, is irradiated and not localized as in the normal subject. The capacitance used is approximately half that of the chronaxie in the inactive state, and the time interval between stimuli is  $1/12$  s. In this way, after 12 stimuli, the voltage for the single stimulus is reduced by about half. With facilitation by strong muscular effort and under identical experimental conditions, a more horizontal curve is obtained, corresponding to a lower degree of summation. Whereas 50% of the voltage is saved in the inactive state, only 30% is saved with facilitation. These values correspond to an iteration of medium degree, in

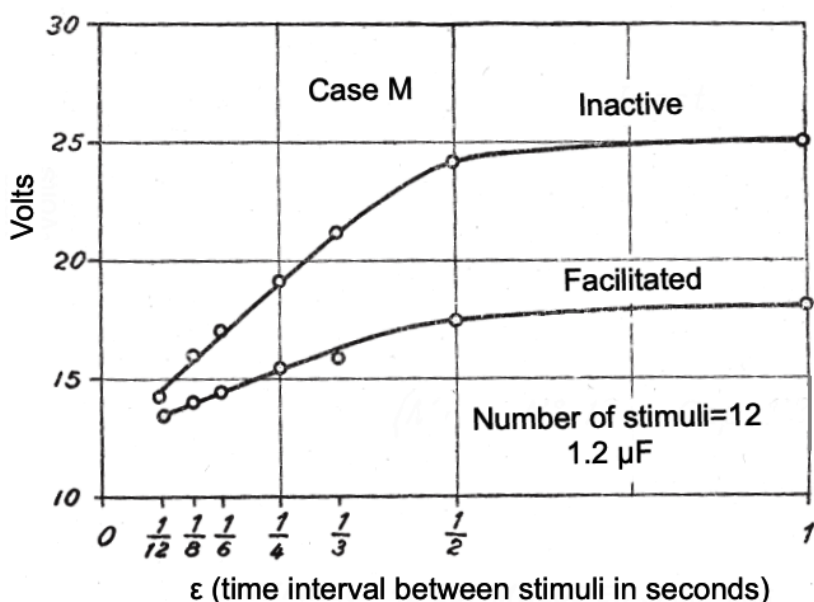


relation to the duration (microfarads) of the stimulus used. Different values can be obtained by varying the time interval between stimuli, as shown in Table 20.

**Table 20.** Iterative excitability in subject M (law of numbers).

Cap. ( $\mu F$ )	$\varepsilon$ (s)	No. stim.	Volts	Condition	Summat. degree
0.1	1/2	1	67.5	Inactive	76 %
		12	16		
		1	49	Facilitated	71 %
		12	14		
1.2	1/2	1	27	Inactive	50 %
		12	14		
		1	18.5	Facilitated	30%
		12	13.5		
20	1/2	1	10.6	Inactive	27 %
		12	8		
		1	8	Facilitated	10 %
		12	7.3		

A behavior similar to that described in the iteration by electrical stimulation of the retina is thus obtained. Suffice it to say that subject M presents in the sense of touch a strong latent addition of stimuli that can reach a degree of summation of 76% or more.

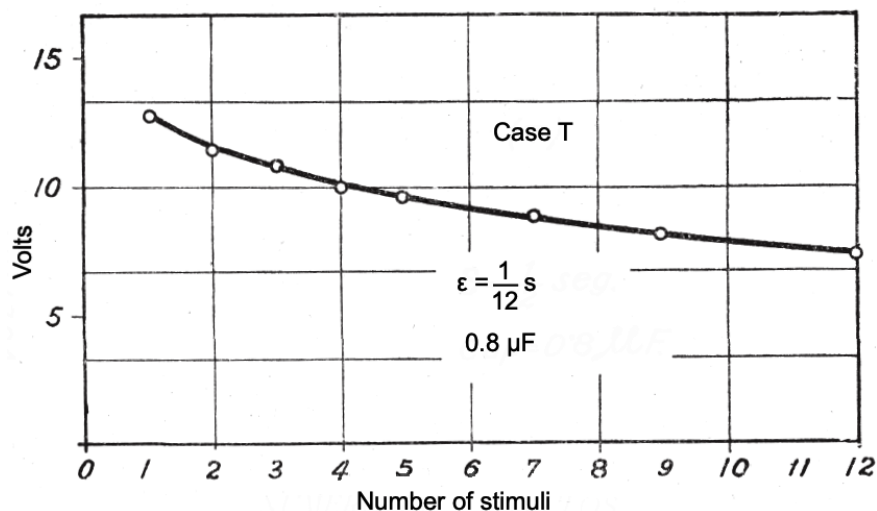


**Figure 87.** Law of intervals in the M case, lower lip midline. Capacitance (stimulus duration) as in the previous figure. Note the different slopes of the curves for the inactive and facilitated state.

As for the law of intervals in this case, Fig. 87 shows the curves for the inactive and facilitated states using a capacitance equal to that used in the law of numbers in the previous figure. Twelve stimuli are used for each determination. By varying the interval between stimuli it is obtained that the smaller the interval the greater the saving of the

threshold voltage. Considerations made on the law of numbers are valid for this law of intervals since it presents the same characteristics under another aspect.

Concerning subject T, of faster excitability than M, we only show Fig. 88 on voltage saving as a function of the number of stimuli. Using a capacitance half that of chronaxie in this subject T, a degree of summation similar to that of subject M under facilitation is obtained, although now the capacitance is much lower. Subject T requires 12 volts for a single stimulus whereas he requires only 8 volts for 12 successive stimuli.



**Figure 88.** Law of numbers in the T case, lower lip midline. The capacitance is half that of chronaxie. Degree of summation about 30%.

With respect to the relationship between the increase in chronaxie and the degree of iterative summation, as well as other considerations on variation by facilitation, etc., we refer to the corresponding section on vision in Vol. 1 in order to avoid repetition. The most important conclusion now lies in the quantitative similarity between the tactile excitability disorder and that of the retina, a consequence of the *homogeneous impact* in pure central syndrome.

## 18 Mechanical excitability

### 18.1 General remarks on mechanical stimulation

The above data on electrical excitability, which allow us to appreciate with great accuracy the functional state of touch in the two cases studied, should be completed with mechanical stimulation. We shall constantly make use of the data under this type of stimulation in the study of complex functions in following chapters.

By applying an esthesiometer to the skin of a hand, with a pressure of a few grams, the required application time (*useful time*) to reach the sensation threshold in subject M in inactive state is about six to seven seconds. This is an extremely high value compared to the normal subject, who in addition to reaching the sensation with a stimulus of much lower intensity, needs at most a useful time of 1/3 second. For the state of maximum facilitation in M, the useful time is reduced to about four seconds in addition to lowering the stimulus intensity. In the much less affected subject T, the useful time is about 2 seconds. Such data according to the different intensity of the brain lesion and different facilitation compensation, follow the law of [Hoorweg \(1892\)](#), and show a behavior similar to that of the light excitability of the retina.

It is important to indicate the different *modes of application of a stimulus* on the skin: 1st, by a single, fixed contact of desired intensity and duration; 2nd, by reiteration of a stimulus at given time intervals; and 3rd, by a kinetic stimulus sliding over the surface of the skin. The above data on useful time correspond to the first method. As for reiteration, a single stimulus that does not cause sensation, applied repeatedly in the same place produces a cumulative effect, and sensation is easily achieved by "temporal summation", provided that the reiteration is sufficiently rapid in relation to the characteristics of the subject's excitability. It should be noted that iterative stimulation does not need to be applied at the same site on the skin; thus, successive stimuli can be applied at different sites close to each other, with a small time interval, until a minimal contact sensation is triggered. In this case, in addition to temporal summation, there is a "spatial summation" effect as in the case of the facilitation phenomenon. This leads us to consider stimulation by a kinetic stimulus, for example, a piece of cotton sliding over a limb or the whole face. If the stimulus is motionless on the skin, it does not cause sensation no matter how much time elapses, due to its weak pressure, whereas by sliding the stimulus over the skin, there comes a moment when contact sensation is triggered. This requires that the moving stimulus covers a certain space (sufficient number of receptors) and at a certain speed (sufficient frequency of receptors). If this speed is

extremely small, the result is the same as if the exciting agent were completely motionless. It is then clear that the conditions in a moving stimulus are the same as in the iteration with a single stimulus repeated at different locations (temporal and spatial summations). A stimulus of subliminal intensity that slides over the skin, causes successive nervous modifications of a certain duration that are accumulated (temporal summation) until, after a certain distance (spatial summation), the sensation appears. The slow reaction time of the nervous centers allows the kinetic stimulus to give rise to the accumulation of the different spatio-temporal residues. In these subjects, stimulation with a moving object on the skin is therefore a mixture of excitation by temporal and spatial summation. In all these stimulation modes and when the energy does not exceed a certain limit, only a sensation of contact with an extensive irradiation and lack of spatial localization is obtained, as we have already indicated with respect to electrical stimulation. Therefore, with a kinetic stimulus, no line or movement is perceived on the skin, and the sensation thus obtained does not differ from that obtained with the other modes of stimulation. Usually, with a kinetic stimulus of a certain speed, a path of about 10 cm in length is sufficient to obtain a contact sensation.

According to what is said, the *development of tactile sensation* is rather slow depending on the case. In the inactive subject M, maximum latency (delay) is obtained between the application of the stimulus and the appearance of the sensation, which, in addition, lasts for a certain time after the stimulus has been suppressed. Of particular interest is this persistence of the sensation, which in the inactive subject M can reach about two seconds at most. This persistence gives rise to very remarkable phenomena if facilitation acts on the sensory residue. Thus, a sensation of simple contact (without any localization in these cases) during the period of persistence after the stimulus has been removed, can be markedly intensified if facilitation by intense muscular contraction of the whole body is rapidly applied. This intensification not only increases the clarity of the sensation, but also endows it with a spatial nature, making its localization possible. Obviously, the new effect achieved is extremely brief but sufficient for the change to be felt. Such "reactivation" of the sensory residue is also obtained in the case of vision. For example, let us consider a vertical arrow in conditions such that the image perceived by the inactive subject M is tilted  $90^\circ$ ; it turns out that when the arrow is removed from the visual field and facilitation by muscular effort is immediately introduced, an inclination of about  $30^\circ$  is obtained and all perception disappears very rapidly, since the facilitation has acted only on the residual image of the sensation, i.e. on the residue close to extinction. The longer the time elapsed between the exclusion of the stimulating agent and the application of facilitation, the lower the sensory level obtained, since summation by facilitation is exerted on an increasingly weaker sensory residue. In both vision and touch, if the elapsed time is 1.5 seconds, reactivation is almost nil, and several tests indicate that in both sensory systems, the maximum sensory persistence does not exceed 2 seconds. As for the dependence of the degree of intensification and localization of the tactile residue on the time at which facilitation is applied, see the chapter on tactile space for understanding the phenomena of desynchronization in spatial localization.

Concerning the *facilitation* effect, we know from electrical excitability its different effect in the M and T cases. In the T case, facilitation also exerts an unquestionable

summative effect on mechanical tactile stimulation, although to a lesser degree than in the M case. Except for muscular contraction, other types of cross-modal sensory facilitation are of little importance in touch. Thus, both acoustic and visual stimulation hardly facilitate tactile function, due to the minimal central effect they probably exert. By contrast, the sensation of a tactile stimulus is significantly improved when a second tactile stimulation coexists, such as a gentle touch on another region of the skin. Thus, a *bi-tactile effect* (spatial summation of stimuli) is obtained.

*Sensory fatigue* is very noticeable in our cases. Using a minimal stimulus in subject M to obtain a sensation of simple contact, and maintaining that stimulus indefinitely, it happens that after about 30 seconds from the emergence of the sensation, it ceases to be perceived. If the maximum facilitation by strong muscular effort is then applied, the sensation reappears and, moreover, it is localized, a fact that did not occur before. But when facilitation stops, the localization disappears immediately, and the remaining sensation of contact is lost more quickly than in the previous experiment, in just about 10 seconds. This fatigue may be due in part to a phenomenon of sensory accommodation (Adrian 1928), but to a greater extent is due to an increased threshold caused by sensory fatigue.

Regarding tactile *adaptation*, it is worth mentioning the process of sensitive adaptation or tactile recovery after some alteration of the sensory organ. For example, when altering a fingertip by rubbing it over a rough, hard surface for half a minute, the normal subject recovers most of its pre-alteration sensitivity within a minute after the end of the rubbing, whereas the inactive subject M may take up to four minutes. Since we have not quantitatively determined the evolution of this adaptation process, we can only give rudimentary data, but it is to be expected that it follows an analogous behavior to the adaptation process studied in vision.

The different behaviors of subject M in relation to tactile sensation can be illustrated by considering *weight sensation*. He greatly underestimates a considerable weight held in his hand. This is demonstrated because under facilitation by strong muscular contraction, the sensation of weight increases greatly, well over twice, as indicated by the subject. The differential threshold also increases; thus, when successively holding different weights with the same hand, or with both hands at the same time, weights of 0.5 kg and 1 kg seem nearly the same. Only when he makes a few small movements to compare the weights, does he perceive a minimal difference. Under facilitation by muscular effort and without movements to compare the weights, both are perceived as heavier and very different from each other, to the point of presuming that one is twice as heavy as the other.

In the inactive state and without any type of movement, subject M perceives a difference, although not excessive, when comparing weights of 0.5 kg and 2 kg. This difference increases when performing movements to compare weights. With facilitation, the perceived difference can become three times greater than in the inactive state, and even greater with movements to estimate weights. Thus, in the M case, there is a decrease in the perceived weight (absolute threshold) and in the comparative differences (differential threshold), varying according to the corrective effect of facilitation, which never entirely normalizes the function.

To obtain sensations of similar intensity in the inactive state and under facilitation, different weights are needed to compare them successively. Thus, 1 kg in the inactive state can be equated to 300 grams under facilitation. Also, 1 kg in the inactive state is sometimes perceived as equal to 0.5 kg under facilitation. However, the subject rejects that 1 kg in the inactive state is equal to 200 grams under facilitation because he perceives that the latter weighs less. All this means that weights are perceived to be about three times heavier under facilitation than in the inactive state.

All these determinations refer to evaluation of strengths, since the weights hang from a ring that the subject grasps with his fingers. It is clear that these measurements are not very accurate, but they are useful to get an idea of the subject's functional capacity. In particular, the equating relationship between inactivity and facilitation seems to be well established since it is verified in other types of tests described below.

## **18.2 Vibration sensitivity. Intermittent stimulation**

The examination of vibration sensitivity in our patients by means of tuning forks of different frequencies will show important features about intermittent stimulation, appreciation of time intervals, fusion of successive stimuli, etc.; all related to the nervous excitability disorder.

Regarding *vibration sensitivity*, there are very discordant opinions among authors. [Dejerine and Dejerine-Klumpke \(1914\)](#) and [Egger \(1899\)](#) consider it to be a specific sensitivity of the osseous system. Later it was shown that the sensation was equally obtained on soft tissues. In particular, [Frey \(1910, 1913, 1916/1917, 1928\)](#) argues that it is a modality of pressure sensitivity that belongs to cutaneous sensitivity and has no relation to deep sensitivity. Bones are particularly sensitive to vibrations because they transmit sensation to a large surface of skin. [Piéron \(1936\)](#) states that there is no independent sense of vibration, but a vibratory excitability as a type of mechanical excitability involving superficial and deep receptors, predominating on bones for anatomical reasons. More modern neurologists think, in line with Frey, that vibration is not a sensory individuality, although there are still many who believe otherwise.

Given the excitability characteristics of our brain-injured patients, the mere application of a tuning fork on the skin (soft or bony parts) is not sufficient to get an idea of the state of vibration sensitivity, since we could make the mistake of accepting a loss of it. As in other determinations, the correct approach is to study the function along a quantitative scale, accurately determining excitability characteristics. For this purpose, we use a series of tuning forks for clinical use with frequencies between 32 Hz and 1024 Hz. The tuning fork is struck at maximum and immediately placed softly on the left styloid process of the radius, so that the contact or pressure of the tuning fork does not mask the sensation of vibration. The results obtained in both subjects are shown in Table 21 where the duration of the perceived vibration for tuning forks of different frequency is indicated.

**Table 21.** Vibration sensitivity. Duration (seconds) of the perceived vibration.

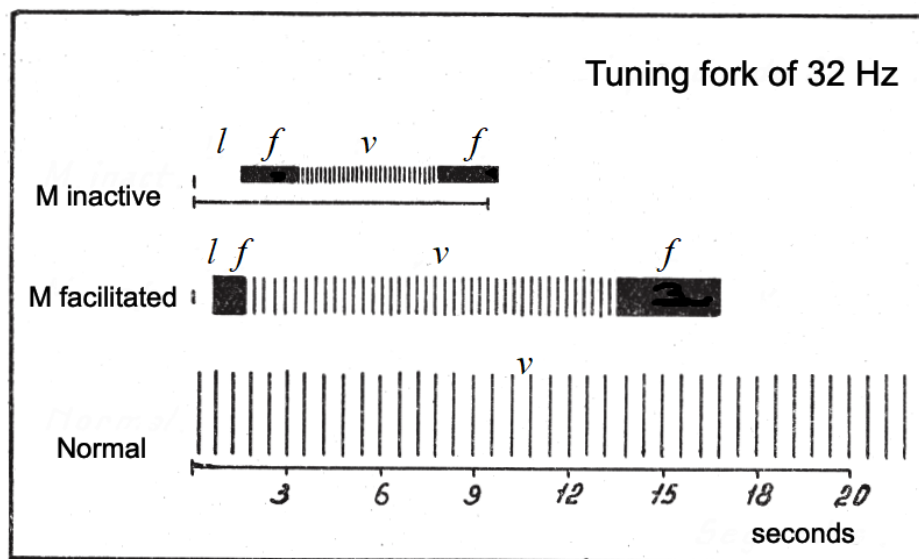
<i>Frequency (Hz)</i>	<i>M inactive (s)</i>	<i>M facilitated (s)</i>	<i>T inactive (s)</i>	<i>T facilitated (s)</i>	<i>Normal subject (s)</i>
32	4.5	7.5	12	18	25
64	4	6.5	12	18	30
128	2 - 1.5	3	8.5	13	18
265	0.7	1.5	5	8	12
435	---	?			
512	---	---	4	6	9
1024	---	---	---	---	??

It should be noted that the tuning forks have unequal mass, so the duration of vibration is different. However, the normal subject is a good reference with which to compare the pathological cases. First of all, the different duration of vibration perception in the different cases is noted, as well as the different upper limit of frequency, all related to the already known characteristics of excitability. Under maximum facilitation by strong muscular effort, the M case doubles the duration with respect to the inactive state, whereas in the T case the duration increases by only half a point more. Note that the duration in subject T under this type of facilitation is still well below that of the normal subject, although T is able to perceive all the frequencies of the normal subject. In contrast, M loses high frequencies, and for those at which he perceives vibration sensation, the duration is very brief, especially in the inactive state. Subject M in the inactive state feels vibration only in very low frequencies, always during a brief period of time, but when examined with a tuning fork for clinical use such as the one of 435 Hz, he is not able to feel the least sensation of vibration, and there is also great difficulty when using the 256 Hz tuning fork because of the short duration of the sensation. However, it would be a mistake to deny him capability to feel vibration sensation, since this is possible using very low frequencies. As for the normal subject, with the 1024 Hz tuning fork, it is very difficult to obtain a vibration on the styloid process of the radius, and one can practically say that there is no perception. Only by paying close attention and striking the tuning fork strongly, it is perhaps possible to feel a very slight vibration signal of negligible duration.

Apart from the considerable reduction of vibratory sensitivity in our patients, especially in the inactive M, we must consider the *evolution of the sensory process* just after the tuning fork is applied, in order to understand the nature of the disorder. In the normal subject, the latency time from application to vibration perception is negligible compared to pathological cases, in which this time can be easily measured with a stopwatch. In subject M inactive, using a low frequency tuning fork, there is a latency of 1 to 2 seconds. However, it is very important to note that this latency time is not to awaken the sensation of bone tremor, but to obtain only uniform contact sensation without any vibration. This sensation of uniform contact lasts in turn for some time, and is followed

by the sensation of vibration as a small tremor. In the above Table 21, the duration values refer exclusively to this true vibration phase, not to the whole sensory process. The process ends by evolving from tremor to uniform sensation again and, finally, to the complete loss of all sensory activity. It should not be considered extraordinary that even the sensation of contact ceases even if the tuning fork is still applied to the skin, since the tuning fork does not press on the skin with its full weight, but is applied laterally to transmit exclusively the vibratory movement. In addition, it should not be forgotten the high threshold of excitability due to the brain disorder, especially in the inactive state. When the rather large tuning fork of 32 Hz is applied, without vibrating, with its full weight on the skin of the inactive subject M, it takes him 5 to 6 seconds to perceive the sensation of contact, whereas when it vibrates, it takes only 1 to 2 seconds to have the same kind of sensation. This clearly indicates that in vibratory stimulation there is an accumulation (summation) of discontinuous stimuli.

By studying in detail the evolution of the vibratory sensory process when a 32 Hz tuning fork is used in the inactive subject M, *several phases* are determined that depend on the changes in nervous excitability during the tuning fork action time. A diagram of this process is shown in Fig. 89.



**Figure 89.** Qualitative diagram of vibratory sensory phases. Latency, *l*; fusion, *f*; vibration, *v*. In the diagram, the scale is not the same for the three cases. See data in Table 21. Note the different duration of the phases in the three cases considered. The height of the lines indicates the intensity of the sensation.

In addition to the aforementioned sensory latency, there is a phase of continuous sensation and then a phase of vibratory sensation. The first corresponds to the fusion of stimuli, that is, to the fusion of the vibratory tremor. In this phase the excitability is still low, and the vibratory impulses are perceived so slowly that there is no possibility to discriminate between them and they seem fused. However, since sensitivity tends to improve by accumulation of discontinuous impulses, there comes a time when the tremor can be perceived because the sensation becomes more intense and rapid. However, the



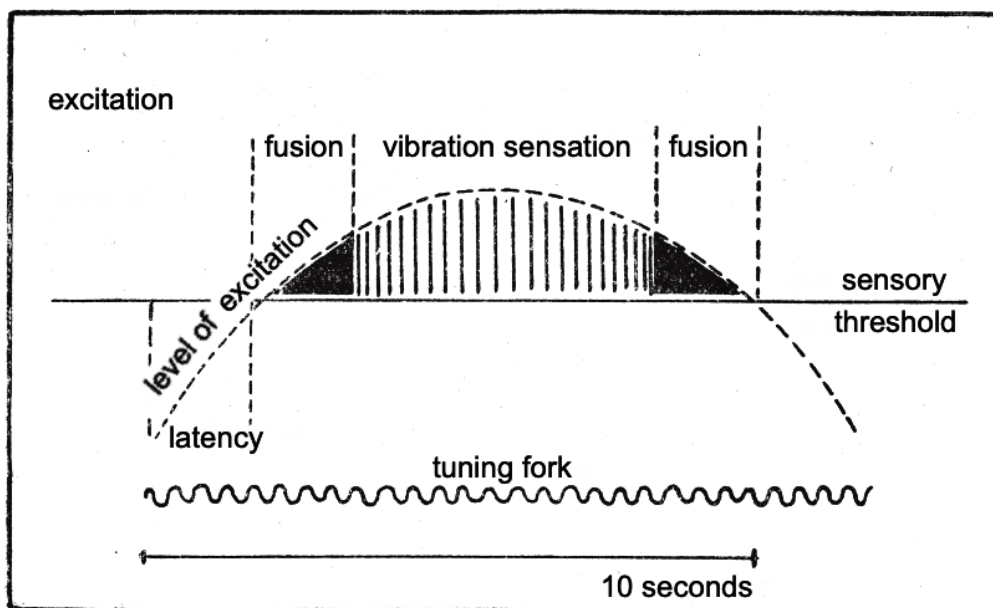
interval that can be perceived is much smaller than that perceived by the normal subject; therefore, the vibratory tremor results to be overestimated since it is perceived very quickly. Thus, facilitation not only intensifies the sensation but also makes the perceived rhythm slower, although it is still faster than in the normal subject. The situation is the same as for intermittent stimulation in vision. In a normal subject, it already happens that when fusion is obtained for a given rhythm and luminous intensity, if the illumination is increased, the discontinuity of the flickering appears because the reaction becomes faster. Here, in tuning fork vibration, the indicated change is due to the improvement of the sensory level as a result of the accumulation of discontinuous stimuli, which is very easy in view of the high iterative capacity of subject M.

As for the disappearance of sensation, the process occurs in reverse order, i.e., from vibration to fusion and, finally, to sensory silence even though the tuning fork is still applied on the skin. To interpret this process, one must realize that after the tuning fork has begun to vibrate at its maximum by the action of a strong blow, it tends to dampen the intensity of the vibration and therefore, the stimulus becomes less and less intense and the process of evolution of the sensation reaches a point where it is reversed, ending as it began. The duration of the whole process is very short in the M case compared to the T case, and much shorter than in the normal subject, either because the small decrease of the tuning fork vibration intensity influences very soon to lower the level of functional activity prematurely being impossible to maintain an indispensable minimum, or because in these subjects there is a very rapid adaptation to vibration which tends to erase it (Adrian 1928, Piéron 1936) as it happens also in the normal subject. In either case, both factors could act together (see Fig. 90).

It should be noted that the phases correspond to different spatial levels. In the phases of mere contact or fusion there is no localization, whereas in the vibratory tremor phase there is a spatial localization that is more perfect (in accordance with the real place stimulated) the clearer and more lasting the vibratory sensation is. Quantitative data on this point are given in the chapter on spatial localization further on. Regarding the indicated phases, when a tuning fork of 435 Hz is applied laterally on the styloid process of the inactive subject M, he perceives neither tremor nor contact or fusion; instead, under facilitation, he goes beyond the fusion phase but does not clearly perceive the vibration, which seems practically null.

The *exclusion of high vibration frequencies* (see Table 21) can be explained by the impossibility of latent addition due to the extremely small interval between stimuli that would appear in the refractory period of the preceding stimulus. We know that the smaller the interval, the greater the degree of summation, but this occurs up to a certain frequency ("optimum frequency") beyond which, if the time interval decreases, the degree of summation also decreases. Finally, the degree of summation becomes null when the time interval is equal to the duration of the refractory period of the first stimulus (Bremer 1930 a, Bremer 1930 b, Koehnlein 1934). Given the great slowness of nervous processes (hence increased refractory period) in our brain-injured patients, especially in the inactive subject M, the limit for latent addition is reached very soon, and for this reason tuning forks of medium and even low vibration frequency (such as 256 Hz) hardly produce a slight and short-lived vibration sensation (Table 21). Since tuning forks are not of the same mass, it

is difficult to correctly establish comparative relationships, but it is perhaps possible to state that almost all tuning forks whose vibration can be perceived by the inactive subject M are in a range in which the latent addition tends to decrease when the frequency of vibration increases.



**Figure 90.** Diagram of the sensory periods according to the level of excitation (dashed curve) during vibration stimulation in subject M inactive. Same conditions as in Fig. 7.

This same slowness in nervous reaction explains that when the tremor is finally felt, it is perceived much faster than it really is, due to the fact that the interval between stimuli becomes very short. This perception is similar to that in intermittent visual stimulation with respect flickering and fusion (Section 5.3 of Vol. 1), and in general with respect to the perception of movement, which in both vision and touch become accelerated. Facilitation, by improving excitability, corrects in part this acceleration, lengthening the sensory interval somewhat.

Thus, it is possible to obtain a *sensory matching of vibration* between the inactive state and the state under facilitation, e.g., to feel tremor with the same frequency both with 32 Hz stimulation in the inactive state and with 128 Hz stimulation in the facilitated state (by strong muscular effort). In general, facilitation produces a decrease in speed two or three times that perceived in the inactive state (for a suitable tuning fork); therefore, when the tuning fork frequency perceived under facilitation is two or three times higher than that perceived in the inactive state, the matching can easily be achieved by means of facilitation. It can then be easily inferred that the vibration felt by a normal subject with a low-frequency tuning fork corresponds to an extremely high vibration sensation in subject M inactive. And given this correspondence, when using medium or somewhat high frequencies, whereas the normal subject still perceives them easily, they produce only fusion of stimuli or no sensation at all in subject M inactive. In relation to what said, the following frequency equating is plausible: 32 Hz in M inactive = 128 Hz in M under

facilitation = 435 Hz in T = more than 512 Hz in normal subject; thus obtaining a relationship between the values very similar to that of their respective chronaxies.

As for *frequency discrimination* by the inactive subject M, the 32 and 64 Hz tuning forks at first seem the same to him, and later he thinks there is some difference, although very small. He appreciates the difference between the 32 and 128 Hz tuning forks much better. Under facilitation, the difference between 32 and 64 Hz is easily perceived.

All the above tests refer to the application of the tuning fork on the bone (styloid process of the radius), but can be approximately valid as well for a soft part of the skin, such as the fingertip. In this last case, the sensation is weaker and more pressure is required when applying the tuning fork.

The study of the factors that come into play in the perception of vibration by tuning forks in these subjects shows quite clearly that vibration is only a modality of excitation of the ordinary sense of pressure or contact, since the properties of vibration correspond entirely to the characteristics of pressure or contact in rhythmic stimulation. Consequently, any specificity of a presumed sense of vibration must be rejected.

Some clinicians think that there may be a separation between vibratory sensitivity and surface touch sensitivity, as if they were independent senses, for example, with regard to certain spinal syndromes. In an observation of [Guillain \(1905\)](#), the elementary sensitivity was abolished, but the vibration of a tuning fork was perceived. In such a case, it is to be thought that, whereas a single stimulation by pressure was not perceived, the accumulation of stimuli by the tuning fork finally resulted in a sensation, even a tremor sensation. There is therefore no real separation between specific senses, but rather different reactions to different modes of stimulation, and the severed or injured spinal cord is only able to respond to iterative stimulation. We give this example to highlight the inconsistency of establishing many clinical “specificities” without having studied in depth the physiological nature of sensory and nervous processes.

As for the cortical disorders of tactile sensitivity, it can be stated that tactile sensitivity is very easily altered since it is a discrimination function (perception of very short intervals), showing in general a behavior similar to that of sensitivity to surface or deep movement. We must not lose sight of the fact that, as already stated in this research, the alteration of a given sensory system is always global, thus affecting all types of activities, which appear reduced according to their physiological demands. The degree of vibration disturbance will then depend on the degree of brain excitability disturbance, as indicated in Table 21. This table demonstrates the need to test vibration across a range of frequencies to provide a clear picture of the state of the function.

## **APPENDIX: Other cases with bilateral tactile disorder in unilateral cortical lesion (central syndrome)**

In this section we draw attention to a number of cases reported in the neurological literature in which different tactile disturbances have been observed in both halves of the body despite presenting a unilateral lesion. Such manifestations have remained without

adequate interpretation, and for our part we can assert that they simply constitute alterations of the central syndrome type, i.e., the prototype of disturbance in brain dynamics.

A first case of this nature is the one described by [Oppenheim \(1906\)](#). Over the course of a year, the subject presented weakness for movements on the entire right side of the body, but only abolition of fine movements of the fingers. He also had Jacksonian seizures on the right side with subsequent generalization to the whole body. Deep tendon reflexes were slightly hyperactive on the right side, with doubtful Babinski's sign. As for sensitivity: on the *right side*, slight decrease of tactile sensitivity; in addition, considerable alteration in perception of attitudes and very great astereognosis. On the *left side*, normal motor functions and sensitivity, but astereognosis was present.

The author was inclined to admit the existence of a unilateral lesion on the left parietal region and he tried to explain the bilateral astereognosis on the basis of the functional dominance of the left hemisphere. Therefore, surgery was advised. A tumor was found, the size of an egg, on the middle part of the postcentral gyrus and posterior parietal lobe. Five days after tumor resection, the astereognosis of the left hand had diminished considerably.

In 1915, a similar observation was made by [Goldstein \(1915\)](#). A 53-year-old man developed a right incomplete hemiparesis, bilateral apraxia and bilateral astereognosis, within a period of one year. Language was possible for simple words, and affected for more complex functions. Elementary sensitivity to touch, pain and temperature were little altered. By contrast, the appreciation of positions was very disturbed on both sides of the body. The author states that the alterations in sensitivity are not so great as to explain the patient's severe astereognosis.

Necropsy revealed a large cystic cavity in the left hemisphere extending to the postcentral, supramarginal and inferior parietal gyri. Histologically, no anatomical abnormality in the right hemisphere was found to explain the left astereognosis.

In these two first cases of bilateral astereognosis with a single left lesion, we are dealing with an incompletely examined central syndrome. In the former, the side opposite to the lesion is slightly more affected (as in our case T), and elementary alterations of tactile sensitivity, although slight, are clearly observed; instead, in the homolateral side, only the perception of spatial forms is affected. In this left side, touch is less impaired, but it is possible that elementary disturbances have gone unnoticed, and a quantitative examination of excitability would have shown a significant generalized disturbance.

As for the second case, alterations of the elementary sensitivity for contact, pain and temperature are observed, and if they are less pronounced than the perception of position or configuration of objects is due to the exclusion of different functions according to their different physiological demands (dynamic reduction). The dominant action of the left hemisphere is not enough to explain everything. In addition, we have verified the dynamic repercussion of the central syndrome in both left and right unilateral lesions, as will be seen in the corresponding place. The supposed particularity of the left hemisphere is already a repercussion, but this issue is left for other chapters. Since these cases are of the central syndrome type, many other bilateral disturbances of other sensory systems -and in all their activities- should be present, but are easily overlooked in ordinary examinations. However, in the case of [Goldstein \(1915\)](#), more varied symptoms (astereognosis, aphasia, apraxia, etc.) are mentioned.

The characteristics of the central syndrome are even more evident in the four cases collected by [Foix \(1922\)](#), with a clinical syndrome characterized by hemiplegia and

aphasia of variable intensity, ideomotor apraxia and bilateral alterations of sensitivity. Regarding the latter, the author specifies that these alterations predominate on the notion of position, and secondarily on the stereognostic sense; other modes of sensitivity being only affected in an occasional way. The touch impairment, homolateral to the lesion, is interpreted as an anesthesia due to agnosia. The author insists on asserting that the whole set of symptoms described undoubtedly depends on a single lesion, so that the bilateral sensory alterations evolve in an absolutely parallel manner on both sides, as do the rest of the alterations.

In one of these cases of [Foix \(1922\)](#), the examination of sensitivity shows on the *right side* (with hemiparesis): moderate hypoesthesia for all modalities (evident alterations of the notion of position, tactile sensitivity, stereognostic sense, etc.). On the *left side*: very evident hypoesthesia, slight touch lost on all the side except on the face, very altered position in small joints, delay of heat perception and evident astereognosia.

The same features are present in the other cases of Foix, in which the intensity of their involvement varies somewhat, but they always show bilateral pathological manifestations in sensitivity, and almost always a little more pronounced on the paretic side. The most severe disorder is for position sense, but on the left side, homolateral to the lesion, minor alterations of elementary sensitivity (omission of some contacts, delay in the perception of pain and temperature) are also found in a very constant way in most cases.

The location of the lesions in the cases of [Foix \(1922\)](#) is very similar to that of the case of [Oppenheim \(1906\)](#) and the cases of [Goldstein \(1915\)](#).

We can say that in all these observations we are dealing with tactile disorders of the central syndrome type and in a rather pure form, i.e. with a well pronounced symmetrical distribution (bilateral parallelism highlighted by Foix), since the small difference in favor of the contralateral side to the lesion is almost negligible, as is the case in our T case. The difficulties to explain such symptomatology within the brain localization system are obvious. [Foix \(1922\)](#) arbitrarily mentions “anesthesia by agnosia” to interpret the tactile disorder homolateral to the lesion, also assuming the functional supremacy of the left hemisphere. But it is evident that in this way, the sensitivity disorder for elementary qualities (pressure, pain, temperature) remains unexplained, and as for other perceptual activities, the interpretation is rather forced. Proof of this is that some authors try to understand the cases of [Oppenheim \(1906\)](#) and [Goldstein \(1915\)](#) (with bilateral astereognosis due to a left tumor) as due to bilateral lesions, even if they are minimal, in order to explain the homolateral tactile alteration within the brain localization system.

In short, for all these cases, some authors invoke the dominance of the left hemisphere (when the lesion is on that side) while others, not believing that this is sufficient to explain the variety and depth of the symptoms, are in favor of the existence of bilateral lesions. Finally, within brain dynamics, such cases are easily interpreted as types of central syndrome, which even allows assuming the existence of many other symptoms in other sensory systems.

We find again all these difficulties in the study of [Goldstein and Gelb \(1919\)](#) on the tactile functions of the Schneider case, increasing even more the complication with new

theories that far from solving the problem, make it more obscure. But before commenting on some circumstances of the Schneider case, it is worth noting that many of the cases of visual agnosia studied by [Stauffenberg \(1914\)](#), also with bilateral astereognosis, correspond to the central syndrome as well. Elementary tactile sensitivity should also have been more or less impaired, and if the disorder of form recognition was more prominent than others, it was only due to the characteristics of the dynamic reduction, already repeatedly indicated.

Moreover, certain experiments of *resection in the cerebral cortex* of vertebrate animals allow to obtain favorable evidences on the importance of the quantity of removed gray matter (magnitude of the lesion) for the intensity of the functional disorder. This supports both the investigations of [Lashley \(1929\)](#) and the fact that the effects of lesions depend on their magnitude and position according to the present brain dynamics investigation. For example, [Rothmann \(1914\)](#) obtained in the macaque, by simultaneous resection of the postcentral gyrus and the supramarginal gyrus, much more severe and lasting alterations than those obtained by the isolated removal of either of them. There are also analogous observations by [Minkowski \(1917\)](#), in which the more complete the resection of the parietal lobe, the more intense and long-lasting the tactile sensitivity disorder.

Likewise, we must bear in mind the experiments of [Pavlov \(1927\)](#) on the destruction of the cortical area of the analyzers. This experiments show that the discrimination of stimuli decreases as the quantitative destruction of this area increases, and this is valid for any sensory system.

In the solution of these problems, particularly in the human brain, careful clinical observations and proper analysis of the altered functions play the main role. The Schneider case of [Goldstein and Gelb \(1918, 1919\)](#), although lacking a proper physiological examination, has been significant for the very detailed clinical study of the intricate visual and tactile disturbances, but above all, for the singular characteristics of the disorder. In touch, there is an alteration of spatial functions (localization, spatial discrimination, forms, etc.) and of recognition, all of these alterations varying with certain muscular jerks.

Also in the Schneider case there was a bilateral alteration of touch as in the case of [Goldstein \(1915\)](#), but while in the latter the author is inclined to admit the dominance of the left hemisphere, as [Oppenheim \(1906\)](#) in his case, in the case of Schneider the interpretation changes radically. This is because upon detection of a complex visual disorder, the entire bilateral spatio-tactile disorder was made dependent on the loss of the visual spatial influence. Therefore, touch is considered basically intact. Thus, the disorder lies in the visual system, and this in turn has psychological effects on touch, depriving it of its spatial quality.

Besides the fact that this hypothesis of visual influence is a matter to be proved, it must be borne in mind that in both the case of 1915 and Schneider, elementary sensitivity is not completely intact. Hence, the debatable hypothesis of this influence cannot even be accepted as an explanation for the tactile disorder. In the first case (1915), elementary sensitivity to touch, pain and temperature is little altered. As for the Schneider case, the second work on this patient points out that the elementary sensitivities (pressure, pain,

temperature), although essentially intact, are somewhat reduced over the entire surface of the body, i.e., on both sides. But [Goldstein and Gelb \(1919\)](#) think that this decrease in elementary sensitivities should be interpreted as a deficit of attention on the part of the patient during the examination, and in an explanatory footnote they ask themselves “how else could a decrease in sensitivity over the entire surface of the body occur?”. This question ceases to be problematic in the brain dynamics we present, since the alteration of elementary excitability for the whole body in a unilateral lesion corresponds precisely to the central syndrome pattern. Ruling out the attentional deficit explanation is not difficult, nor is the visual influence explanation, considering the following reasons: first, the significant concentric reduction of the visual field in Schneider already indicates a disturbance in visual elementary excitability which, as in touch, does not derive from any attention deficit; second, the tactile disturbances encompass the most elementary functions, which are outside the presumed spatial influence; third, the intensity of the disorder in both systems, vision and touch, is the same and equally distributed in both halves of the body (concentric reduction and decrease of elementary sensitivity in the whole body). Therefore, the classification into primary disorders for vision and secondary disorders for touch is arbitrary. The above arguments show the difficulties and theoretical obscurity of the interpretation of the Schneider case by [Goldstein and Gelb \(1918, 1919\)](#). A strong argument is the finding of dynamic action phenomena in our M and T patients and the characterization of the central syndrome (see Vol. 1), which clearly determines the state of brain excitability and the distribution of the disorder (on both sides of the body and for all sensory systems). It is then possible not only to easily understand the multiple disorders of the Schneider patient but also, by including him in the central syndrome, to predict many others that have not been found in him.

A brief review of a series of cases with brain lesions published at very different times ([Oppenheim 1906](#); [Goldstein 1915](#); [Foix 1922](#); [Stauffenberg 1914, 1918](#); [Goldstein and Gelb 1918, 1919](#)), only a few among many others, shows that if the examination of the patients is rather careful, the interpretation of the paradoxical symptoms encountered presents great difficulties within the theory of brain localization. In order to maintain this theory, the authors need to resort to additional hypotheses at the expense of arbitrarily diminishing the value of certain alterations or even denying or disregarding them.

As for astereognosis as an isolated or independent defect, the position of [Stein and Weizsäcker \(1927\)](#) against such autonomy is noteworthy. They rely on experimental facts about lability of the threshold of sensation, abnormal fatigability, etc., all of which refers the defect to a basic disturbance of nervous excitability.

A detailed discussion of each case would take a long time and would lead us to the same results and conclusions that in general terms have already been stated. These are: all these cases show more or less fragmentarily the characteristics of the central syndrome, and the difficulties of interpretation they raise are automatically solved within the brain dynamics exposed. The more complete the examination of all brain functions and especially the better the quantitative determination of the different excitabilities, the more accurate is the inclusion of a case in the central syndrome.

This last requirement is of great importance in order to reveal all the elements of the central syndrome, especially the alteration of the most elementary functions, thus

demonstrating the overall alteration of a given sensory system. Otherwise, since in ordinary examinations attention is usually paid only to the abolition or presence of functions but not to the *degree of impairment*, many functions are easily admitted as intact, particularly the more or less elementary ones, leading to erroneous conclusions about the specificity and localization of certain functions. In addition, there is the masking of symptoms due to facilitation actions, ways of applying stimuli, usual examination techniques, etc., as has been repeatedly indicated throughout the exposition of this research (see Vol. 1), and as will be seen in the next chapters on tactile functions. All this is to point out that, before raising fundamental theoretical questions, careful investigation is required, which is not easy, and it is only with much patience that sufficient data are obtained to understand the characteristics of the disorder. In fact, at the beginning of these investigations, our M and T patients did not seem to be as profoundly affected as the detailed study of them later revealed.



# Tactile sensations

## 19 Dynamics of tactile sensations

### 19.1 General aspects. Heterotactile interval

The so-called fundamental *tactile sensations* are *pressure, pain and temperature*, which suffer in our brain-injured patients a dynamic alteration evidenced both by a considerable decrease in excitability and by a peculiar disaggregation due to a functional desynchronization. A certain parallel can now be established with the disorder in colors although the organization of these tactile functions is much simpler. A characteristic of colors and tactile sensations is the reduction of excitability and the noticeable interval between the initial sensation and the more defined sensation, called photochromic and photo-heterochromic interval in colors (see Sec. 7 in Vol. 1), and now *heterotactile interval*.

According to the results of our study, the three fundamental tactile sensations mentioned above should not be considered as belonging to the same functional level, unlike what is usually accepted. In both vision and touch (and the same is true for hearing), the most primitive sensation can be separated from the more defined sensations or sensory qualities. In vision we have as first sensation, simple colorless luminosity, and as a more defined sensation, the different colors. In touch we have as initial or elementary tactile sensation, simple contact (pressure), analogous to simple colorless luminosity, and in another more elaborate range are the tactile sensations of pain and temperature. Since the alteration of any system is global, these two types of sensory activity, initial and more defined sensation, are affected in different ways due to their peculiar excitability characteristics.

The behavior of the initial sensation (contact) in our brain-injured patients is established in the previous sections on general excitability, either by electrical or mechanical stimulation. In addition to the considerable deficit of excitability, there is a spatial disorder that impedes spatial localization of the elementary sensation of contact. Thus, there is a pronounced hypoesthesia, the degree of which depends on the intensity of the cortical lesion, and is partly attenuated by facilitation. The deficit of excitability is clearly defined, so it is inappropriate and arbitrary to attribute the hypoesthesia to attention disorders, as [Goldstein and Gelb \(1919\)](#) have assumed for their Schneider case in order to avoid the serious theoretical obstacle of impairment of sensibility in both halves of the body when there is only a unilateral lesion (see Sec. 18.3).

The hypoesthesia also includes the tactile sensations of pain and temperature (hot and cold), so that a pinprick as well as the application of heat or cold, when not very intense, may be felt only as an unclear contact without any trace of spatial localization. To reach the physiological level of well-defined sensations, the stimulation must be more intense and so, whereas a normal subject perceives a tactile quality of remarkable intensity, the patients describe only a slight qualitative sensation. In short, pain and temperature are less perceived due to a reduced sensitivity. True sensory qualities (well-defined sensations) are accompanied by a certain spatial localization, which is diffuse (“irradiated”) when the sensation is weak, and more perfectly localized, in extension and place, when the sensation is very intense.

The most remarkable aspect of the dynamic reduction of sensory qualities is a peculiar decomposition (disaggregation) of them, thus appearing the heterotactile interval. A sufficiently intense thermal or painful stimulus is chronologically decomposed into two stages: a first stage of simple contact sensation and a second stage of sensation of the quality in question. In this way, we obtain what we call *tango-algic interval* for pain and *tango-thermal interval* for temperature, thus, the fundamental experience for tactile sensory qualities is similar to the one studied for colors. This asynchrony makes it possible to separate the true sensory qualities, pain and temperature, from the primitive tactile sensation of contact. Moreover, they correspond to spatial categories of very different development in our pathological cases, since only the true sensory quality shows a certain localization. In short, with adequate stimulation, the sensory quality is not established from the beginning, but is preceded by a primitive tactile sensation devoid of qualitative differentiation and spatial localization.

This type of asynchrony through the aforementioned heterotactile interval is of great theoretical significance in questioning the physiological individuality of sensory qualities, since the interval shows the existence of a primitive component from which algic and thermal qualities evolve and differentiate; just as the simple primitive luminosity serves as a starting point for chromatic qualities. Dynamic reduction, through the asynchrony of functions, is therefore the keystone for approaching the analysis of seemingly simple or individual functions. From tests in our brain-injured patients, it is shown that there are really no specific or independent functions (whether sensory qualities, spatial functions or otherwise), but rather, the different sensory levels (many of them usually considered irreducible to other elements) appear as the result of a progressive evolution and organization developed from a very primitive element. It is simply conventional to try to identify individualized functions throughout this development. More properly, it is only possible to consider different degrees of differentiation or of sensory organization; not the existence of different individual functions.

## 19.2 Pressure and pain

The data previously shown on the state of general excitability in touch, already suggest that there is a notable hypoesthesia extended to the whole body and on both halves approximately equally or with minor differences. The intensity of hypoesthesia is related

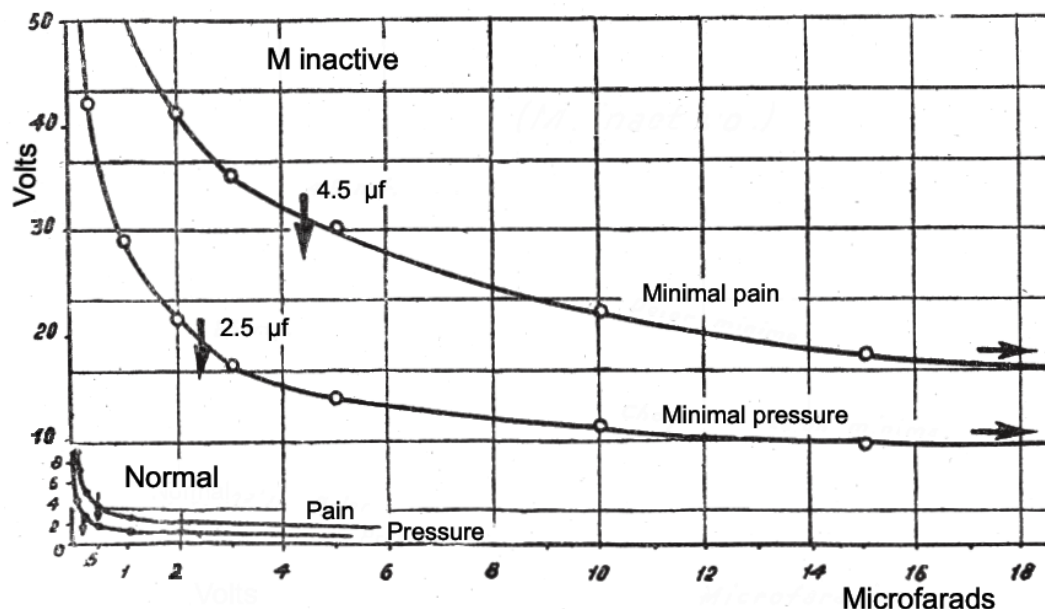
to the magnitude of the brain lesion; thus, it is severe in M and moderate in T. Especially in case M, it is necessary to examine the subject in a fully inactive state in order to uncover the whole magnitude of the defect. It is noteworthy that despite the deficit in tactile sensitivity (in stimulus intensity and timing), these subjects were never spontaneously aware of this disorder nor of the significant lag that leads to a considerable alteration in stimulus localization. This unawareness is due both to the compensatory action of facilitation and to the habit of paying attention only to stimuli that are well perceived, discarding all others, because they are too weak or cause a fragmentary perception.

As already mentioned, the sensation of contact or pressure can be considered a *primal activity of the tactile function*, like simple luminosity in vision. Whereas for some authors this sensation is completely independent of the other two qualities (pain and temperature), other authors, especially Goldscheider (1917), accept a gradual transition between pressure and pain, in such a way these sensations are extreme modalities of the same function. Therefore, Goldscheider (1917, 1925) admits that a touch on the skin can provide either pressure or pain depending on the intensity of the stimulation. This is equivalent to deny the existence of different specific points for pressure and for pain as Frey maintains (Frey 1910, 1913, 196/1917, 1928). Goldscheider (1917, 1925) admits the existence of certain points where weak stimulation produces only pressure, and more intense stimulation causes pain; and in analogy with the photochromic interval of colors, he mentions a tango-algic interval. This interval, originated by the duality of the response according to the stimulation energy, seems to be present even in regions where only pain points are usually admitted (e.g., the cornea). Thus, threshold excitations cause only contact sensation according to Kiesow (1924). Likewise, Hoefler and Kohlrausch (1924) report that under threshold electrical excitation at pain points, they always obtain an initial sensation of contact. It has also been observed that in sensory adaptation to pain, when pain disappears, only a sensation of pressure or simple contact remains.

In our cases, mainly in the inactive subject M, it is repeatedly proven that stimuli that are clearly painful for a normal subject are only perceived as contact, and when a more intense stimulus triggers pain, the sensation goes through a previous phase of contact. The tango-algic interval is extremely marked and when a skin site is stimulated, the sensory interval that appears is so large that it is very easy to reveal it. A prick of a certain intensity with a fine needle, if it is brief, only gives rise to a perception of contact, and if the time of application is prolonged, the pain phase is reached. With facilitation by muscular effort, the interval also exists but it is not so long. Analogously in subject T. Cutaneous chronaxie determinations in a normal subject already show different values for pressure and for pain. Bourguignon (1929, 1933) finds that the chronaxie for the so-called “superficial light pain” is twice that for pressure (or pushing); and for “deep blunt pain” it is up to four times that for pressure. By determining with electrical stimulation the curves for pressure and pain in the two extreme states of subject M and subject T, as well as in the normal comparison subject, it is found that the greater the deficit of excitability, the greater the tango-algic interval. The tests are performed, as in general excitability, on the middle of the lower lip. Since a 0.5 cm diameter electrode is used, the stimulation is on surface and not on selected contact points, but this does not prevent distinguishing between pressure and pain sensations in relation to the stimulation energy.

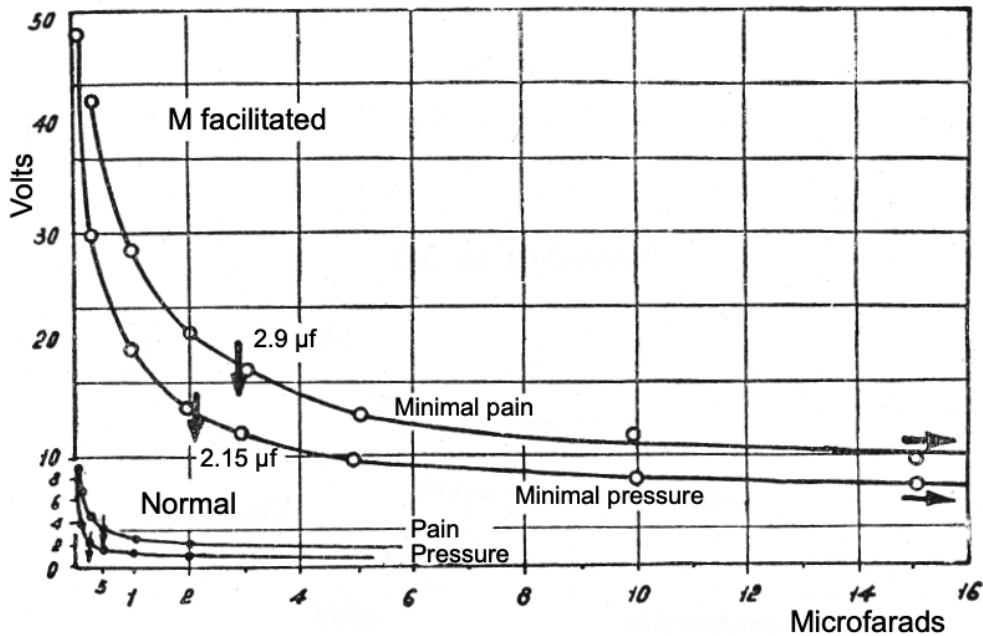
The strength-duration curves for pressure and pain sensations in Fig. 91 show the large difference between subject M in the inactive state and the normal subject. The curves of M are markedly higher, corresponding to a very large rheobase increase, they also have less curvature than in the normal case due to the significant increase in chronaxie, and they are also much farther apart between them than in the normal subject, which means a considerable enlargement of the tango-algic interval. In electrical stimulation, the sensation of pressure corresponds to a sensation of very slight shock due to the passage of current when the capacitors are discharged. The sensation of pain corresponds to a very slight and superficial pain but clearly distinguishable from the previous sensation.

In these tests, the chronaxie capacitances in the normal subject for pressure and pain are  $0.25 \mu\text{f}$  and  $0.4 \mu\text{f}$  respectively. In subject M in the inactive state, the respective values are  $2.5 \mu\text{f}$  and  $4.5 \mu\text{f}$ . Thus, the two values in M inactive are about ten times greater than in the normal subject. Hence, the relative pressure-pain ratio remains unchanged in the pathological case, i.e., in both M and normal, the chronaxie for pain is almost twice the chronaxie for pressure. As for the absolute values, the difference is very large, since in the normal subject the variation in chronaxie is of tenths whereas in M inactive it is of several units. In short, there is pathologically an extremely wide tango-algic interval, which explains that in the stimulation of a same cutaneous location, pressure and pain are obtained with great separation of time ( $\mu\text{f}$ ) or intensity (volts), the latter due to the fact that the rheobases suffer an increase parallel to the chronaxies both in electrical and mechanical excitation.

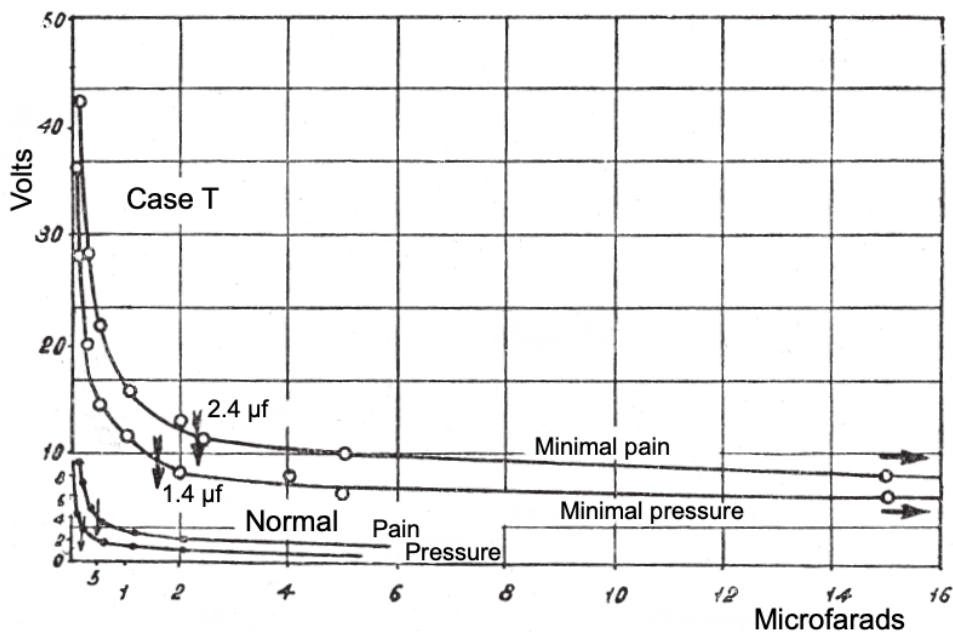


**Figure 91.** Strength-duration curves for pressure and pain in the inactive subject M (upper curves) and for the normal subject (lower curves). Electrical stimulation on the midline of the lower lip. Cathode 5 mm in diameter. Note the large elevation of the curves in subject M and the large separation between the two curves compared to the normal subject. Note also the different chronaxie values.

For subject M under facilitation by strong muscular effort, the above considerations are valid. This case (Fig. 92) corresponds to an intermediate physiological level between the inactive state and the normal subject, therefore the tango-algic interval now has an intermediate amplitude. The pressure and pain curves are lower than in the inactive state and are also closer together, but the interval still has a considerable amplitude.



**Figure 92.** Strength-duration curves for pressure and pain in subject M under facilitation (maximum contraction of the entire musculature). Other conditions as in Fig. 91.



**Figure 93.** Strength-duration curves for pressure and pain in subject T. Same conditions as in Figs. 91. Note the differences with the two aforementioned cases.

Finally, in subject T the interval is narrower as shown by the closer proximity of both curves (Fig. 93), which correspond to much faster chronaxies than in the two mentioned states of M.

In these cases, the amplitude of the interval is closely related to the level of excitability, i.e., to the characteristics of the pressure and pain curves. The ratio between the values for pressure and pain remains approximately constant in all cases, including the normal subject. However, the absolute value of the differences increases as the deficit of excitability is greater. Table 22 shows the values corresponding to Figs. 91, 92 and 93.

**Table 22.** Values corresponding to Figs. 9, 10 and 11.

	<i>Pressure</i>		<i>Pain</i>	
	Chronaxie ( $\mu$ f)	Rheobase (volts)	Chronaxie ( $\mu$ f)	Rheobase (volts)
M inactive .....	2.5	9.5	4.5	16
M under facilitation	2.1	6.5	2.9	9
T .....	1.4	4.5	2.4	5.6
Normal .....	0.25	1.2	0.4	1.6

One of the most remarkable features accompanying the sensation of pressure and pain in our subjects is, as stated, the alteration of spatial localization. Pressure or simple contact does not cause any sensation of localization, and a pinpoint pressure causes a kind of cutaneous irradiation of indeterminate localization on the surface of the skin. Rudiments of spatial localization appear only with sensory qualities, and in the case of pain, as soon as the sensation of pain begins, even if it is very slight, a certain localization appears. But this one is quite deviated from the stimulus application site; in addition, the mentioned irradiance gradually decreases, although it is still prominent. The more intense the pain (e.g. in a pinprick), the more normal the spatial structure of the perceived stimulus. In this way both deviation and irradiation gradually decrease according to certain laws. A pinprick causing almost unbearable pain is necessary for the localization to become completely normal, at least in subject M inactive, who presents maximum functional lag (asynchrony). This question will be studied in a separate chapter, and now it suffices to mention the general characteristics of pressure and pain sensations. Therefore, in addition to the huge enlargement of the tango-algic interval, there is a sensory-spatial interval due to the lag or asynchrony in the nervous centers involved in the localization mechanism. This last interval does not exist in the normal subject since any tactile stimulus, no matter how weak or brief, is always perfectly localized. This fact is completely new, without any precedent in functional normality.

We must mention the special type of pain admitted by Piéron (1935) consisting of a stinging sensation (piqûre in French). Indeed, it is a tactile sensation close to pain but extremely spatially circumscribed, producing a very slight pain of pinpoint character. Due to the considerable spatial irradiation in our pathological cases, such a stinging sensation is completely abolished since the sensation of minimal or incipient pain occurs in a very spatially diffuse manner. This defect is detected in the clinical test of distinguishing

between pinhead contact and pinpoint contact. This distinction based on sting-like perception is impossible in subject M inactive, within rather wide limits, even if a certain degree of pain occurs with the corresponding stimulus. The sting is thus essentially a sensory complex of mild pain according to a certain spatial modality, and there is no reason to consider it an individualized function within the sensation of pain. More details on this issue are given in the chapter on spatial localization.

As for the excitation conditions for pressure and pain, it should be noted that strong pain is very easily obtained by iterative stimulation of a mere pressure sensation. In this way, not only the wide sensory gap of the tango-algic interval disappears, but an intense, almost unbearable pain sensation is reached, which is very close to the normal localization. Already in the normal subject, the sensation of pain shows some aptitude to iterative stimulation (Schriever and Cebulla 1938, Altenburger 1933, Piéron 1935) but in our cases, particularly in M, it is greatly increased. In relation to this behavior are the old observations of Egger (1899), who points out that no tabetic analgesia withstands stimulation by means of a needle fixed to a tuning fork providing 60 pulses per second. Pain is either triggered or recruited by summation in time (latent addition), as in our brain-injured patients.

Analogous to iterative summation, although less effective, is facilitation by muscular effort. Thus, a stimulus at the pressure threshold is transformed into a sensation of pain by means of a vigorous muscular effort, overcoming the tango-algic interval and sometimes reaching a sensation of accentuated pain if the effort is very intense.

Finally, as for sensory adaptation to pain in subject M, this is evidenced within the limits already indicated when discussing tactile adaptation in general. With stimulus applied continuously with just enough intensity to produce pain, the pain ceases after half a minute, and only the sensation of pressure remains, which in turn disappears after about half a minute or a little less. This adaptation that excludes algic sensation, occurs very easily, probably due to the increased fatigability in these pathological cases. During adaptation, the tango-algic interval occurs in the opposite direction.

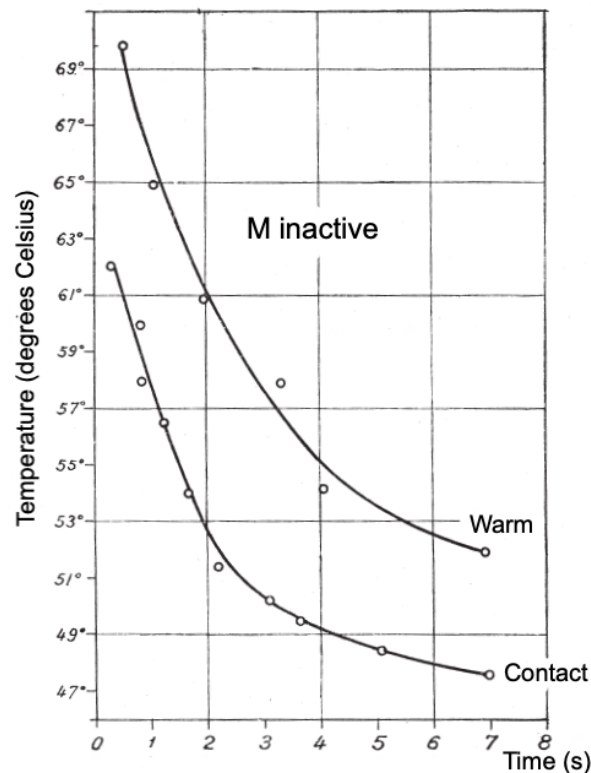
From all that has been said so far, it follows that pressure and pain are closely related physiologically, giving rise to the idea that pain is a differentiated modality derived from pressure.

### 19.3 Thermal sensations

Thermal sensations show in our cases a full parallelism with pressure-pain sensation, namely, a deficit of excitability with required increase in intensity and time of stimulation, and also a tango-thermal interval. The deficit is related to the magnitude of the brain lesion and the conditions of the central nervous system by facilitation.

When touching a cold or hot object, only contact is felt, without any thermal sensation when the temperature of the object is not excessively far from the neutral point of thermal sensation. This occurs in both subject M and subject T, although with the difference due to the different level of excitability in each subject. If the temperature of the touched object is very different from the body temperature, thermal sensation finally

occurs but it is clearly observed that in the slow sensory development, the thermal sensation is preceded by one of simple contact or pressure. This shows the existence of the tango-thermal interval, which does not seem to exist in a normal subject. In our cases it is clearly obtained by both the difference in intensity thresholds (higher for temperature than for pressure) and the different speed of reaction (slower for temperature). The heterotactile interval is thus common to pain and temperature, both sensory qualities developing from the primitive pressure sensation.

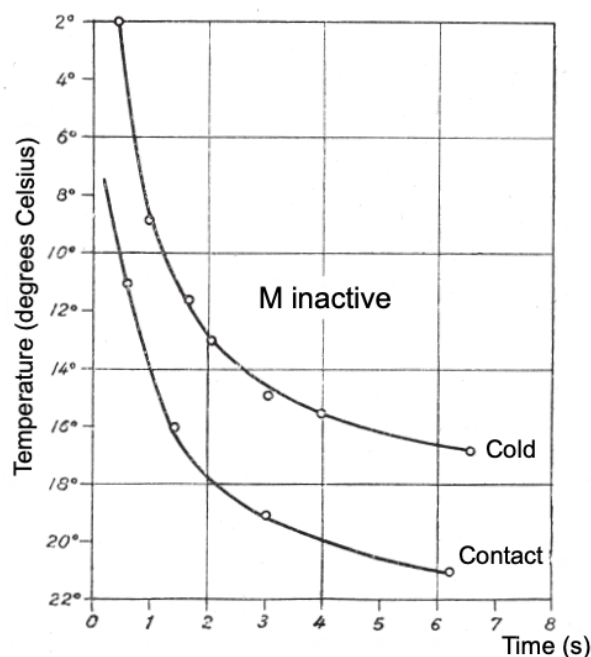


**Figure 94.** Strength-duration curves for contact sensation and warm sensation in subject M inactive. Note the tango-thermal interval. Stimulation by applying a test tube with hot water over the midline of the forehead, over a surface of approximately one square centimeter and avoiding pressure. The curves correspond to the average values of a series of tests.

There is no agreement among authors on how to obtain thermal sensation by electrical stimulation. Some, like [Schriever \(1929\)](#), consider to have achieved it for cold. [Bourguignon \(1929, 1933\)](#) as well as [Schriever \(1929\)](#) found that the chronaxie for temperature is eight to ten times the chronaxie for pressure and about four times the chronaxie for slight surface pain. Thus, temperature is by far the tactile quality with the highest chronaxie. We will now limit ourselves to stimulation with thermal, not electrical, stimulus. The tests are performed with water at different temperatures in a test tube, applying its base on a small area of the skin. Other ways of stimulation, water drops, radiant heat, etc., serve to complete the study. In this way, it is not difficult to get an idea of the sensitivity to temperature in its heat and cold modes, and strength-duration curves showing the state of the function can be determined.



Figures 94 and 95 show the results obtained in subject M inactive. A same thermal stimulus gives rise to two different sensations corresponding to respective curves of different excitability. The higher excitability is for pressure sensation (simple contact), the lower excitability is for thermal sensation, either warm or cold. This duplicity with the same stimulus shows the existence of the tango-thermal interval. In addition, the alteration of excitability for cold and for warm are of the same quantitative order, and the range of the pressure-temperature interval is about 5° Celsius. With facilitation (by strong muscular effort), the interval is much smaller, only 1°C or 2° C between contact sensation and warm sensation. The interval is also found in subject T, although much smaller than in M under facilitation. Therefore, the behavior is similar to that indicated for the tango-algic interval in the cases considered.



**Figure 95.** Strength-duration curves for contact sensation and cold sensation in subject M inactive. Note the tango-thermal interval. Stimulation conditions as in Figure 94.

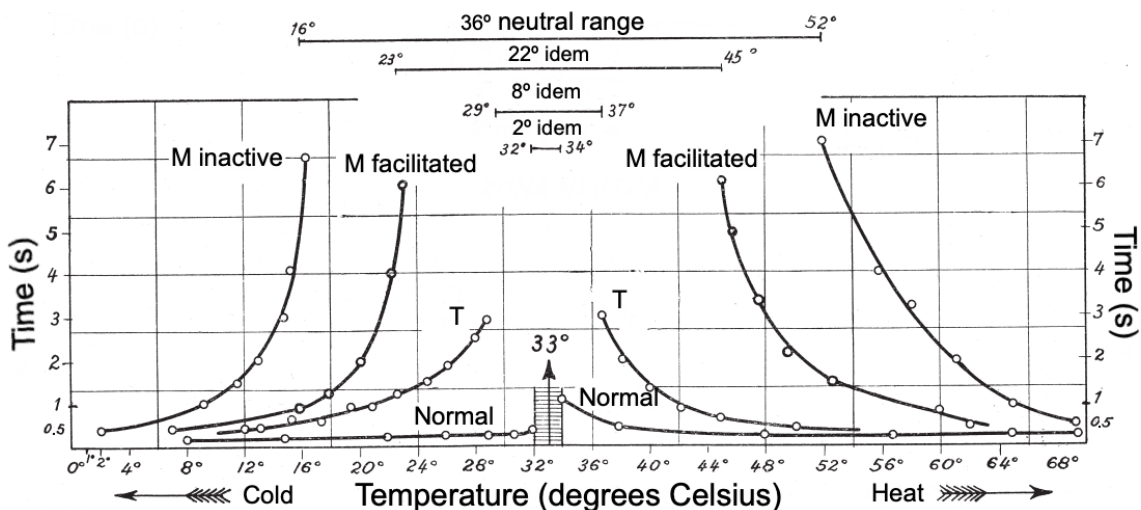
In all these cases, when the intensity of the stimulus is not high enough, the thermal sensation is not produced, only the first contact sensation is obtained, that is, the aforementioned interval is not overcome. Moreover, if the thermal intensity is even lower, although the wall of the test tube is resting on the forehead of the subject, not even the sensation of contact is obtained. Thus, the thermal stimulus is what determines the sensation of contact, not the pressure (contact) of the test tube, which only serves (especially in inactive M) to transmit heat. In order to verify this remarkable finding, we proceeded to perform further tests in the absence of any possible pressure stimulus. For this purpose, the test using drops of water at a certain temperature on the skin is easy to perform. The drops, due to their very low weight, do not act as a pressure stimulus in these subjects. In this way, the tango-thermal interval can be obtained for a single drop at a certain temperature, since, as we know, the development of sensation is very slow in

subject M. If the temperature of the drop is reduced, only contact sensation is obtained, and below that temperature it is no longer possible to achieve any pressure sensation even if drop after drop is deposited on the skin.

Another even more conclusive test involves the use of thermal radiation. By directing radiant heat from an incandescent electrical resistor onto a certain surface of the skin, it is possible to determine a distance at which the thermal flux on the skin is felt as a contact without any heat. It is clear that lacking all mechanical contact, the sensation of contact (pressure) must be attributed to the sensory degradation suffered by the pure thermal sensation because of the heterotactile interval.

In addition to the important phenomenon described on the tango-thermal interval, there is a characteristic disorder of temperature perception which is the considerable enlargement of the neutral temperature range (intermediate between cold and warm) to which the surface of the human body is adapted and which does not give rise to thermal sensations. In the normal subject, this range is about two degrees, and in the most usual conditions it ranges from 32°C to 34°C. Enlargement of the neutral range was first reported by [Head and Riddoch \(1920\)](#) in tactile disorders due to parietal cortical lesion. Head found decreased thermal differential sensitivity, i.e. increased thermal differential threshold, and enlargement of the neutral range as a consequence of also increased absolute threshold for cold and heat. The authors give as a general explanation a deficit of discrimination, without mentioning at all nervous excitability.

Figure 96 shows the variations undergone by the neutral range in the various cases considered. It can be seen that the widening of the range is related to the deficit in nervous excitability. The thresholds for cold and heat increase, and also their useful time, as we know. Whereas the neutral range in the normal subject is about 2° C, in M inactive, the most extreme case, the range reaches 36° C, i.e. 18 times higher.



**Figure 96.** Arrangement of the strength-duration curves for cold and heat allowing us to observe the enlargement of the neutral range as a function of the deficit of excitability, related to the magnitude of the lesion. Curves for M inactive, M under facilitation by strong muscular effort, T inactive and normal subject (data for the latter according to [Piéron \(1935\)](#)). Stimulation on the forehead with a thermal surface of one square centimeter.

It is also observed that the alteration is the same for cold and heat, the corresponding curves being symmetrically located with respect to the central point of the neutral range (33°C). A comparison of the different threshold values shows that the threshold increases considerably in subject M, both when inactive and under facilitation, but especially in the former state. This is consistent with the profound deficit suffered by a function much less sensitive than pressure or pain. In subject M (of greater brain deficit) the disorder in temperature sensation is very pronounced, since functions suffer greater disturbance as they are less excitable. Thus, within the tactile sensations, those of temperature show to be the most intensely altered.

The values indicated by the curves correspond to laboratory experiences somewhat distant from ordinary life conditions. For a normal subject, a temperature of 50°C applied to a very small surface of the skin is perceived as heat after about 1/3 s, whereas in subject M inactive, it must be about 52°C to feel heat after a few seconds. After this period of time, the normal subject would feel an acute sensation of strong heat, while M is at the beginning of the thermal sensation, i.e., at the sensation threshold. Thus, it must be concluded that there is an acute thermal hypoaesthesia in M. However, in the conditions of ordinary life when touching objects, this does not occur so pronouncedly since the contact usually takes place on larger surfaces, and the thermal deficit is corrected significantly by the summative action of the surface. In the experiment with the test tube filled with hot water, if instead of applying the bottom of the tube on the forehead or on a cheek on a surface of one square centimeter or less, the tube is applied laterally over its entire length, the values obtained in the inactive state become equal or even lower than those of the curve in the facilitated state when the surface is one square centimeter. In ordinary life, the thermal defect is greatly reduced, both by the more or less intense muscular effort that the subject exerts spontaneously and which acts as facilitation, and by the action of the surface, thus achieving two types of summation that significantly compensate for the defect.

The spatial properties of thermal sensations are analogous to those discussed in the case of pain. In the tango-thermal interval, the first sensation of simple contact is not accompanied by spatial localization and only a fleeting sensation more or less extensive is perceived in some part of the body impossible to specify. But as soon as the second phase is reached, thermal sensations become localized and irradiation tends to decrease. As in the case of pain, very intense thermal sensations are required for localization to be normal, i.e., without irradiation, and without deviation with respect to the stimulation point, as we shall study later on. In short, only pain and temperature have a spatial character, i.e., localization of the corresponding stimulus, and only at high intensities do they become close to normal.

Thermal adaptation and differential threshold have not been studied systematically enough to obtain usable data. In addition, a number of circumstances make this type of testing difficult. Nevertheless, it can be said that *thermal differential sensitivity* in M inactive is severely impaired. The intensity threshold for the sensation of heat and cold is markedly increased, as we have seen, but so is the differential threshold. Whereas the normal subject can appreciate differences of fractions of a degree between certain

temperatures, subject M needs up to 2°C or 3°C in the most favorable conditions (certain thermal ranges in which differential sensitivity tends to be better). As for thermal adaptation, this occurs more easily than in the normal subject due to the enlargement of the neutral range, and may also be due to increased sensory fatigability. More complete experiences would be needed to determine more precisely these phenomena.

## 20 Organization of the tactile sensations

### 20.1 Criticism of the so-called “tactile dissociations”

Neuroanatomical concepts often find an important theoretical and practical application in the tactile system. Anatomical specificity consists in admitting certain receptors with a well-determined function (tactile points), as well as different conduction pathways and terminal centers also linked to specific tactile activities. This anatomical independence of the different functions should be particularly evident in the multiple “clinical dissociations” of the tactile functions according to the type of anatomical lesion. Therefore, tactile dissociations are considered very important in clinical diagnosis.

This view certainly responds to clinical needs, but it should not be set up as a systematic theory or general law. It constitutes only an adequate approximation for certain particular cases, whereas it fails completely in the interpretation of many others. A revision of this approach began with the studies of [Head and Riddoch \(1920\)](#) and [Head et al. \(1920\)](#), who reduces all tactile activity to only two types of sensitivity, namely, primitive or protopathic and more differentiated or epicritic. But, apart from this, the anatomical aspect remains quite firm in this new theory since protopathic and epicritic pathways and centers are assumed. The organization of functions is simplified, but the hypothesis of anatomical specificity is preserved for the two types of sensitivity.

Later on, [Foerster \(1936\)](#), an author closely linked to the theory of localization and anatomical specificities, was compelled, in order to interpret many clinical observations, to accept a *functional principle* in addition to the principle of localization. Thus, it can be said that when the tactile system is affected, a constant regularity is shown in the way its different functions are altered, and this occurs independently of the anatomical type of lesion. The sensory quality that is always the most affected is temperature followed by pain and finally pressure. When a lesion significantly impairs tactile activity, and after a certain time functional recovery occurs, the following order of appearance of the qualities is always observed: pressure, pain and finally temperature. However, [Foerster \(1936\)](#), a consistent localist, interprets this functional order of affection by resorting either to diffuse biological hypotheses on a greater firmness of the most basic functions, or by means of the anatomical argument of the supposed greater spread of the anatomical elements corresponding to the best preserved functions. This argumentation in order to maintain the approach of anatomical specificity is not very resistant to criticism, but it is not appropriate here to deal with this issue.

Finally, we find a new approach in [Stein and Weizsäcker \(1927, 1928\)](#), much more explicit, coherent and rational than the previous ones. These authors consider the tactile

system as a functional unit that has a number of activities, not as much because it contains different sensory elements, as because it is activated in many different ways. Such considerations lead them to refute in depth the dissociated alterations of sensitivity, as well as the usual teaching of clinical neurology on sensitivity as set forth in classic treatises such as that of [Dejerine and Dejerine-Klumpke \(1914\)](#) among others. For them, it is absurd to seek for each new function a special organ, i. e. a specific pathway or a new anatomical-physiological element. Due to the special *change of function* that occurs in lesions of the tactile system, it results that while the sensitivity is suitable for a certain function, it remains unable for others. It is therefore a futile effort to try to infer from these changes (dissociations) conclusions about the autonomy of receptors and diverse specific pathways. In short, these authors establish that when there is a certain disorder of the tactile system, its excitability is modified, and the changes in the response do not correspond to anatomical dissociations of specific elements or neuronal regions for a certain function, but are due to physiological disorders of the whole tactile system, which by altering its excitability, disturbs the capability of functional differentiation.

These theories of the aforementioned authors show a growing tendency to replace the old and classical tactile dissociations (according to multiple anatomical specificities) by more functional or physiological conceptions that tend to simplify the problem, either by reducing the number of sensitivities ([Head and Riddoch 1920](#), [Head et al. 1920](#)) or by establishing a constant sequence of alterations ([Foerster 1936](#)), and finally, by focusing the problem on the excitability process of a functionally unitary tactile system ([Stein and Weizsäcker 1927, 1928](#)). This latter approach is thus the most radically opposed to the classical theory of anatomical dissociations. For Head et al, sensitivity is either primitive and weakly differentiated (protopathic), or more discriminative and evolved (epicritic). For Foerster, there is also a sequence in the evolution of the alteration, and finally, for Stein and Weizsäcker, the principle of functional alteration (type of differentiation, sequence in the evolution, etc.) is made to depend simply on the excitability disorder of the tactile system, without resorting to further anatomical considerations as is still the case for the previous authors.

Taking into account that in normal situation there are already very different chronaxies for the different sensory qualities, Foerster's functional order of alteration is easily explained. Thus, the less excitable functions (of higher chronaxie) are more easily excluded. This does not mean independent alteration of some functions with respect to others since the alteration of excitability is always global. It is therefore not possible to obtain absolute dissociations, but only relative ones. Some functions suffer more than others, but all of them are affected to some degree, as shown in the cases studied here. The alleged absolute dissociations are then more apparent than real, and they result from a purely qualitative examination. By making quantitative measurements, as in our cases, and determining the level of excitability for all types of activities, the global character of the disorder is demonstrated, although diversely manifested in the different functions, thus giving rise to the dynamic reduction that we already know. The issue of tactile dissociations and, in general, of all types of functional dissociations, acquires a meaning quite different from that usually accepted.

## 20.2 Organization of the tactile sensations

The dynamic reduction in tactile sensitivity presents several aspects in our two patients that should be remembered. Vibratory sensitivity is greatly reduced due to a great loss of reaction speed. Pressure, pain and temperature show a remarkable deficit, more pronounced in pain and especially in temperature. In addition, the dynamic disorder shows very special characteristics due to functional asynchrony, giving rise to tango-algic and tango-thermal intervals, which are clearly evidenced. To this must be also added the different type of spatial localization that accompanies each phase of these intervals. As for temperature, it should not go unnoticed that the considerable enlargement of the neutral range takes place at the expense of an equal deficit for heat and cold. All these phenomena lead us not only to a negative criticism of tactile dissociations, as already mentioned, but also, on the positive side, to establish a *new concept about tactile organization*, mainly concerning tactile sensory qualities.

We must consider the system as a functional unit which, according to the various modalities of the central excitation process, gives rise to the different activities. As for the sensory qualities of tactile sensitivity, an important problem of physiological organization arises with the heterotactile interval. As already mentioned, pressure on one side, and pain and temperature on the other, are shown to belong to very different sensory levels. Not only do they have different chronaxies, but also phenomenic features with very different evolution, which is of great importance for classifying these tactile modalities and getting an idea of their physiological organization. These distinguishing features are the heterotactile interval and spatial localization.

Pressure is the specific elementary activity (*tangibility*) in the sense of touch, like luminosity in vision and sonority in hearing, but not as a quality per se. The meaning of quality should be reserved for pain and temperature. This is analogous to the difference between luminosity and colors, since these also show in our cases a physiological organization quite different from luminosity, such as photochromic interval and spatial phases (e.g. colorless when losing motion perception and appearance of hue when motion appears).

To determine the meaning of these two *true qualities* (pain and temperature), one must examine: specificity, functional nexus with the elementary sensitivity and origin of these qualities; all aspects of a same problem. If in order to accept the specificity of a given function one adopts the usual principle of physiological individuality, i.e. the non-dissociable character of the function, one must then pronounce against the specificity of the algic and thermal tactile sensations, due to the aforementioned heterotactile interval. Particularly in the case of temperature, it is unquestionable that a pure thermal stimulus, i.e. devoid of any mechanical pressure (e.g. thermal radiation), can be reduced to a simple sensation of contact under appropriate conditions. This non-individuality of the true qualities results in the acceptance of a functional nexus between the elementary activity of simple pressure and the qualities that arise through a process of differentiation from the primitive function. That nexus leads to examine the possible origin of pain and temperature. Since certain functions can be "isolated", they can be assigned at least a relative specificity, since it could not be absolute as stated above. This relativity allows

us to suppose some evolutionary process by which the qualities we are concerned with arise. If this were not the case, we would have to accept them as irreducible and primary manifestations, as undoubtedly is luminosity in vision and tangibility (simple pressure) in touch. But a process of progressive organization always adds something new, and the true qualities would participate simultaneously of primitive function and evolutionary function, hence a relative specificity is assigned to them. This type of specificity seems to be different for pain and for temperature. In fact, the gradual transition from pressure (primary manifestation) to pain (secondary manifestation) seems phenomenologically very clear and natural, as it occurs in the gray scale or in the transition from weak light to bright light. However, it is very difficult for temperature to find such a type of transition. Therefore, thermal specificity seems much more evident than pain specificity. Moreover, thermal specificity is not entirely homogeneous since it includes cold and heat, admitted by many authors as independent qualities. In this respect, it is observed in our cases that both thermal modalities undergo an identical alteration (producing an enlargement of the neutral region by an equal deficit for cold and heat) which leads to incline the opinion towards the unity of the thermal quality and not its duality, as some authors (Hahn 1928, Goldscheider 1886) believe.

As for the origin of the thermal quality, Bourguignon (1929, 1933), in a study on tactile chronaxies, makes the bold hypothesis of supposing that the thermal sensation is the result of a combination of the sensation of tingling and of pressure, or at any rate, as a differentiating effect from both. If this were admitted, a rather gradual transition between primordial tactile sensation and thermal quality would result, but at present there is insufficient basis for this hypothesis, and any relationship between primordial sensation and quality is limited to the phases of the tango-thermal interval.

Concerning the so-called fine touch as another modality whose alteration is easier and of longer duration, it should be noted that the persistent deficit of sensitivity to light pressure and to fine contact discrimination only means an increase in absolute and differential thresholds, which corresponds to the basic excitability disorder (increased rheobase). Likewise, the deficit in the appreciation of very small time intervals (increased chronaxia and therefore refractory period) leads to the inability to perceive high frequency vibrations, even in cases of mild brain lesion. There is thus no reason to think that fine touch deserves a special place as a differentiated function.

All these considerations sufficiently indicate the new dynamic approach, common to any type of function (not only sensory qualities) and valid for any sensory system.

With respect to the sensory specificity of the peripheral receptors (whose current theory is totally opposed to the dynamic considerations maintained here), such specificity would be rather a preliminary to the dynamic action of the centers. This applies to colors as well as to tactile sensations and the rest of the more complex sensory functions.

As a general conclusion, it could be said that instead of having an anatomical-physiological individuality for the various functions (giving rise to well-defined absolute dissociations depending on the nature of the lesion), there is a global disorder and neither functional individuality nor isolated disorders can be considered. Dissociations, if they exist, would in any case be relative. The functions arise according to the degree of organization of the sensory field determined by the physiological level of excitability.



The development of this organization makes it possible to establish different physiological levels, and thus a dynamic classification into two levels of the tactile functions we have discussed. At an elementary level we have tangibility (pressure) as an elementary function, and at a higher level, the true qualities of pain and temperature.

# Tactile space

## 21 Tactile localization

### 21.1 Spatial disorder

So far, knowledge about spatial localization disorder is scarce, both in its manifestations and in the physiological mechanism of the pathological process. In certain lesions of the parietal cortex (tactile projection area), the usual clinical observation comes to establish that, for example, the patient localizes the stimulus with error or deviation, that he mistakes the place of stimulus application, that he is unable to pay attention, etc. In more severe cases, and also rarer, it has been observed that the patient is barely able to localize the stimuli, no matter how intense they may be. He knows that he is touched somewhere on his body, but is totally unable to indicate where. This last type of deficiency corresponds to an extreme degree of impairment. In moderate disorders, some irregularity in localization is found, as well as a tendency to irradiation under a point stimulus, indicated by [Head and Riddoch \(1920\)](#), [Head \*et al.\* \(1920\)](#) and later by [Stein \(1928, 1930\)](#) and [Stein and Weizsäcker \(1926\)](#). A point stimulus is localized somewhat uncertainly, and it is perceived as a sensation in a wider region, not as a point. More systematic and precise determinations are lacking.

In a good number of wounded patients with parietal lobe lesions and diverse tactile symptomatology, studied with some accuracy in 1938, we have been able to establish a certain sequence in the localization of stimuli in relation to the severity of the disorder. This sequence, from lesser to greater severity, is as follows. First, the patient makes more errors when pointing at the location than when mentioning it verbally, i.e. naming is better than pointing, because when pointing, the precision must be greater. Second, stimuli are localized more towards the center of the body than they really are (‘proximal deviation’), thus, in the extremities, the localization is perceived more towards their origin, with an evident deviation of a few centimeters. An opposite result, i.e. a distal deviation, is very rarely found, and therefore proximal deviation can be accepted as a rule. Third, the patient is only able to vaguely point at the place where the stimulus has been felt, merely indicating with one hand a region of the body, but keeping the hand in the air without deciding to specify a place (localization in the air). Thus, some patients indicate large areas of their body without giving further details. Similar to this state but much more disturbed is the fourth stage in which there is a complete loss of localization of tactile stimuli and the subject says “I am pricked, but I do not know where”. We have observed

this type of response (besides in the two cases studied) only in brain-injured patients in the acute phase, when even very intense stimuli are not able to change the situation. But this complete absence of localization is not usually maintained for a long period, and in patients who survive, after a few days there is a tendency for some rudiment of spatial localization to reappear, especially with very intense or repeated stimuli.

These four stages show that localization does not disappear as a whole; on the contrary, depending on the severity of the disorder, different phases appear which are of great importance for unraveling the mechanism of spatial localization. To the above specifications must still be added the frequent tendency to irradiation under pinpoint stimuli, since the stimulus with the tip of a pin is felt as a tingling of some size, sometimes covering a large part of a limb, a fact already first pointed out by [Head and Riddoch \(1920\)](#) and [Head \*et al.\* \(1920\)](#). We were also able to observe various phenomena qualified by some authors as attention deficit, [“Beachtungsmangel” in German ([Kleist 1934](#))] or lability, fatigue, etc., which further complicate the results when the examination lasts a long time. It should also be noted that the different brain-injured patients we examined presented a global disorder of tactile sensitivity. Spatial functions were more affected than simple qualities, and within the spatial functions, localization was much less impaired than discrimination between two points (Weber), passive movement and posture, the latter two being maximally affected in all cases. To reach a considerable disorder of localization (third or fourth stage), a very deep impairment of the tactile system is necessary. All these data do not go beyond the empirical clinic, however interesting they may be, since a systematic study and particularly a physiological investigation are lacking. These last two aspects are fulfilled in the brain dynamics developed here on the two cases we are dealing with. The significance of the mentioned stages will be discussed later, and now they are only a preliminary to show the *existence of gradations* in localization disorder.

These gradations acquire importance when considering the only existing case (apart from those presented here) with lasting abolition of tactile localization, i.e., the Schneider case of [Goldstein and Gelb \(1919\)](#). Many years after being injured, this subject showed a complete abolition of localization, as he only felt contact without any possible indication of localization, unless certain movements or involuntary muscular jerks intervened, in which case localization was perfect, according to the authors. Despite the detailed study of the phenomenology of the patient, no intermediate forms are described, which in our opinion must necessarily exist, as will be seen in our cases. Even more than in the work on vision, in the second study on touch, the authors focus exclusively on highlighting the great difference when muscular jerks are absent compared to when they are present. These are two extreme states corresponding respectively to a total abolition of spatial functions and to a complete normalization of them (only seemingly according to the authors); and no gradual transition between these two extremes is reported. The change is abrupt and does not allow us to unravel the phenomenology, much less the development of the localization and spatial function in general. This issue (presence of stages with and without muscular action, the latter being only a facilitation), and the fundamental importance of the development of localization and other spatial activities, will be discussed again throughout this chapter.

The general thesis of the two studies by [Goldstein and Gelb \(1918, 1919\)](#) on the Schneider case has been exposed in the first part of this work, as well as the experimental rebuttal of their interpretations together with the rational physiological interpretation of this patient. Although it is not the main purpose of this work to criticize the Schneider case, given his enigmatic symptomatology and antecedents in relation to our cases, it is very necessary to know his characteristics, both for the necessary remarks we shall make and to highlight the essential differences in the method of study and in the results obtained.

As already indicated, patient Schneider also presents some impairment of elementary sensory qualities, although the authors try to play it down and relate it to an attention disorder. Hence, in all the transcriptions of this case, it is said that the qualities were completely intact, which is not the case as stated above. According to the authors, kinesthetic perceptions (muscle, tendon and joint perceptions) show a behavior similar to that of the qualities; in other words, without resorting to any facilitation (muscle jerks, etc.), a passive movement of a joint is clearly perceived, and it can even be appreciated whether it is fast or slow. This statement entails several important contradictions. Perception of the passive movement of a joint is already a spatial function that reveals an advanced degree of spatial organization, which is inconsistent with the general and absolute loss of tactile space that the aforementioned authors postulate. [Stein \(1928, 1930\)](#) and [Stein and Weizsäcker \(1926\)](#), who generally agree with what has been established regarding this patient, note the difficulty that arises in accepting passive movement as intact. They think that it should be as impaired as the other spatial functions, and that the contradictory result indicated would be due to a compensation produced by possible movements such as muscular jerks, even if minimal. Moreover, this preservation of passive movements in contrast to the abolition of the rest of spatial functions is in contradiction with all that is known about the sequence in the alteration of tactile activities, according to the most relevant authors. [Head and Riddoch \(1920\)](#) and [Head \*et al.\* \(1920\)](#) established that position perception and passive movements are affected first and greatly, followed by spatial discrimination (with Weber's compass), and afterwards by spatial localization. The same was established by [Foerster \(1916, 1930\)](#), as well as by us based on brain-injured patients that we have had the possibility to study prior to this research on brain dynamics. This sequence of alterations is based on the dynamic reduction of functions, and it is understandable that since movement, both articular and cutaneous (mobile stimulus on the skin), is a very complex spatial activity, it must necessarily derive from more elementary spatial structures such as localization and even spatial discrimination. It is inconceivable that the more complex structures subsist whereas the elementary ones are abolished. In conclusion, the supposed preservation of the aforementioned movement in the Schneider case is an error of observation, since in an inactive state, it would certainly be abolished under certain conditions of stimulation. It is also a serious lapse on the functional organization of tactile space.

As for tactile localization of stimuli, the study of [Goldstein and Gelb \(1919\)](#) reveals certain phenomena entirely favorable to our interpretation, phenomena not duly taken into account by these authors because they have completely ignored the disorder of nervous

excitability. Localization as well as other spatial functions manifest a radical change depending on whether muscular contractions are present or the subject is inactive.

These authors find: 1. Even in the inactive state, if the stimulus is applied for a long time, the subject ends up localizing it. But they think this is due to the subject's inability to remain immobile under prolonged stimulation. In our opinion, the localization is due to the action of the stimulation time which in this patient, as in ours, is greatly increased (increased chronaxie). 2. Even in the case of allowing the patient muscular jerks (state of moderate facilitation), they note as a remarkable fact that they must use very strong stimuli for the patient to be able to localize them, much stronger than when the patient is only asked to say whether he has been touched or not. It is evident, although the authors have not noticed it, that simple contact and localization correspond to stimuli of different intensities (because of asynchrony) and that even with muscular jerks, such separation of excitabilities is patent. They try to attribute these singularities to attention deficit, but since attention would also be impaired for simple contact without localization, the explanation is not clear. 3. Even when there are muscular jerks, the stimulus must last for a certain time to be localized. If this time is too short, localization is impossible. In this respect, the authors think that the patient does not have enough time to perform well the muscular mechanism; but taking into account the probable disorder of excitability, it is easy to understand that even with a moderate facilitation chronaxie is still increased. Thus, in order to achieve a complex function such as localization, a certain intensity of the stimulus and a certain time of excitation are necessary, both increased in relation to the values of a normal subject.

In observation 1, it was argued that in the inactive state, if the stimulus lasted long enough, it was finally localized, the authors admitting that it was impossible for the patient to remain still; however, in observation 3 with muscular jerks, they think that if the duration is very short, there is no localization because the muscular action does not act sufficiently. All this seems a bit arbitrary, needing auxiliary hypotheses at every step, and if they had proceeded to rigorous observations measuring the intensity and duration of the stimuli applied in the different states, a single and simple explanation would have spontaneously emerged. We refuse as a whole the interpretations given by the authors on the three observations mentioned above, and we strictly adhere to the disorder of excitability relationships (increase of rheobase and chronaxie, asynchrony, summative facilitation, etc.). Concerning the theory of Goldstein and Gelb on that muscular jerks as well as other issues, we shall have occasion to discuss them later.

Having briefly reviewed the knowledge thus far on tactile localization, it will be very instructive to consider now the two patients studied in this work. In the course of several years of study of these subjects, we have gained insight into different aspects of spatial tactile disorder, particularly the fundamental one of tactile localization, until the complex and difficult problem of stimulus localization has been fully elucidated.

In the first observations made in 1938, the tactile examination of subject M was performed very superficially. The disorders found were limited to alterations of posture and passive joint movement. Given the predominantly occipital lesion, no attention was paid to sensory disorders that were not expected to be found. Sometime later, when inverted vision and other visual spatial

orientation disturbances were discovered, we found that tactile space also showed pathological changes.

In contrast, in patient T, who had been examined earlier, it was possible to observe from the outset that tactile localization was based on certain muscular jerks without which localization was almost completely lost. At that time, we did not know in detail the investigations of [Goldstein and Gelb \(1918, 1919\)](#) on the Schneider case. Subject T showed the muscular jerks so flagrantly that we were able to prevent them easily in order to observe their effect on localization, and indeed the failure was then very great and constant. In such a circumstance, it was extremely unpleasant for the patient to sense the contact of the stimulus without being able to localize it. He was aware that thanks to the artifice of muscular contraction he was able to succeed in the localization tests. The same behavior was shown in passive joint movement and posture. All these alterations especially concerned the contralateral side of the brain lesion, at least at the beginning of the examinations. Later it was observed that both sides were impaired, with little difference. The patient became aware of his particular tactile disorder during the examinations, and then tried to contract the musculature in one way or another, which made the tests very difficult, and he was even reluctant to do them for fear of failing them. Such muscular contractions, of enigmatic significance for us at the time, were also observed by us with varying clarity in some other brain-injured patients with tactile disorders (1938). Also [Kleist \(1937\)](#), commenting on Schneider's case, reports having noticed them in some of his patients.

As for patient M, in 1939, shortly before the finding of the dynamic action, it was already extraordinarily striking how long it took him to localize a stimulus. In trying to find the muscular contractions, already observed earlier in the T case and so much studied in the Schneider case, it turned out that although they could be found occasionally in M, they were not as constant as in the mentioned cases. In M, there was a certain degree of spontaneous and unconscious sustained muscular tension that he performed spontaneously and unconsciously. However, what was very noticeable was the long time it took him to localize a stimulus whereas it seemed as if he had perceived simple contact much earlier. But once the phenomenon of facilitation by muscular effort in vision had been discovered (at the end of 1939), and the other questions of visual excitability elucidated, all these new data were applied to the investigation of touch, which showed characteristics entirely identical to those of vision. It is remarkable the phenomenon observed in the tests that led to the discovery of the facilitation phenomenon. When the patient's head was held firmly, in order to avoid jerks and even certain movements of muscular tension, it was impossible for him to localize stimuli that at other times he was able to do. The patient then believed to explain the process by saying that in order to localize a certain location in his body he first had to know its situation with respect to his head, and therefore he had to "activate" his head first. This interpretation does not strictly correspond to the facts, but it is a fact to keep in mind when studying the body schema disorder in this subject. In fact, by means of facilitation, nervous summation immediately brings out the spatial function as a whole, without the patient having to resort to indirect orientations. Moreover, a precise knowledge of the excitability conditions revealed that, even without any facilitation, a stimulus sufficiently intense and of adequate duration could be perfectly localized. Thus, the conceptions of [Goldstein and Gelb \(1919\)](#) were discarded in this and other matters. In addition, being able to exclude any facilitating action, made it possible to study the patient in his true natural state, i.e. in the inactive state, thus bringing out the full extent of asynchrony (time lag). In this situation, intense stimuli that would be painful for a normal subject, if applied briefly, could go unnoticed, usually being perceived as a simple contact without reaching the slightest trace of localization. However, it was sufficient to prolong the application time for the function to be normal.

During the following years we devoted our attention almost exclusively to the study of visual functions in accordance with the new ideas of dynamic action, and to systematizing the new theory, as well as to checking the dynamic phenomena in case T. For these reasons, the phenomenology of localization in touch and other associated circumstances on excitability made little progress. However, it is worth mentioning the observation of tactile irradiation, which was compared with chromatic irradiation (pathological vision of flat colors). In subject M, the two extreme cases of localization (simple contact and normal pinpoint localization) were already known, as well as the fact that simple contact, besides being devoid of a specific localization, was

perceived as an extensive burst when stimulation was, for example, with a pin. Also, when the subject indicated a location, this did not usually correspond to a point, as it was in reality, but to a certain cutaneous surface.

The study of all kinds of new phenomena is, in any case, very arduous, and a progress in providing data and their rational interpretation needs a multitude of examinations and verifications. In addition, patients are unwilling to give details of the phenomena, and when they are obtained, very careful control is necessary in a series of tests, so as not to fall into considerable errors at every step. Thus, effective progress is a very slow work which must necessarily extend to numerous observations over several years. As mentioned elsewhere, the Schneider patient underwent a continuous study for about two years; however, as we know, neither a large number of pathological phenomena have been discovered in him nor has it been possible to take the necessary step to diagnose the patient from a physiological basis. Such a basis is what provides a correct and simple solution to the disorders found, and to many others that the subject should necessarily present.

By 1943-44, continuing the analysis of tactile localization disorder, a quantitative determination of the asynchronous beam of curves for both M and T under electrical stimulation was performed. This beam is the set of strength-duration curves corresponding to the stages of spatial asynchrony, already known at that time: 1st, simple contact without localization; 2nd, diffuse localization (irradiation), and 3rd, normal localization without irradiation. Such rigorous determinations provided a great advance in the knowledge of the question, especially with regard to the different physiological levels due to the difference in the magnitude of the lesion in both cases, and to the variations within each case through the action of maximal facilitation, mainly in the M case, as we already know. Subsequently it seemed convenient to complete this already encouraging set of data, and a meticulous work yielded very valuable results although it took a great deal of patience on our part and that of the patient. To the three stages of evolution in the localization process, others were added that provided new phenomena of extraordinary interest, especially inverted localization. Thus, a pathological situation was obtained in touch similar to that in vision. The significant deviation of the localization of a stimulus from the real location of the stimulus, a phenomenon that was not sufficiently clear before, was also studied in great detail. The finding of new well-characterized stages in the asynchronous process of localization meant not only an enrichment in the phenomena but also a readjustment of previous data.

In summary, the continued study of our brain-injured patients since 1938 has gradually led, through a series of stages, to a full understanding of the nature of the localization of tactile stimuli. These stages of improvement are as follows:

1938: Diffuse findings of tactile spatial disorder (muscular jerks in T and lengthening of localization time in M), difficult to explain according to the usual theories.

1939: Finding of the phenomena of dynamic action and their application to problems of tactile space, especially localization. Experimental rebuttal of the ideas of [Goldstein and Gelb \(1919\)](#) on the Schneider case by interpreting all the disorders according to dynamic changes in excitability (strength-duration curves, asynchrony or time lag, facilitation, etc.).

1941: Observations on tactile irradiation, a phenomenon comparable to chromatic irradiation (pathological vision of flat colors).

1943-44: Correct determination of the strength-duration curves for three phases of the localization process (simple contact, diffuse or irradiated localization and normal localization), thus forming a set of asynchronous curves with varying degrees of asynchrony depending on the patients considered. These quantitative measures are complemented with other types of graphs such as recruitment, etc.

1945-46: An even more precise phenomenological analysis supported by a very complete series of tests and quantitative determinations, finding the important phenomenon of inverted localization, various aspects of localization deviation, and types of irradiation, etc., until reaching a great systematic coherence in the process of tactile localization.

Such lengthy analysis reveals the complexity of a brain function as seemingly simple as tactile localization, and the difficulty in identifying the fundamental features of the process. Certainly, the analysis of tactile functions is much less amenable to objective evaluation than visual functions, and therefore, the separation between different stages in the asynchrony process is difficult and can lead to multiple errors. The main procedure consists of subjecting patients to very particular conditions of both state of excitability and stimulation, in order to conduct a sufficiently detailed investigation. The effort and guidance of the work must fall entirely on the person doing the observation and not on the patient, who in principle is completely unaware of his own anomalies.

If we have dwelt on the general aspects of spatial disorder, highlighting the background of the localization problem as well as the development of the research presented here, it is due to the utmost importance of knowing the localization disorder and its mechanism for understanding all kinds of spatial structures in touch. It is harder in touch than in vision to separate some disorders from others, and at every step it is necessary to pay attention to a multitude of aspects that evolve at the same time and in which localization is always involved. This is so regardless of the tactile activities, from the most elementary tactile response to the most complex organization of schema.

The study that we shall now present on tactile localization, and tactile space in general, constitutes a completely novel part of brain pathophysiology.

## **21.2 Phenomenology of asynchrony in tactile localization**

Because of asynchrony, the tactile localization of a pinpoint stimulus is broken down into a series of intermediate phases, thereby showing the evolution of localization as a function of stimulation energy. Thus, there is no all-or-nothing behavior, as in a normal subject, i.e., either a specifically localized contact or the complete absence of any sensation. Instead, there are pathological partial functions that have no precedent in a normal subject.

The partial phases that can be distinguished are theoretically indefinite, but in order to describe the process by focusing on the most salient aspects, it is possible to distinguish five quite individualized phases which, as the intensity of a given tactile stimulus increases, appear in the following sequence: I, primitive perception; II, medial deviation; III, inversion; IV, proximal deviation; and V, specific localization. Only the latter is normal whereas the others are pathological effects of asynchrony, and therefore, the greater the asynchrony, the better the phases are manifested. In the development of localization, several factors are present, among the main ones are: irradiation, resulting from the destruction of localization at one point; spatial deviation due to a reduction of the body schema; and contralateral inversion of localization due to a disorder of tactile spatial orientation. The evolution of pain and temperature during the gradual process of

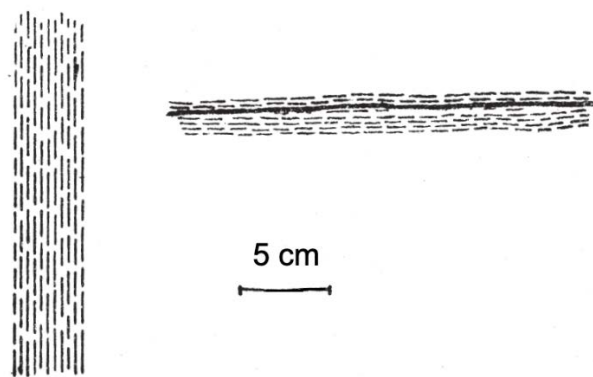


localization must also be considered. Thus, at each moment we obtain a very diverse evolutionary set, a multiplicity of interwoven sensory factors showing the full complexity of tactile localization. The five phases indicated above are a fundamental starting point for understanding all types of spatial activities, and in this case, in relation to touch. In subsequent chapters, we shall continually refer to these phases to understand other types of tactile functions.

The phenomenological description of these phases has been carried out almost entirely on the basis of subject M, who is the one who presents maximum asynchrony, and is therefore easier to study. M is also the patient who has undergone more frequent and detailed studies, not only because of his characteristics, but also because of the many difficulties to access the second subject (case T). The considerable amount of coherent details and their constant manifestation throughout multiple observations during the last years is a guarantee that allows us to describe entirely new phenomena, in which it is very easy to make observational errors, as we shall see. Within this section, we shall proceed to expose the phenomenology of the alteration, and in the following sections we shall study quantitative aspects by means of numerical relations on excitability and related graphs. In the following we describe the five phases mentioned above.

### 21.2.1 Phase I: Primitive sensation

By means of a mechanical stimulus on the skin of any part of the body, a tactile perception without any possible localization is obtained in both subject M and subject T when the intensity and duration of the stimulus are conveniently reduced. A sensation in the form of a gust spread diffusely over the skin is perceived. The subject can say that he has been touched but does not know where.



**Figure 97.** Diagram of the primitive sensation in subject M in the inactive state. A pinpoint stimulus gives rise to a band of irradiation (left) in some part of the body. Note the depth of the band (right) which penetrates and also protrudes from the tegument. Dashed lines indicate faint sensation, as a gust of air of the size indicated by the scale.

The test is performed on subject M in the inactive state and with eyes closed to avoid distractions. When the stimulus has the intensity to reach the first sensation, he perceives a band of irradiation that he estimates to be a span long, i.e. 20 to 23 cm, and about two fingers wide, i.e. 3 to 4 cm. He also believes that the band has a certain depth (1 to 2 cm?),

as shown in Fig. 97. This irradiation is very uniform and it is not possible to distinguish individual zones or points. It is completely impossible for the patient to localize it on the body and to indicate which orientation it adopts, if along the body or perpendicular to it. He only feels a cutaneous situation that seems to penetrate somewhat into the tegument and also protrude from it as swelling, the former being more pronounced than the latter.

While it is true that such a diffuse irradiation sensation may share certain characteristics of a cramp or paresthesia, it does not correspond at all to a protopathic or hyperpathic sensation. The band of irradiation is essentially neutral, and it is impossible to arouse thermal or painful qualities at this stage. The intensity of the sensation thus obtained is the lowest possible.

Regarding the mode of stimulation, whether by single pinpoint or iterative stimulation, scratching, application of a given surface or blowing on the skin, the result is always the same: a similar band of irradiation for all cases provided that the appropriate level of excitation is not surpassed.

All these stimuli are very high in relation to the threshold of the normal subject who by functional synchronization correctly localizes the stimuli even with the tactile threshold. This large sensory deficit explains that if the subject is given a light slap on the face with a few fingers, he does not feel anything, reaching only the primitive sensation phase. The different regions of the body have no effect on the result, so it is possible to obtain this primitive phase both in the stimulation of the body surface and inside the cavities (stimulation of the oral cavity or the surface of the tongue). Even in the sclera it is possible to obtain the first phase, and sometimes also in the cornea by stimulating with a hair, although due to the reflex movements of the eyelids the second phase is often reached. The cutaneous reflexes are therefore triggered by the mere sensation of contact, without any localization. The same occurs when the wing of the nose or the cheek near the eye is lightly and briefly tapped, since the reflex response of palpebral contraction is elicited. This means that such reflexes take place subcortically and are completely independent of the sensory localization of the stimulus. Thus, the reflex is localized but the sensation is not. However, this does not entail any difficulty of interpretation. This is a common observation in certain lesions of the nervous system (e.g. of the spinal cord). Different nervous centers are also involved: from the brainstem for the reflexes mentioned, and from the cerebral cortex for sensory organization. In any case, the reflex muscle activity that may appear is very slight, enough to be observed by the researcher but not by the subject who is totally unaware of it. Only in the case of somewhat abrupt stimulation of the cornea can it happen that repeated contraction movements of the eyelids promote more generalized defense movements, and by acting as facilitation, the simple contact phase changes to other more evolved phases.

In relation to the different zones, it seems that perhaps the central zones of the body (face, chest) are somewhat more sensitive than the distal zones (extremities, especially the most distal part). When the same stimulus is applied to all of them, a slightly more intense sensation and perhaps a little less irradiation is sometimes felt in the central zones, without exceeding the limit of the first phase.

At the beginning of the examinations in these subjects (1938-1939), this first phase of simple contact was difficult to be observed, not only because of the partial and automatic effect of facilitation by muscular action, but also because it was an unusual and strange sensation for the patients. They tend to exclude or disregard a tactile sensation that is not accompanied by a more or less specific localization; thus, it was possible that facial contacts able to elicit reflex contractions were considered by the examined subject as stimuli without any effect. When the stimulus was of sufficient intensity, the subject's sensation was waited for to develop until it reached at least a rudimentary localization. In this case he took a long time to give a response to the stimulation. All these circumstances can give rise to many errors, especially if the excitability conditions are not well known, or in cases with little asynchrony such as case T. It is therefore necessary to instruct the testing subjects conveniently, indicating to them the need to give their responses with great detail and precision. At the same time, it is necessary to adjust the stimulation mode to their excitability characteristics.

Under the action of facilitation by maximum muscular effort, the first phase can also be obtained in subject M, but it is necessary to use very slight and brief stimuli (for example, rubbing with cotton), otherwise the second phase is very easily reached. In areas of the body that seem to be more sensitive, such as the face, especially the lips, eyelids, etc., as well as the midline of the chest, the first phase is difficult to obtain under the aforementioned facilitation and is often almost confused with the second phase. In contrast, it is still easy to obtain simple contact on the limbs as long as the intensity of the stimulus is suitably reduced. It should be added that in the hands, facilitation by maximum contraction of the musculature reduces considerably the duration of the first phase, i.e. the first and second phases become very close to each other. If other types of facilitation are added, such as abrupt and wide movements of the limbs, a weak and brief stimulus (rubbing with cotton) on the hand is felt only as a simple contact, and sometimes as a second phase, but does not go beyond that.

As already mentioned, slight differences can be obtained within the first phase depending on the areas, i.e. the irradiation surface and the intensity of the sensation may vary slightly in skin areas of very different sensitivity (limbs and lips, for example). Likewise, there is a difference when comparing the inactive state with the state under facilitation: the irradiation of the simple contact under reinforcement seems to be somewhat smaller (perhaps three or four fingers shorter), and is very close to the second phase, which, apart from other characteristics, shows a smaller irradiation than the first phase.

In the T case, the first phase also seems to exist, although the size of the irradiation was not precisely determined. The essential point is that by using a weak stimulus, and above all a brief one, contact is perceived without knowing where. Because of the smaller excitability deficit, the size of the irradiation is more difficult to obtain than in the M case.

A convenient and reliable procedure to correctly determine the thresholds in this first phase when the subject is under facilitation by muscular effort, is the stimulation by moving a single hair on the back of the hand by means of a stylus. In this case there is no reaction, whereas in the normal subject a complete sensation of localization is produced. By repeating the stimulus, the first phase is obtained, and if continued a little longer, the second phase is reached. In the inactive state, this hand hair procedure does not produce the first phase no matter how long the repetition of moving the hair is maintained. However, it is possible to obtain it using eyebrow hairs, which are stiffer, especially if several are stimulated at the same time.

Finally, we point out the sensory change that occurs by facilitation. Let us consider a stimulus of adequate intensity so that in prolonged duration the first phase is not

surpassed. If the patient makes maximum muscular effort, he instantly passes from the first phase to the third or fourth. However, he does not reach normality, since from the fourth to the fifth (normal phase) there is a considerable gap (interval).

The primitive sensation phase lacks the possibility of differentiation, and stimulation with moderate cold or heat only provides primitive sensation. It is also not possible to register spatial and temporal intervals (vibration). Regarding the characteristics of general excitability in this primitive phase (useful time, fatigue, persistence, etc.), we refer to Section 18.1 on general excitability under mechanical stimulus, which corresponds to this phase studied.

Summarizing the characteristics of the first phase, there is maximum cutaneous irradiation in the form of an elongated gust of a certain thickness, uniform texture and without orientation with respect to the body. In addition, there is a total absence of localization as well as qualities and also of spatial or temporal structures.

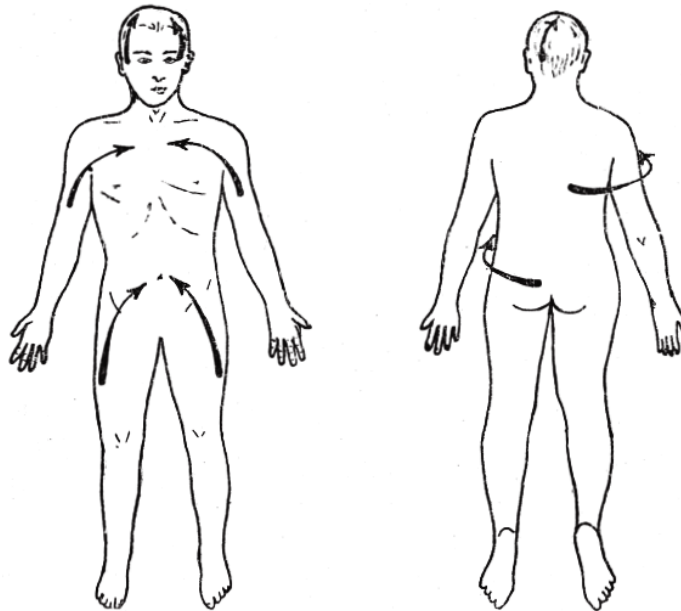
### 21.2.2 Phase II: Medial deviation

This second phase, which is difficult to explore, has distinctive characteristics that allow its identification. A rudiment of localization or spatial organization appears whose most important manifestation consists in the deviation of the perception of all types of stimuli towards areas of the anterior midline of the body.

This phase was impossible to be distinguished at first, and then confused with the first phase or with more advanced phases such as the third and even the fourth (see Fig. 120). It was finally possible to uncover it in subject M by conveniently fractionating the examination of the localization process and trying to note the smallest details accurately. Quantitatively, the stimulus is very little higher than in the first phase, and moreover, the step width for the next phase is very small, even in inactive M. A convenient method of adjusting the necessary stimulation consists in iterative excitation, by which, with repeated contacts or gentle rubbing with the tip of a piece of paper or absorbent cotton, different phases are recruited slowly and successively, stopping then at the appropriate one.

For the first time a certain degree of spatial localization appears, although due to the maximal proximal deviation at this stage, stimulation anywhere on the body is sensed only on the body axis, i.e., there is an *anterior medial localization*. Careful tests show that in the midline there are three zones, head, thorax and abdomen, which collect stimuli coming from the head, upper limbs and lower limbs respectively. The sensation is still, as in the first phase, an elongated band or gust of a certain thickness, perhaps about four fingers shorter than in the first phase, with analogous reduction for the other dimensions. All these measurements are approximate and subjective since the patient under examination can only indicate them indirectly. The irradiation band is still homogeneous, with no differentiation of a central point. Being slightly smaller in size than in the first phase, it should offer a slightly higher intensity of sensation. An irradiation of about 17-18 cm in length can be attributed to subject M in inactive state, especially in the case of stimuli on a limb which then produce a band of irradiation on the midline of the thorax. In this phase, unlike the first one, the irradiation band shows a well-defined orientation on the body, in the direction of the body axis.

At this stage it is still impossible to obtain tactile sensations. Painful or thermal stimulation only produces a sensation of contact or pressure. Perception of intermittent stimulation (vibration) is also not possible. Spatial discrimination in the usual sense (with Weber's compass) is abolished. However, sensation of duplicity in simultaneous stimuli may be obtained under special circumstances which we shall see later.

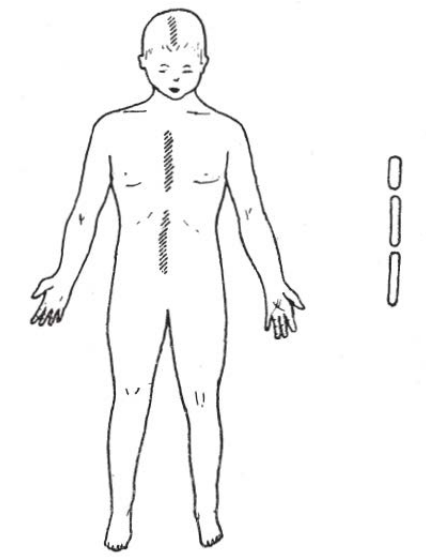


**Figure 98.** Phase II of localization (medial deviation). Deviation toward the midline and anterior plane of the body. (See also Fig. 101).

The *medial deviation* that sensations undergo in response to stimuli has several important peculiarities, and deserves to be described in some detail (see Fig. 98). In this phase, whatever the site of stimulation on the limbs, the sensation is perceived by subject M, inactive, on the midline of the trunk, and with such a special distribution that the upper limbs are represented on the thorax and the lower limbs on the abdomen. If the stimulation is on the trunk, e.g., on the sides, the stimulus will be perceived on the abdomen or on the thorax, depending on whether the site of application is low or high, respectively. In all these cases the irradiation band is located on the trunk axis. When the most distal part of a limb (fingers or toes) is stimulated, a maximum spatial deviation is obtained. This can be considered the *maximum effect of proximal deviation*, which is more moderate in more advanced phases, as we shall see. In any case, both in the case of a stimulus on the side of the trunk and on the fingers, there is no other possible localization than on the midline of the body, as already indicated. Moreover, it makes no difference whether the stimulus is applied on the anterior or posterior part of the limbs, since in all cases the patient gives the same response: on the anterior midline of the trunk, not on the posterior. When the back is stimulated, either on the midline or on the sides, a localization is also obtained on the anterior part, either on the thorax or on the abdomen, depending on whether the height of the stimulation point is on the upper back or on the lower lumbar region. It should be noted that this localization sensation cannot be compared in clarity to that experienced by a normal subject when stimulated in the chest or abdomen. These pathological sensations are diffuse and labile, and it is necessary to pay great attention to localize them in the midline of the trunk, and even more so to localize them on the anterior plane of the trunk. Several tests seem to demonstrate that this plane constitutes a zone of predilection, and that the posterior plane is systematically excluded. Such a deviation from the back towards the anterior plane of the trunk is confirmed in subsequent phases, although more moderate, as we shall see later on.

The prevalence of the anterior midline or of the anterior plane of the body over the exclusion of the dorsal plane is also demonstrated by the recent experiments of [Rey \(1947\)](#) on cutaneous localization by the stroboscopic method. Already in the normal subject, under special conditions, the anterior predominance is demonstrated by certain dynamic continuity effects (stroboscopic phenomenon) as follows. Two electrodes on the skin, distant from each other and successively excited in a short time interval, cause a sensory continuity between them. If the stimulating electrodes are placed one on the inner side of the thigh and the other on the outer side, at the same height, continuity is perceived on the anterior side of the thigh. The same result is obtained in the leg, arm and also the trunk. In the latter case, the electrodes were placed on the sides, their dorsal separation being shorter than the ventral. Despite such a difference, the dorsal region remained neutral and the sensory continuity was perceived in the abdomen following a ventral arc. Sensory effects seem therefore to be polarized towards the anterior part of the body. Thus, our findings on anterior predominance or deviation, obtained prior to those cited, and in another type of phenomena, may find support in the observations reported by [Rey \(1947\)](#).

As for the *head*, a stimulus anywhere on the head causes a sensation in an area located on the midline of the cranial vault. Facial stimuli on the sides or in the center are felt above the forehead with sensation of irradiation in the region from the cranial vertex to the root of the hair on the forehead. Thus, it can be said that the head behaves as a region independent of the rest of the body; and within the head, the face appears as a distal region, and the top of the skull up to the forehead, as a proximal or central zone. As for the neck, it is part of the zone that includes the head, and only in the lower part does the sensation deviate towards the chest.



**Figure 99.** Phase II (medial deviation) with indication of the three small regions (striped part) in the anterior midline: cranial vault, thorax and abdomen, working as areas of predilection to localize stimuli. On the right, size of the body schema in this phase.

There is therefore in this phase a strong deviation of stimuli anywhere on the body towards the mentioned three zones of predilection in the anterior midline of the body (Fig. 99), which collect excitations from their ascribed cutaneous regions. The body schema in this phase is reduced to three segments along the body axis, independent of each other and of very reduced width. The length of the segment corresponds to the length of the irradiation band, seeming equal in the thorax and abdomen, perhaps somewhat shorter in the cranial segment. The size of the body schema is considerably reduced, since the irradiation bands are only about two fingers (3 cm) wide, resulting in an almost filiform

body schema of about 50 cm in height. When the stimulus is more intense and the third phase is reached, the segments lateralize away from the midline.

The independence of the three medial sectors is also corroborated by the fact that it is possible to obtain double or even triple sensation by simultaneously stimulating the different medial segments or their dependent distal cutaneous territories.

Thus, two stimuli, one on the hand and the other on the foot, or one on the thorax and the other on the abdomen separated by more than 24 cm, are perceived as two on the anterior midline. In the case of the hand and foot, the separation between sensations on the medial zone does not exceed 3-4 fingers. In contrast, if the two stimuli fall in the same subsidiary territory (foot and abdomen, or leg on one side and thigh on the other), only a single stimulus is perceived on the medial zone. When several points on the head or face are stimulated, only a single homogeneous irradiation sensation is perceived on the cranial vault. When the anterior plane of the trunk is directly stimulated and the separation between stimuli is less than 20 cm, it is not possible to achieve double sensation, but with more separation, a band of irradiation on the chest and a similar one on the abdomen are perceived sufficiently well. If one stimulus is applied at an extreme point of the thorax (near the neck) and the other at an extreme point of the abdomen (e.g., the groin), the interval between the corresponding longitudinal irradiations is not much greater than in the previous case. In the first case, of hand and foot stimulation, the separation between the two sensations on the midline is similar to that in the previous test. Spatial discrimination is therefore quite rudimentary, and some sign may only appear under very particular conditions such as those indicated. Thorax and abdomen appear closer than thorax and head when stimulated simultaneously. By stimulating distal areas of the three medial segments (e.g. ear, forearm and knee), three longitudinal irradiations are obtained along the body axis, independent of each other and separated from each other. It can then be stated that the total length, including gaps, can be about 50 cm, and the width about 3 cm. In this second spatial phase, the body schema is reduced to such dimensions (Fig. 99). That is, only on the surface determined by these dimensions is it possible to perceive tactile stimuli, regardless of their origin. It should be noted that all this is blurred and unstable, at the limit of functional capability, and also difficult to evidence. In no way can it be compared with stimulus localization in a normal subject.

Finally, we shall mention some aspects on the action of facilitation. In order to obtain the second phase in subject M under facilitation, the stimulus intensity must be markedly reduced with respect to the inactive state. Especially on the face, the stimulation must be very brief, otherwise, the third or even fourth phase can be easily reached. This phase II was not explored in subject T since he was examined for the last time long before the finding of the medial deviation. Subject M inactive, at this second phase, can reach the fourth phase by means of maximum muscular effort. For example, if being in the inactive state and conveniently stimulated in his right hand to obtain sensation in the thorax, with the mentioned facilitation, the irradiation becomes much smaller and with a central point, all in the right forearm (therefore, moderate proximal deviation), which corresponds to fourth spatial phase. With less facilitation, the third phase would be reached, which consists of localization in the left arm, i.e., lateral inversion and more pronounced proximal deviation than in the fourth phase.

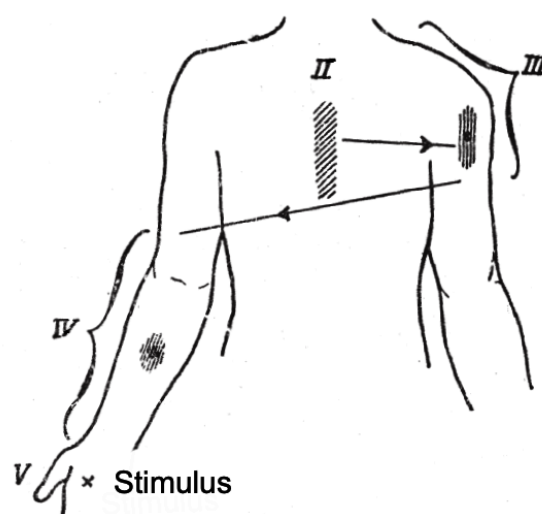
In summary, the main features of the second phase (medial deviation) are as follows. There is only a weak localization in the anterior midline in the form of a band of homogeneous irradiation, slightly smaller than in the first phase and oriented along the body axis. There are three segments of predilection in the anterior midline (cranial vault, thorax and abdomen) which pick up the stimuli applied in the ascribed territories,

including the dorsal plane of the trunk. The sensory body schema is greatly reduced (filiform about 50 cm long). There is no sensation of qualities or intermittency. There is double sensation of stimulation only when stimuli are applied simultaneously in autonomous regions.

### 21.2.3 Phase III: Inversion

This is a spatial phase of great physiological importance because it is equivalent to the inversion of the orientation of the visual image. For this reason, and given the complexity of the phenomenon, we shall devote a special chapter to it. We limit ourselves now to continuing the study on the development of spatial localization.

This phase, like the previous one, shows very small functional separation with the adjacent phases (see phase curves further on in Fig. 120). Therefore, only in subsequent more detailed tests, phases II and III were revealed. In spite of having some resemblance with the second phase (irradiation, labile localization, very reduced and poorly differentiated body schema, etc.), phase III already shows a significant progress in the localization process. It is remarkable the strong proximal deviation of the sensation without reaching the medial deviation of the previous phase, and above all, the inverted sensation with respect to the midline (*contralateral localization*). For the first time, tactile specific sensations appear, as well as certain rudiments of spatial organization (movement, duplicity, etc.). In addition, different gradations of the phase are obtained by varying the stimulus energy, i.e., some differentiation within the phase is possible. For this phase to appear, a significant asynchrony is probably necessary, as in subject M, inactive. This subject, under maximum facilitation, no longer presents the contralateral inversion of the third phase. Therefore, it is presumable that subject T, in whom this phase was not explored, does not present it, as will be discussed in other chapters. The following exposition thus refers to subject M in inactive state.

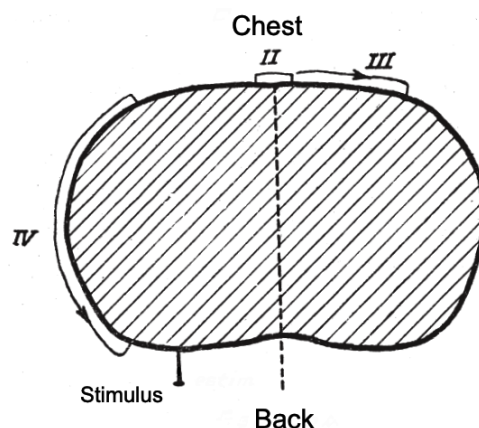


**Figure 100.** Diagram showing contralateral localization in phase III. Note its relation to phase II and phase IV



An iterative stimulus (repeated rubbing with the tip of a somewhat hard piece of paper) applied to the back of the right hand, for example, causes a sudden change from phase II (irradiation on the thorax) to phase III (left shoulder, contralateral), and then continues along the arm on the same left side (Fig. 100). It is difficult, even when the stimulus is regulated, to achieve a gradual transit between the midline and the contralateral shoulder. Instead, it is possible to obtain, by varying the stimulus a little, some recruitment within this phase III, tending to lower the localization from the shoulder to the elbow, until it is finally located homolateral to the stimulus. Whatever the degree of the third phase, there is always irradiation but it is no longer homogeneous due to the presence of a point in the center of the irradiation band. This band maintains its orientation along the axis of the body or of the limbs when it is located in the latter (only in the most proximal zone). The area of irradiation in this phase is smaller than in previous phases and, within the subjectivity of all these measurements, can be estimated at 16 and 10 cm in length at the beginning and end of the phase respectively. When stimulating in one hand, the irradiation in the contralateral shoulder would be the greatest, and when passing to the elbow (contralateral to the stimulus) it would be the least. Analogously, the width and thickness of the irradiation band are reduced. A probable moderate intensification of the sensation with respect to the previous phases would be due to the appearance of a central point in the band, which stands out just enough to be noticed.

The most appropriate way to study the characteristics of this phase is to observe it in the case of stimulation on the *extremities*, as has been indicated in part in the above examples. If a stimulus is applied on one hand and its intensity is gradually increased, a progressive transition from the second to the third phase can sometimes be found with great care. From the midline of the thorax, a slight contralateral deviation of three to four fingers begins, with a faint central point appearing in the band of irradiation. By increasing the stimulation, irradiation is directed first to the shoulder and then to the arm (both contralateral). Three different degrees of proximal deviation are thus obtained, all contralateral to the stimulation site. When the stimulus is applied to the dorsum of the hand, the transit to the homologous side (fourth phase) usually occurs at the level of the elbow. All this is valid for both upper and lower extremities and for both left and right sides.



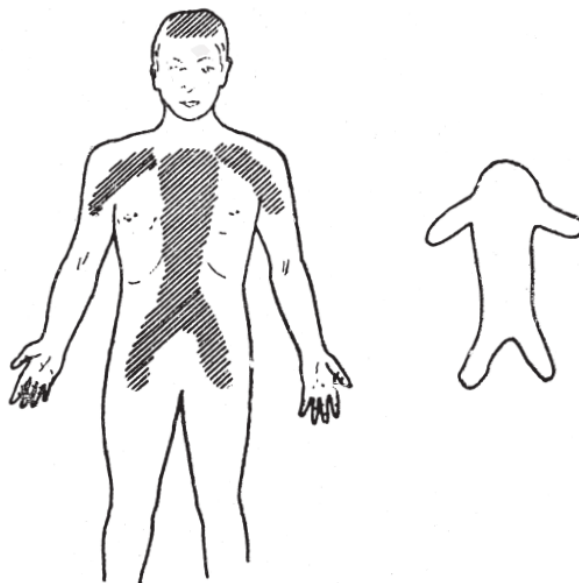
**Figure 101.** For stimulation on the back, the corresponding localizations are strongly deviated towards the chest (phase II), and also contralaterally in phase III.

For the *trunk*, the same type of proximal deviation appears, which is strong and contralateral. A stimulus on one side of the thorax, toward the flank, is localized centrally in the second phase,

and on the contralateral side in the third phase. In this phase, the distance to the center is much smaller than the distance from the stimulus to the midline. When the excitation is intensified, the localization moves to the fourth phase, and the lateral inversion is suppressed, but some deviation toward the midline still remains. This behavior is also observed when the excitation is applied to the back (Fig. 101). In this case, the proximal deviation is more pronounced, and in the third phase the subjective localization falls on the anterior plane (the chest), towards the contralateral side, even when the stimulus is applied three or four fingers away from the midline on the back. Therefore, the back behaves as a *distal sector*, and since the anterior plane predominates in the proximal deviation phenomenon, the back is excluded in the third phase. As we shall see in the next phase, the distal sectors of the body and the back begin to become involved.

As for the *head*, taking into account what was mentioned above about the face (distal) and the cranial vault (proximal or central), we can guess what happens in this phase. For example, a stimulus at the ear is perceived at the vertex in the second phase, and contralaterally two or three fingers away from the midline in the third phase. Even if there is a transition to the fourth phase, there is still deviation, and the sensation is homolateral toward the temple. The third phase is therefore localized exclusively in the cranial vault, regardless of where the stimulus is applied on the head.

In addition to the inversion, the strong proximal deviation of the third phase implies a notable reduction of the body schema, which, although increased with respect to the second phase, is far from reaching its normal shape and size (Fig. 102). Thus, the possible body schema, besides being very small, is diffuse and unstable; the posterior plane does not yet exist, the extremities only in their proximal half, and the face and neck are also absent.



**Figure 102.** Body schema in phase III (striped area). On the right, the corresponding homunculus. In addition to the general reduction in size, all distal parts of the limbs are excluded, as well as the face and back.

When the localization is contralateral (inverted) in the proximal part of the limbs, it is important to note that it is not possible to distinguish whether the stimulus is anterior or posterior (palm or dorsum of the hand). It seems that the subjective location of the irradiation band with central point is either indeterminate in the proximal extremity, or it is located dorsally (external).

In the latter case, it would be a *dorsal predominance*, in the same way as the aforementioned predominance of the anterior plane of the body. In relation to the lability of localization in this phase, it should be noted that shortly before the discovery of the phenomenon of localization inversion, the subject, stimulated on one limb, responded that he had felt something on one limb, but he was unable to tell which side. Later, it was found that a localization with lateral inversion had occurred.

Another characteristic of the third phase is that pain and temperature appear for the first time, albeit very slightly, and more precisely towards the middle of the phase; that is, for a painful stimulus in one hand, the algic sensation appears weakly when the subjective localization corresponds to the contralateral shoulder-arm. The same occurs with temperature. As for spatial organization or differentiation, a certain aptitude for duplicity of stimuli already appears at this stage, for example, within the same limb. However, considerable separation is required between the two simultaneous stimuli; thus, for a distance of at least one stimulus on the hand and another on the elbow, the subject perceives the duplicity contralaterally and towards the shoulder. All these circumstances are most probably due to the fact that the irradiation is smaller than in the previous phases, but above all to the existence of a central point, and to the progress in the whole functional organization. There is also perception of motion, although with spatial characteristics that we shall see later. This perception is proximal and contralateral (lateral inversion) and of inverse direction to that of the real moving stimulus. However, the intermittency of stimuli (vibration) belongs rather to the fourth phase, and if it existed at the end of the third phase, it would be difficult to appreciate because it would be a very weak indication.

Finally, we shall examine the effect of facilitation on this phase. By means of maximum muscular effort, the stimulus, which in the inactive state gives rise to the third phase, causes the passage to the final part of the fourth phase, i.e., to almost normal localization. Nevertheless, some proximal deviation still appears, although minimal. This occurs in the case of stimuli on a hand, an ear, etc. Thus, the stimulated site is not completely reached by such facilitation, but it is very close. When the subject is under facilitation by maximum muscular effort, and the aim is to obtain the third phase by considerably reducing the energy of the stimulus with respect to the inactive state, it is impossible to obtain it; either phases I and II arise or there is a transition to the fourth phase, but the characteristic lateral inversion of the third phase is never reached. This absence persists in many other tests, so the exclusion seems to be confirmed. This does not preclude that on rare occasions a third phase under facilitation is achieved, which can be interpreted as due to a submaximal effort that allows some margin, albeit very small, for the inverted third phase to appear.

In summary, the most salient features of the third phase are as follows. The irradiation band is smaller than in the second phase and with a central point, contralateral localization to the stimulus, and strong proximal deviation. It is possible to differentiate several degrees within the phase. The body schema is very small and diffuse, excluding the distal half of the limbs, as well as the posterior plane of the trunk, face and neck. There is perception of motion (kinetic stimulus on the skin), some spatial duplicity and pain and temperature sensations. There is not yet aptitude for intermittent sensations (vibration).

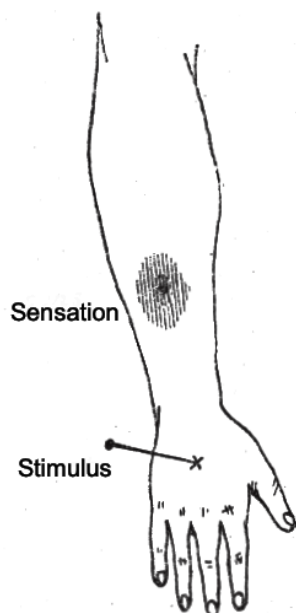
### 21.2.4 Phase IV: Proximal deviation

This phase is easy to demonstrate in both subjects (M and T). Already in the first years of special examinations (1940-41) it was evidenced. It is present even in cases of mild asynchrony, therefore its incidence in brain tactile disorders must be very high, in contrast to the previous phases, especially intermediate II and III.

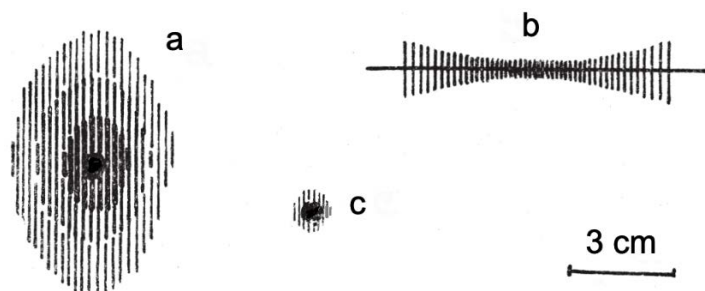
This phase, with a larger step in excitability (see phase curves further on in Fig. 120), starts when the localization is no longer inverted, i.e., just after the transit from contralateral to homolateral localization, and ends when the localization is practically normal, i.e., without deviations or irradiations of any kind, which corresponds to the fifth phase. It involves considerable recruitment, for which a progressive increase in stimulus intensity is necessary. The degree of proximal deviation and irradiation, much lower than in the previous phases, is related to such intensity. In subject M, inactive, the heterotactile interval of the qualities is overcome, and there can be a more or less intense qualitative sensation. With mechanical or electrical stimulation, a very slight pain is awakened at the beginning of the phase, becoming very intense at the end of the phase, this end being indispensable for recruiting the more advanced degrees of sensation, at least in the inactive state. The most notable characteristic of this phase is a moderate proximal deviation, always homolateral. In addition, the irradiation tends to adopt the shape of an oval of variable size, depending on the intensity of stimulation.

As soon as the localization moves from the contralateral side (third phase) to the homolateral side (fourth phase), or shortly thereafter, the spatial localization tends to become clear, i.e., it is known whether the stimulation corresponds to an anterior or dorsal area of a limb, or whether it corresponds to the face or towards the back (considered as distal areas), even when there is a noticeable proximal deviation at the beginning. In contrast, on the contralateral side, the subject tends either to locate the site on the dorsal (outer) side of the limb, or it remains undetermined, as mentioned above. The irradiation that occurs is highly variable. At the beginning the irradiation band is 10 cm or a little more, with the central point of the previous phase more intensified. Towards the middle of the phase it is about 6-7 cm, and oval-shaped, which means a type of irradiation with a spatial organization. At the end of the phase, if the stimulus is a painful pinprick, the oval decreases considerably, although a residue of 1.5 to 2 cm in diameter always remains (Fig. 104), impossible to remove in the inactive state. If, for example, subject M in the inactive state is moderately pricked on the dorsum of one hand for a time long enough not to enter into consideration in the excitability relations, he feels pain in the middle of the contralateral arm (Fig. 100); but with very slight intensification of the stimulus, the sensation moves from the level of the elbow to the homolateral limb, appearing a band of irradiation of about 10 cm in length on the upper forearm. By increasing the stimulus, the proximal deviation and irradiation decrease, and the above-mentioned oval of irradiation appears in the lower third of the forearm (Fig. 103). The irradiation is shortened at the ends, and the contour tends to acquire an elliptical shape approximately. In any case it can be known that it is neither a band, as before, nor a circle. The distance from the point

of application of the stimulus to the center of the oval is in this situation 8 to 10 cm. When the oval has become as small as possible by increasing the stimulus, it still has a certain size (1.5 cm), and it is already localized on the hand with a very small deviation, almost negligible, like that of a finger. The pain due to the prick is at that moment extremely annoying.



**Figure 103.** Proximal deviation in phase IV. Stimulation on the back of the hand and sensation on the forearm (oval-shaped irradiation with a more intense central point).

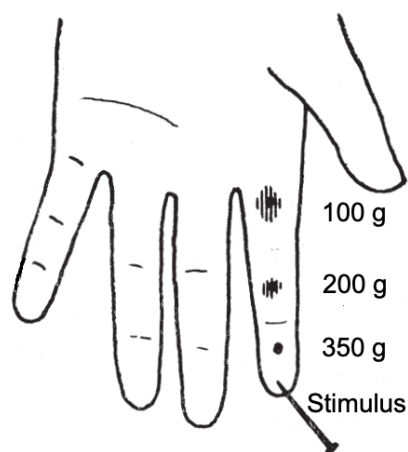


**Figure 104.** Irradiation in phase IV. (a) Characteristic oval shape with a more intense center. (b) Oval profile (thickness in the skin). (c) Irradiation at the end of phase IV.

The characteristics of the oval-shaped irradiation deserve comment in some detail (Fig. 104). The irradiation presents a point in the center where the sensory intensity is greater and with some moderate superficial pain that gradually decreases towards the periphery. It seems that there is still a more eccentric zone devoid of quality. Since the irradiation is not only superficial, but also in depth to some extent, it can be felt that the thickness of the oval is very small or almost null in the center, and that it increases significantly towards the periphery, i.e., it adopts a biconcave shape (Fig. 104 b). It should be noted that the oval shows some organization, and all its characteristics can be best perceived when it is at its maximum size. The subject is also able to perceive the orientation adopted by the oval, especially as long as it does not reach too small a size, in which case it cannot be distinguished from a circle. Like the irradiation band in other phases and in this one at the beginning, the oval is oriented on the skin generally following the

body axis, and along the limbs. At the beginning of the oval (maximum oval), its size varies somewhat according to the regions of the body: very small on the eyelids and tongue (size between a coin and a lentil), somewhat larger on the lips and cheeks, and much larger on the limbs and trunk. The oral cavity presents special characteristics. When the inner wall of a cheek (mucosa) is stimulated and the third phase is reached, contralaterally, the sensation is localized externally, up on the skull; and in the fourth phase, it is localized homolaterally on the inner wall. If facilitation is then applied, the sensation is localized closer to the stimulus. All this has been tested several times with stable results.

In the fourth phase it is possible to localize stimuli on the various areas excluded in the previous phases (face, back, neck, more distal part of the limbs, etc.), but a certain moderate proximal deviation persists, which diminishes with intensification of the stimulus but does not disappear completely, nor does the small oval or circle. A very painful stimulus on the earlobe is always localized a little in front of the stimulus site or on the cheek, no matter how painful the stimulus is. However, by means of facilitation, the localization coincides with the stimulus application site. All this corresponds to the end of the phase, since at the beginning, the localization may be on the homolateral temple. The same occurs with other parts of the face. A stimulus on the lips may be felt, depending on its intensity, either on the forehead or on the nose, and only by painful stimulus the localization comes close to the place of its application. Excitation at the tip of the tongue seems to be localized towards its base, and although the deviation is small, it is sufficient to be felt. With more intense stimulation, or with facilitation, the deviation disappears.



**Figure 105.** Phase IV. Proximal deviation in very distal areas. Deviation and irradiation vary according to the intensity of the stimulus.

In order to indicate the subjective localization of the irradiation oval, subject M, inactive, answers verbally with eyes closed or without looking at the examined area. It is relatively easy to indicate the localization on very significant areas (elbow, wrist, tip of the nose, ear, chin, etc.), but on other regions he can only give somewhat approximate answers (along the limbs, trunk, etc.). In the latter case, M may be asked, for the sake of accuracy, to compare the localization in the fourth phase with the practically normal localization of a strong stimulus under facilitation by maximal muscular effort. To inform about the differences, he can do so either verbally or by pointing on a graphic model of the examined part, or on his own body or on that of the examiner. Thus, it is possible to specify precisely the localization deficit at the end of the fourth phase with an observation of great interest (Fig. 105). For example, if a pinpoint stimulus is applied to a fingertip (calloused skin) with a weight of 100 g, it is localized at the proximal end of the first phalanx. If the weight is 200 g, it is localized at the end of the second phalanx (rather painful). If the weight is 350 g, it is located with intense pain in the center of the third phalanx or fingertip. There is always some proximal deviation which at the end of the phase may be 1-1.5 cm with an irradiation of the size of a lentil. Absolute normality therefore does not belong to the fourth phase

for subject M inactive. Under facilitation by muscular effort, complete normalization of localization and size is obtained, even with a stimulus lower than the maximum in the inactive case, about 300 g or less. In any case, a fairly accurate localization can be obtained with much less painful stimuli than in the active state, since what is achieved with 350 g in the inactive state, is achieved with 200 g under facilitation, a stimulus saving that varies between one-third and one-half.

Finally, it should be noted that most of the qualitative and spatio-temporal tactile discriminations take place in the fourth phase. Already from the beginning of this phase, pain and temperature qualities are perceived, as well as motion over the surface and duplicity of stimuli. The latter two sensations tend to improve considerably towards the end of the phase. Intermittency (vibration) appears for the first time, most probably towards the middle of the phase. In addition, the body schema increases considerably in size and especially in coherence, compared to the third phase. However, this situation is far from being similar to the normal state, and in addition to the necessary increase in stimulus intensity and application time, a series of functional reductions appear, which we shall deal with further on.

In summary, the characteristics of the fourth phase are as follows. There is localization with moderate proximal deviation, and homolateral to the stimulus. The irradiation has an oval shape with a certain structure consisting of an intense central point with decreasing tactile quality towards the periphery, and a much smaller thickness of the oval in the center, etc. There is localization in the area close to the stimulation, being possible the localization in previously excluded zones (face, back, distal zones of the limbs, etc.). Motion, intermittency (vibration), duplicity and intense qualities are perceived, and the body schema reaches a near normal size. However, there is still a marked functional reduction.

### **21.2.5 Phase V: Specific (normal) localization**

In the previous phase, a completely normal specific localization is not achieved in subject M inactive. No matter how much the stimulus is intensified, some irradiation and deviation remains. This is what happens in vision, for example. If M is in the inactive state, it is neither possible to completely suppress color irradiation nor to achieve a completely correct visual orientation in monocular vision. The recruitment of the asynchronous levels is not complete, and there is always a remnant of asynchrony, albeit very small. When the functional state is more favorable and the asynchrony much smaller, for example in M under facilitation or in subject T, the mere increase of the stimulus is sufficient to reach a specific (normal) localization. In this case, the fourth phase will lead directly to the fifth (normal) phase.

It follows from the above that *fully specific localization* without deviation and irradiation is only possible in subject M by facilitation and a fairly intense stimulus, although its intensity may be somewhat less than that needed to achieve the end of the fourth phase in the inactive state. Examples of this have already been indicated (Fig. 105). Anomalous traces of irradiation and deviation that may exist in other areas, such as the back or others, are more difficult to examine because these areas are extensive and with

low specificity. Such remnants should also disappear and the specific localization reach the whole body. If even with intense stimulation and facilitation, some irradiation of the order of millimeters seems to persist, it should be considered within the limits of perception.

All other tactile functions improve considerably when specific localization is achieved (two-point discrimination, vibration, motion, body schema, etc.). However, functional activity is far from normal, although the difference from normal is much smaller than at the end of the previous phase. A detailed description of such behavior will be presented in another chapter.

**Table 23.** Tactile spatial development in subject M in the inactive state. First row: modalities of differentiation or functional organization in approximately increasing order. First column: localization phases, I-V. Phase V only appears with facilitation. (See the text).

	<i>Irradiat.</i>	<i>Deviation.</i>	<i>Body schema</i>	<i>Side</i>	<i>Intens. phase discrim.</i>	<i>Pain, temp.</i>	<i>Spatial discrim.</i>	<i>Motion on skin</i>	<i>Vibrat.</i>	<i>Forms</i>
<b>I</b> <i>Primit. project. (sens.)</i>	Max. uniform band ----- Indist. project.									
<b>II</b> <i>Medial deviat.</i>	Smaller uniform band ----- Along body axis	Anterior medial line ----- Maximal proximal deviation	Max. reduct. ----- Three filiform segms.							
<b>III</b> <i>Invers.</i>	Smaller band with central point	Large proximal deviation	Reduct. difuss. ----- Distal exclus., and face, neck, back	Contralateral	Signs	Signs at mid-phase	Signs	Signs of mov. (invert.)	?	
<b>IV</b> <i>Prox. deviat.</i>	Oval with structure	Moderate proximal deviation ----- Distal loc.	Almost normal size ----- Coher.	Hontralat.	Marked	Very strong at end of the phase	Weber (initiat.)	Larger but sub-normal	In middle of the phase	Unstab.
<b>V</b> <i>Specif. Loc.</i>	Negl. ----- Point localiz.	Specif. localiz. ----- Negl. deviation	Almost normal	Normal	Almost normal	Almost normal	Almost normal	Almost normal	Sub-normal	Almost normal

As a general summary of the asynchrony phenomenon in spatial localization, it can be stated that it is mainly characterized by *irradiation* and *deviation*. Both progressively decrease as the function approaches normality (see Figs. 106 and 107 in the next section). This developmental process is not limited to a few functions, but involves all tactile activities without exception. This global process entails a great functional complexity, as seen for example in contralateral inversion, which involves tactile orientation function.



Hence, a multiplicity of reactions appears in pathological localization. This can lead to a multitude of important errors when studying patients, as they tend to respond in a diffuse or vague manner at first, and also leads to errors on the part of the examiner if he does not keep the test conditions sufficiently constant.

The localization process exposed needs to be completed at several points by taking into account other more complex spatial functions that have so far only been mentioned. The phenomenology now established is fundamental as a starting point for further studies and a summary of it is given in Table 23. In the first column of Table 23, the five phases of localization are indicated. The first row shows the modalities of differentiation or functional organization in approximately increasing order, although some belong to the same functional level. The 'Irradiation' and 'Localization' columns show the main localization phenomena, partly also in the 'Body schema' column. In the table it can be seen that functional differentiation is null in phases I and II, there are signs in phase III, it progresses greatly in phase IV and tends to normality in phase V.

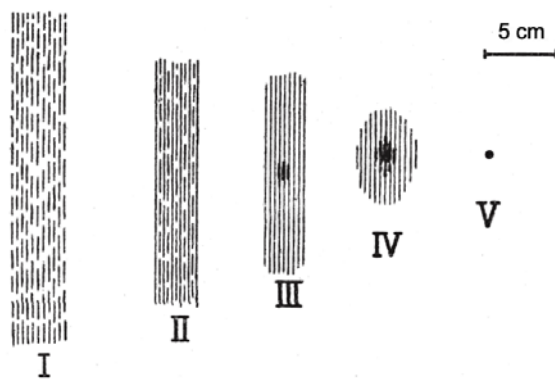
### **21.3 Meaning of the spatial disorder. Structural change of the sensory field**

In view of the set of the above phases, it is now appropriate to give an interpretation of the essential features of the spatial disorder. Leaving aside special circumstances, such as inversion, it is evident that in any phase, localization is determined by the degree of proximal deviation and by irradiation. It is observed that with pinpoint stimulation the disorder is twofold, since there is an absolute alteration of localization (due to deviation) and a relative alteration (due to irradiation) that modifies the pinpoint modality of the stimulus.

#### **21.3.1 Relative disorder**

The analysis of the *relative* disorder (irradiation) shows first of all that its size as well as its thickness are inversely related to the intensity of the sensation (Fig. 106). To the very large irradiation of the first phases corresponds a very weak tactile sensation, whereas in the later phases the irradiation tends to zero, i.e., the sensation tends to become punctiform, corresponding then to a very painful intensity.

This disorder of pinpoint localization gives rise to both color irradiation (vision of flat colors) and tactile irradiation, the tactile system showing phenomena similar to those studied in the visual system. In vision, we find that color can appear as "aerial color", "flat color" and "surface color", with irradiation and voluminosity decreasing in that order. There is still an extreme pathological phase that appears as achromatic fog, where there is no color due to the photochromic interval, and the faint luminous sensation is voluminous, diffuse and wide, even though the stimulus is chromatic and almost point-like.



**Figure 106.** Diagram showing the irradiation in the five phases of localization as the intensity of the stimulus increases.

In the same way, touch presents, according to the phases of localization, different degrees of irradiation in depth and on surface. The achromatic fog mentioned corresponds to the primitive projection in touch (phase I), whereas the aerial colors correspond to the tactile phases with a band of irradiation (phases II and III). Flat color vision (usual in subject M), corresponds in touch to irradiation in a more organized space (phase IV), and normal surface color vision corresponds to the pinpoint localization of phase V. As in vision, now in touch we also find irradiation in all directions (surface and depth), but examining the detail of the phenomena, we also find certain differences that are important to note. In vision, irradiation predominates in the direction of depth (frontal irradiation), being much less pronounced in the other directions (lateral irradiation). In contrast, in the case of touch, the irradiation thickness is considerably smaller than the irradiation surface (lateral). Moreover, whereas in vision, lateral or surface irradiation is similar in all directions, in touch it adopts the marked form of a band. Even when irradiation in the form of a diffuse band tends to diminish, it adopts an oval shape, persisting the predominance of longitudinal irradiation.

Such differences do not seem difficult to interpret if one takes into account the different functionality of vision and touch; the former is closely related to spatial depth, whereas touch is very weakly so. The visual field has a circular structure, whereas the tactile field or body schema is clearly elongated. It is therefore understandable that in the case of touch, irradiance is band-shaped and only moderately thick. Thus, the spatial disorder of irradiation finds, both in vision and in touch, certain *previous molds*. Such an elongated arrangement in the case of touch, due to the influence of the body schema, exists even in the most primitive spatial stages, where all reference of localization in the body is lacking. The *shape of the sensory field*, and therefore the action of the body schema, determines the *irradiation form*.

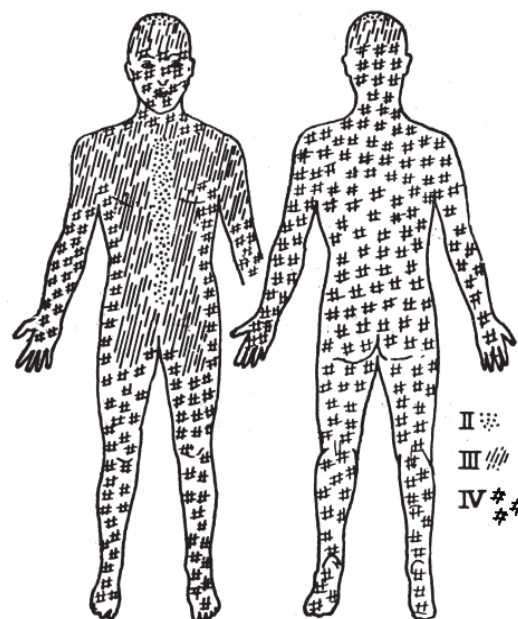
In the comparison between color irradiation and tactile irradiation, it should be noted that although in the study of flat colors, reference was made to the vision of colored surfaces (piece of cardboard), and now in touch, to mainly pinpoint stimuli, such a difference in stimulation does not essentially prevent direct comparison. In the lower phases of tactile localization (I to III), the size of the stimulus applied is indifferent, influencing a little more in the later phases according to their characteristics.

A special issue in relation to irradiation is the stinging sensation, which corresponds to the well-known clinical test to distinguish between touching and pricking (with the head and the point

of a pin respectively), and although it has been discussed when studying pain quality (Sec. 19.2), the issue should now be clarified in relation to the phases of irradiation. In the first phases (at least I and II) such a distinction is impossible since these phases correspond to homogeneous irradiation. This distinction is possible during part of phase III and during all of phase IV, but indirectly, since a true pinprick sensation is never obtained, but rather a somewhat extended pain. When either the head or the tip of a pin is applied on the skin with the same pressure, the sensation is certainly different; for example, the first phases are obtained for the head, and phase IV for the tip, but this distinction is not the one perceived by a normal subject after being touched or pricked. In M inactive, it is possible to obtain in phase IV the same sensation (equal irradiation and deviation) by applying the tip of the pin as by applying the head of the pin with greater pressure, being impossible any distinction that in a normal subject is feasible. All these circumstances have consequences for the acuity function (Weber), for the appreciation of tactile microstructures (texture of objects), for tactile recognition, etc., as will be seen below.

### 21.3.2 Absolute disorder

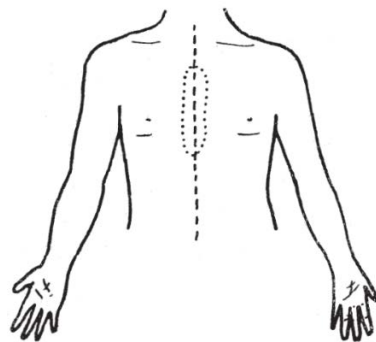
The *absolute* localization disorder (proximal deviation), diversely pronounced according to the different phases (Fig. 107), is in fact a *concentric reduction of the body schema* (Fig. 110). Here again, the similarity of behavior between the visual and tactile systems is evident. Concentric reduction of the visual field and concentric reduction of the body schema just mean concentric reduction of the sensory field in any of the alluded systems.



**Figure 107.** Localization zones for phases II, III and IV. Note their different extension and the tendency to concentric reduction and narrowing of the body schema in the first phases.

Using a suitable stimulus on any part of the body, simple contact sensation can be obtained, but the stimulus can only be localized in phase II in the anterior midline and in a very narrow area. If the stimulus is applied on a finger, this medial localization is an extremely large deviation. This means that the field for simple contact sensation and the field for spatial localization are very dissimilar. The field for spatial localization is reduced to a narrow band in phase II, whereas the field for simple excitability extends

over the whole body (Fig. 108). This size disparity is maximal in phase I, since the spatial field is null because localization is completely abolished. Instead, the disparity tends to decrease through phases III and IV, until in phase V the disparity disappears and the fields coincide, the sensation being localized at the same site of stimulus application. Thus, the spatial field begins to develop, at least in M inactive, when the field for simple contact sensation already exists in its entirety, which indicates that the latter is much more resistant to functional reduction. It is clear that the importance of proximal deviation is related to the degree of *incongruence of the aforementioned fields (simple contact field and spatial field)*. The body schema is thus identified with the spatial sensory field, whereas it is alien to the simple sensory field, a difference which is only possible pathologically, due to asynchrony, since in the normal subject there is not the slightest sign of this difference.

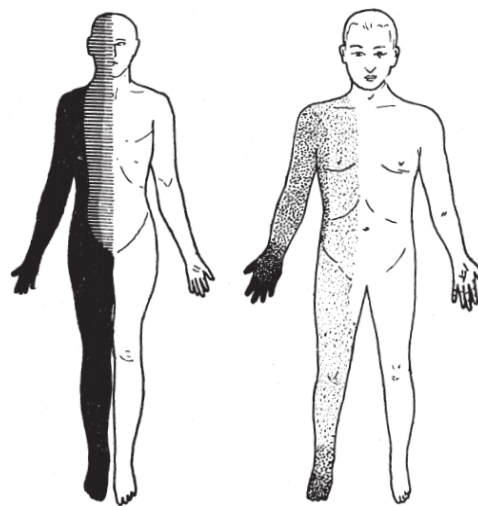


**Figure 108.** Incongruence of fields because of abnormal sensory interval (gap) due to asynchrony. For a functional level of phase II, the whole body surface is excitable, but localization is deviated and restricted to the anterior midline area (delimited by dotted line).

To be more complete about the important process of concentric reduction, it should be noted that this reduction can also occur in the simple (sensitive) field, since in all cases the development is from the center to the periphery because of the *medial privilege*. When describing phase I, it was already indicated that the central regions of the body seem to be more sensitive than the peripheral ones and, of course, the same is true for the other phases. Thus, let us consider a stimulus applied in the central zone, with threshold intensity to obtain phase I; if this same stimulus is applied in the periphery or in a more distal region, it must be below the threshold for this phase. This is a centripetal narrowing of the sensitive field, resulting in a situation identical to that of the concentric reduction of the visual field. In this respect we have not performed sufficiently precise quantitative determinations about the tactile field, but the different sensitivity from the center to the periphery is evident.

It is a well-known fact that in tactile hemianesthesia and hemi-hypoesthesia, distal areas suffer more than central areas, not only in cortical lesions but at any level of the neuraxis, such as lesions of the thalamus, or of the spinal cord. Moreover, in mild lesions or in advanced stages of recovery of the disorder, symptoms are easily manifested only in the more distal areas of the body such as hands and feet.

Redlich (1915) carefully studied the topography of the trunk of the body in cerebral hemianesthesia, and found two or three parallel zones of different intensity of anesthesia, the more distal, the more severe (Fig. 109 left). Goldstein (1915) later confirmed this arrangement of the disorder (Fig. 109 right). The distal predominance of the disorder has also been evidenced by different authors, such as Kleist (1934) and Foerster (1916). The *privilege of the midline*, i.e., its lower intensity of alteration as opposed to the greater distal vulnerability is an important issue, just as in vision is the preservation of the macula in cortical hemianopsias and in the concentric reduction of the visual field. Both phenomena are aspects of the same process for both vision and touch, which poses an insoluble problem within the theory of anatomical localization, but not so within our dynamic conception, as we know. Kleist (1934), for example, tries to interpret anatomically the aforementioned type of disorder by assuming that the bilateral midline innervation would gradually diminish towards the periphery, accentuating the functional deficit of the more distal areas, although he also believes that the hand and foot suffer more because of their greater differentiation. Other authors also go no further, such as Foerster (1916) and also Goldstein (1915). Without entering into a detailed discussion of the various views, it is worth noting the difference in clarity and simplicity between either of the two hypotheses, localist or diffuse biological, and the dynamic physiological interpretation. This could already be illustrated in a hemianesthesia. For concentric reduction by unilateral lesion, as in our cases, both in sight and touch, any explanation outside brain dynamics fails completely. Bourguignon (1933) finds that already in the normal subject, the chronaxie for pressure increases from the center to the periphery of the body, behaving similarly to the chronaxie of the underlying muscle groups. Thus, the situation is entirely similar to that of the visual field whose sensitivity decreases from the center to the periphery. Pathologically, the alteration is more pronounced in the periphery because the less excitable function is affected in a greater proportion. Thus, concentric reduction as well as greater distal involvement appear as a direct consequence of dynamic reduction according to a very simple physiological process, without the need to resort to complicated anatomical hypotheses that have never been experimentally corroborated.



**Figure 109.** Residual cortical hemianesthesia. On the left, after Redlich (1915), on the right, after Goldstein (1915). Note the greater distal involvement, and in the figure on the right, the intact midline.

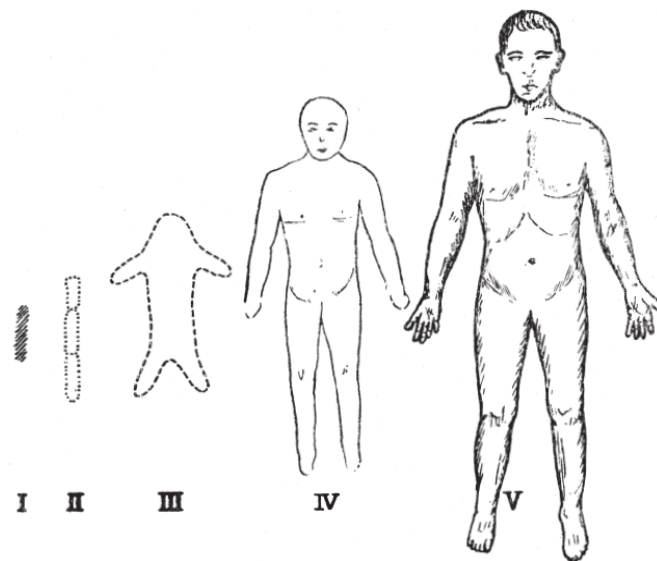
### 21.3.3 Structural change of the tactile sensory field

As we have seen, the phenomenon of irradiation as well as the narrowing of the sensory field evolve in unison (compare Figs. 106 and 107), showing the profound *structural*

*change of the tactile sensory field* with the reduction of phenomena, physiologically explained by the desynchronization of functions that in a normal situation are united.

The pathological alteration is for the entire field, and dynamic reduction is manifested in concentric narrowing of the field by virtue of the physiological privilege of the medial zone, as well as in spatial dedifferentiation. Since the central or proximal area of the body is more sensitive, excitability arises first in this area, and spreads peripherally as functional capacity improves. However, since spatial localization of stimuli requires a higher functional level, absent in peripheral areas, stimuli are diverted to medial areas of greater sensitivity. In other words, the abnormal interval between the sensitive field and the spatial field becomes evident. This means a functional dedifferentiation, also corroborated by tactile irradiation, since spatial specificity is altered by both deviation and irradiation. In short, the *residual field* is a narrowed field due to deviation, and a lax field due to irradiation.

It should also be noted that the cohesion of the body schema, i.e. of the tactile spatial field, may be poorly determined since, in addition to the narrowing, irradiation (on the surface and in depth) tends to blur the cutaneous aspect of localization in such a way that the body itself feels malleable, with diffuse boundaries.



**Figure 110.** Reduction of the body schema according to the spatial phases. In phase IV, the distal zones of the limbs are excluded. In III, there is only an anterior plane of embryonic morphology. In II, the schema is filiform. In I, there is no representation. As a whole, there is concentric reduction of the tactile field and functional dedifferentiation.

Functional narrowing indicates, as in other cases, a tendency to homogeneity in phenomenology, in contrast to the functional diversity of the normal sensory field (Fig. 110). Thus, irradiation tends to fill the field and not to stand out as a point. The narrowing of the body schema leads to the elimination of spatial values for a large part of the field, reducing the complexity of the spatial organization. An extreme case of homogeneity is the band of maximum irradiation without any spatial reference (phase I). However, a

certain structure or influence of the tactile field always persists. For example, in phase I, the shape of the irradiation (as a band) is like that of the tactile field (elongated).

Other aspects of this topic pertain more to the theory of localization and of spatial function in general, and although they are not very different from what is described above, they will be dealt with in another section.

## 22 Localization dynamics

### 22.1 Recruitment of phases in the localization process

After having exposed in detail the phenomenology of spatial asynchrony (or lag), as well as its fundamental characteristics, we can now undertake the study on the dynamic relations of excitability involved in the localization process. These relations describe the fundamental experience in the process of tactile localization by means of recruitment, the asynchronous beam, etc., relations already mentioned in the general part (Part I) of Vol. 1 and which were applied to several visual functions such as visual orientation, etc. As we know, such quantitative data are what give the most peculiar character to the brain dynamics presented here, allowing us to establish the physiological laws that govern the different brain functions.

Within the experimental possibilities, the curves to be studied refer mainly to recruitment processes and to the asynchronous beam of partial (out-of-phase) functions, using either a proper mechanical stimulus or electrical stimulation by capacitor discharge. Tactile measurements cannot in many cases reach the precision of measurements of other sensory functions, since the phenomena of irradiation and proximal deviation involved in tactile localization are difficult to measure accurately. Nevertheless, the data obtained in various tests are sufficient to give an idea of the localization process with both mechanical and electrical stimuli. [Altenburger \(1933\)](#) already warns of the difficulty of performing tactile chronaxie determinations under normal conditions by experienced authors; this is all the more so for pathological determinations.

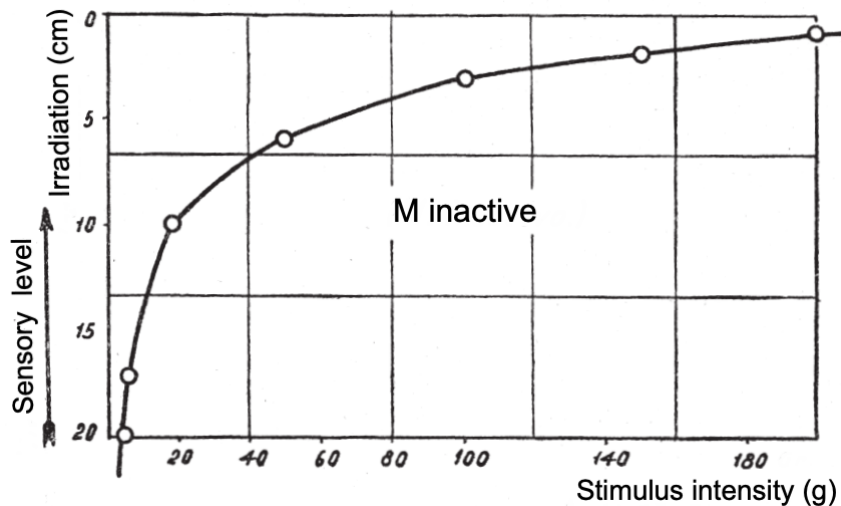
#### 22.1.1 Recruitment with single stimulus

By intensifying the stimulus, the functional reduction diminishes, and a sensory growth is obtained as a result of the recruitment of abnormal sensory intervals. Irradiation and proximal deviation of tactile localization then decrease simultaneously and correlatively, as we know. However, this is not an obstacle to independently consider these two phenomena, as will be done below, starting with irradiation.

Figure 111 shows the functional relationship between stimulus intensity and *irradiation* of the cutaneous sensation in subject M inactive, under gradual point stimulus on the dorsum of a hand. It is observed that with the initial increase of the stimulus, irradiation decreases rapidly and then more and more slowly. The different phases already described thus appear, i.e. irradiation in the form of a band, a smaller band with central point of sensation, an oval, a very small oval, and finally an almost pinpoint sensation. The data are mean values of several measurements. As already indicated, the extent of

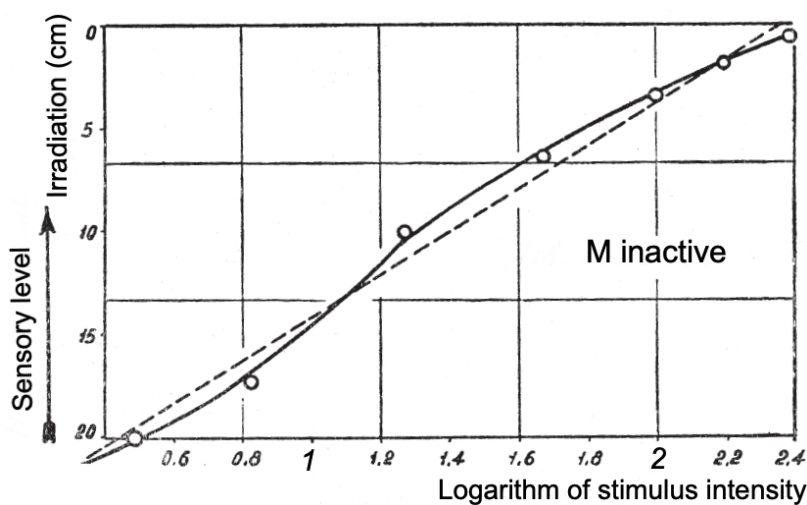


irradiation can only be appreciated indirectly, the patient noting down the extent he feels on a ruler. Thus, the curve shows the transition from an irradiation of 20 cm or slightly more to the minimum possible irradiation, in the inactive state and using a very strong stimulation. It seems impossible to rule out certain tactile irradiation residuals, difficult to evaluate accurately and which probably measure less than 1 cm.



**Figure 111.** Tactile irradiation (in cm) as a function of the stimulus intensity (in grams). Point stimulus applied to the dorsum of a hand of patient M inactive. The arrow indicates the growth of the sensory level, which is inverse to the growth of irradiation. Note that it is not possible to reach zero irradiation in the inactive state.

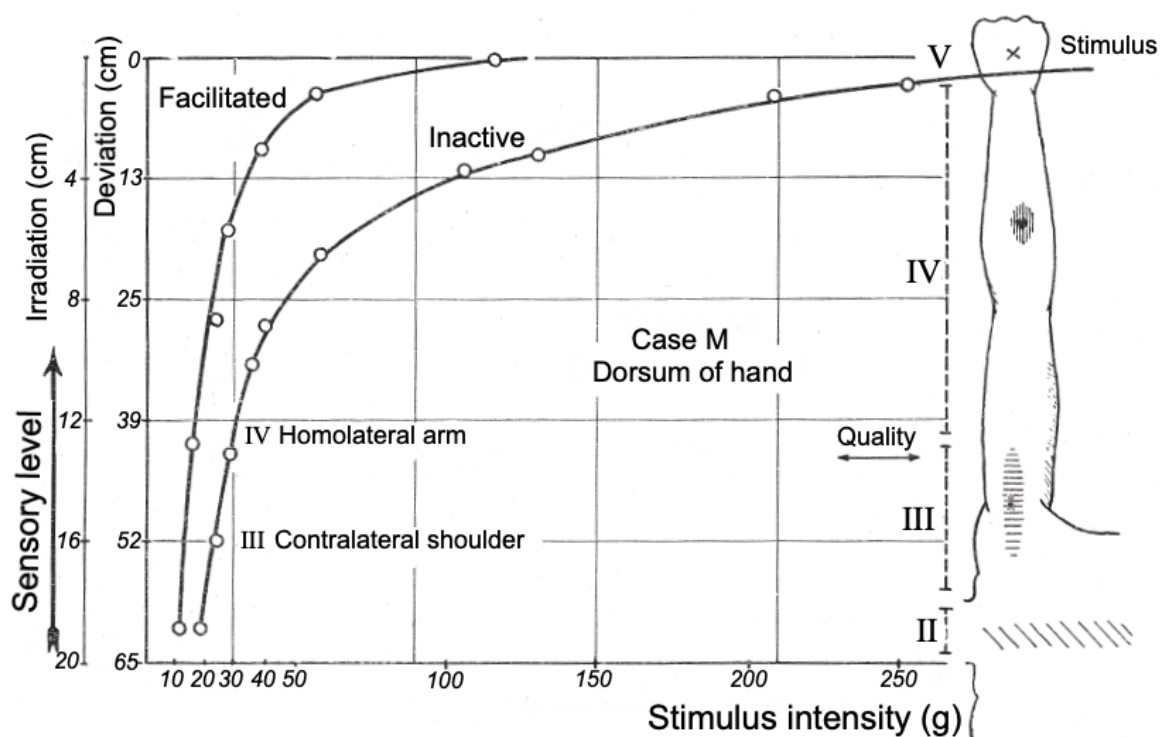
This curve follows the general behavior of sensory recruitment (e.g., in visual orientation). Taking the logarithm of the stimulus intensity, a slightly sigmoid line is obtained that approximates a straight line, showing the proportionality between sensation and logarithm of the stimulus (Fig. 112).



**Figure 112.** The same data as in the previous curve (Fig. 111), but taking the logarithm of the stimulus intensity. Almost a direct proportionality is obtained between sensory growth and that of the logarithm of the stimulus (Fechner's law).

As already indicated, tactile irradiation and irradiation in vision are similar phenomena, and the recruitment curve for tactile irradiation could also be applied to visual phenomena. Due to the inherent characteristics of irradiation in vision, a quantitative experimentation sufficiently complete to be expressed graphically is not possible.

The other important aspect of localization disorder is that of *proximal deviation*, which is the most interesting and complex aspect due to lateral inversion, body schema, etc. The functional relationship between the degree of deviation and stimulus intensity gives rise to a recruitment curve. To obtain this type of curve, lateral inversion has been ignored, indicating the distance as if the localization were always on the same side of the body, thus obtaining the curves shown in Fig. 113. The location of the perceived sensation cannot be determined as precisely as, for example, the degrees of inclination of the visual image. It is necessary to stick to verbal indications that allow only an approximate measurement (“in the middle of the arm”, “towards the shoulder”, etc.) or to proceed to indirect determinations on body models or using other aids. Nevertheless, the results thus obtained after several tests are good enough to illustrate the process.



**Figure 113.** Curves of proximal deviation for subject M in the inactive state and under facilitation by strong muscular effort, as a function of the intensity (in grams) of a point stimulus on the center of the dorsum of a hand. The deviation (in cm) is the distance between the real stimulus site and the subjectively felt site. The irradiation scale (in cm) allows to appreciate the parallel behavior of irradiation and deviation. The indication of the phases (from II to V) and of the body anatomy facilitates the understanding of the process. Note the different slopes of the curves in the inactive state and under facilitation, as well as their positions.

Figure 113 shows the process in subject M, both in the inactive state and under facilitation, using a practically point stimulus on the dorsum of a hand. The mechanical stimulus in this experiment is somewhat blunter than in the previous curves (Figs. 111 and 112) and that is the reason for a small difference in thresholds. The curve is of the same type as the previous one on irradiation, although it is not so perfectly simplified by taking the logarithm of the stimulus, which is attributable to the peculiar experimental difficulties. As shown in Fig. 113, proximal deviation and extent of irradiation evolve together, as can be seen in the corresponding scales. For any other part of the body, a similar recruitment must be admitted, but by choosing a distal part of a limb, there is both a wider field for measuring the deviation and an easier reference to indicate the site where the abnormal localization is felt. On the head and even more so on the trunk, many difficulties would arise for a measurement of this type; in the first case, because of the small size, in the second, because of the difficulty in finding references.

Since these measurements are susceptible to error, it is convenient to indicate the verbal method for obtaining the data, as shown in Tables 24 and 25. All these data are the mean values of the data obtained in several tests performed under as equal conditions as possible, using mainly the subject's verbal information to obtain the degree of deviation. This method seems to be the most appropriate given the patient's characteristics. In these tests only the sensory level (deviation and irradiation) and the intensity of the stimulus are involved, whereas the stimulation time is sufficiently long and is not taken into account, as in Fig. 111. For a given stimulus, the sensation reaches steady state in about seven seconds in the inactive state, and in about four seconds under facilitation. Even the first phases can be obtained under facilitation with a permanent stimulus if its intensity is suitably reduced. (See curves of the asynchronous beam further on).

**Table 24.** Subject M, inactive state. Experiment of Fig. 113. Stimulus applied for 7-10 seconds.

<i>Stimulus intensity (g)</i>	<i>Phase</i>	<i>Sensory deviation from application site (cm)</i>	<i>Sensory irradiation (cm)</i>
16	I		~ 22
19.5	II	~ 60 (Chest)	~ 19
25.5	III	~ 52 (Contralat. shoulder)	~ 15 Mild pain
27	IV	~ 42 (Homolat. mid arm)	~ 12
30	IV	~ 30 (Toward elbow)	~ 9
39	IV	~ 27 (Upper third forearm)	
59	IV	~ 19 (Mid forearm)	~ 7 Oval shape
109	IV	~ 12 (Two fingers above wrist)	~ 6
130	IV	~ 10 (Wrist)	~ 3 – 4 Strong pain
209	IV	~ 5 (Below wrist)	~ 2
250	IV	~ 1.5 – 1 (Still lower)	~ 1.5–1 Very strong pain

**Table 25.** Subject M under facilitation by maximum muscular effort. Experiment of Fig. 113. Stimulus applied for 4 seconds.

<i>Stimulus intensity (g)</i>	<i>Phase</i>	<i>Sensory deviation from application site (cm)</i>	<i>Sensory irradiation (cm)</i>
10	I		~ 20
12.5	II	~ 60 (Chest)	~ 17
17	III	~ 40 (Homolat. mid arm)	~ 12 Mild pain
26	IV	~ 27 (Upper third forearm )	~ 8
29	IV	~ 19 (Mid forearm)	~ 6 Oval shape
40	IV	~ 11 (Wrist )	~ 4
59	IV	~ 3 (Hand)	Like a lentil
109	V	Without deviation ?	~ 0 ? Bearable pain

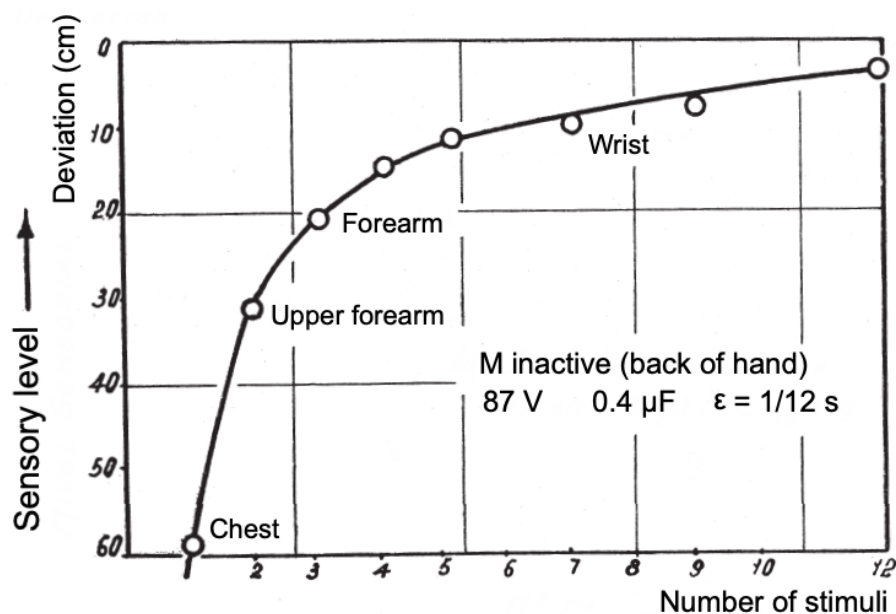
In Figure 113 we can appreciate the different thresholds and slopes of the curves for the inactive state and the state under facilitation. Given the rapid initial growth of the sensory level, the first phases of localization show extremely small transit intervals and can only be individualized by very careful experiments. For this reason, phases II and III may initially remain unnoticed. By contrast, phase IV, which corresponds to a slow sensory increase, is very large and easy to obtain even in the T case. Although the first two phases can be obtained in M under facilitation, phase III seems impossible to be appreciated, at least with maximum facilitation, given the sharp slope. The patient suddenly passes from phase II to the onset of phase IV, and contralateral deviation is never obtained, the first two phases remaining very close to the onset of phase IV. It also happens that phase IV is much shorter under maximum facilitation than in the inactive state, about half, and since the asynchrony is lower, the abnormal phases are much more reduced. During this sensory recruitment, the intensity of the sensation also varies, which at first is like a weak pressure, and with much greater stimulus the sensation is painful, presenting a highly increased tango-algic interval, as we already know. The quality of pain appears towards the end of the third phase in the inactive state, and at the beginning of phase IV with facilitation, in both cases the localization is approximately towards the middle of the arm. With facilitation, the curve reaches the normal localization (phase V), but in the inactive state, some abnormal residual remains which seems impossible to overcome no matter how much the stimulus is intensified.

From the arrangement of the curves in Fig. 113, we can appreciate the stimulus saving involved in the facilitated state. However, the normalizing action of facilitation is limited, and by using a stimulus to obtain phase I or II in the inactive state, the action of facilitation only makes possible to reach the onset of phase IV. This means that a stimulus that is not at all localized, or localized on the chest, moves to the elbow or more distally, i.e., from about 60 cm of deviation it moves to 30 cm of deviation, and sometimes less.

This type of recruitment was not determined in subject T, but given his characteristics, it can be admitted that his curve would be above that of M with facilitation and with a greater slope (see further on).

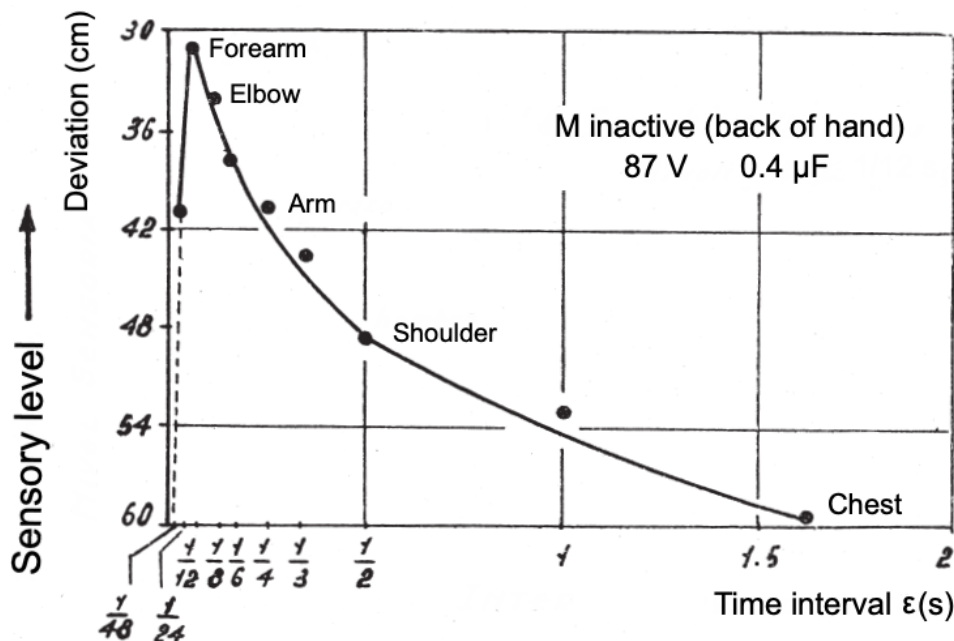
### 22.1.2 Recruitment with iterative stimulation

Given the high iterative capacity of subject M, another type of spatial tactile recruitment experiments can be performed by iteration instead of using a single stimulus. The curves in Figs. 114 and 115 correspond to this type of stimulation according to the number of stimuli and the interval between stimuli, respectively. For iterative stimulation, electrical stimulation is more precise and appropriate. The active electrode of other tests is applied on the back of a hand, and the characteristics of the excitation are conveniently chosen to obtain the most evident effect possible. Figure 114 shows that iterative recruitment in the inactive state is very large, but while achieving a much greater effect than facilitation with a single stimulus, complete recruitment is not achieved, although it comes very close to it. This experiment begins already in phase II with a single stimulus to have a higher sensory level. Using very short stimuli in relation to the subject's excitability characteristics, it is necessary to have a very high voltage. For an inter-stimulus interval of 1/12 second, the proximal deviation decreases as the *number* of stimuli increases, first rapidly and then very slowly.



**Figure 114.** Iterative recruitment of spatial localization, i.e., decrease of centripetal deviation as a function of the number of stimuli in M inactive. The characteristics of the stimulation are shown in the figure.

With two stimuli, the sensory growth is so strong that phase III of contralateral localization is eliminated, and the sensation shifts from the chest (phase II) to the homolateral elbow or lower (phase IV). After a number of stimuli (12 or more), the limit of sensory recruitment is reached, and a residual asynchrony remains that cannot be overcome. If the experiment were started at the threshold corresponding to phase I, the final sensory level would be even lower and localization would not go beyond the wrist.



**Figure 115.** Spatial recruitment (localization deviation) as a function of the time interval ( $\epsilon$ ) between two stimuli. Intensity and duration of a single stimulus as in Fig. 114. The maximum effect of latent addition occurs with an interval of 1/12 s, becoming null when the interval is about 1.5 s. With intervals shorter than 1/12 s, recruitment tends to decrease, since the refractory period of the first stimulus should intervene, making temporal summation difficult.

A complement to the previous experiment is obtained with the test corresponding to Fig. 115, with the same electrical excitation conditions as in Fig. 114 but now the sensory level is a function of the time *interval* between two stimuli. The highest point of the curve corresponds to the sensory level for an interval of 1/12 s, and the localization is toward the upper part of the homolateral forearm, as in the preceding Fig. 114. However, as the time interval increases, recruitment becomes less and less, passing through the contralateral phase III, obtained in its full amplitude, until reaching the chest (phase II) with the same effect as with a single stimulus. Due to the exponential decay of the excitation of a single stimulus, if the interval  $\epsilon$  increases, the second stimulus adds to a smaller and smaller residual of the excitation of the first stimulus that seems evident ends after about 1.5 s in inactive M. If the interval  $\epsilon$  is reduced to less than 1/12 s, the sensory level does not increase but remains constant or rather tends to decrease. Since the iterative cylinder (the mechanism used in these tests) does not allow rigorous control of intervals of about 1/48 s and smaller, it has been impossible to adequately study the action of very brief intervals. In any case, it can be admitted that the decrease in the degree of summation with extremely short intervals is due to the fact that the second stimulus falls on the refractory period of the first stimulus (relative refractory period). It should be noted that as the chronaxie increases, the refractory period increases as well, thus, within the uncertainty of the present curve at very small intervals, it would perhaps be possible to estimate the refractory period for electrical stimulation at around 1/24 to 1/50 s on average. This interval-dependent summation test is similar to that of [Bremer \(1930 a\)](#) by

exciting the nerve of a muscle under the moderate effect of curare, which confers to the muscle the condition of an absolutely iterative organ. Depending then on the interval between only two stimuli, the contraction recorded varies reaching a maximum and two minima (null contraction). One of these minima is for a very small interval (absolute refractory period) and the other is for a large interval (addition time limit). Other methods have been adopted by different authors.

In relation to the effects of iteration on tactile localization recruitment, it is worth noting briefly the *mechanical vibratory stimulation* with different tuning forks in subject M. A normal subject perceives the vibration of a 512 Hz tuning fork applied to the styloid process of the radius for at least 8-9 seconds, after having strongly struck the tuning fork. However, in subject M, due to his excitability deficit which increases the refractory period thus discarding high frequencies, it is necessary to use a 32 Hz tuning fork and apply maximum facilitation to obtain a similar result (see Table 21). This means that only low-frequency tuning forks can be used to obtain sufficient summation effect. In this test the number of stimuli is not taken into account (they act for a sufficiently long time) but the interval between stimuli (frequency) is decisive, as in the electrical stimulation experiment shown in Fig. 115. Thus, the sensory level (localization phase) depends on the tuning fork frequency used, as shown in Table 26.

**Table 26.** Case M. Localization depending on vibration frequency of tuning fork on radial styloid process.

<i>Frequency (Hz)</i>	<i>Inactive state</i>		<i>With facilitation</i>	
	<i>Phase</i>	<i>Localization</i>	<i>Phase</i>	<i>Localization</i>
32	IV	Upper third forearm	IV	Two fingers above wrist
64	IV	Upper third forearm	IV	Two fingers above wrist
128	IV	Elbow or higher	IV	Forearm
256	III	Contralat. shoulder	IV	Middle homolat. arm
435		No sensation	II	Chest (fusion)

A tuning fork of 32 Hz in strong vibration is perceived tactilely for 7-8 seconds by subject M in the inactive state, although only half of that time corresponds to vibratory sensation (see Figs. 89 and 90), which appears in phase IV of localization (upper third of the forearm); the rest of the time, he only feels stimulus fusion, hence some pressure uniform in time, and spatially, he does not go beyond phase II or less.

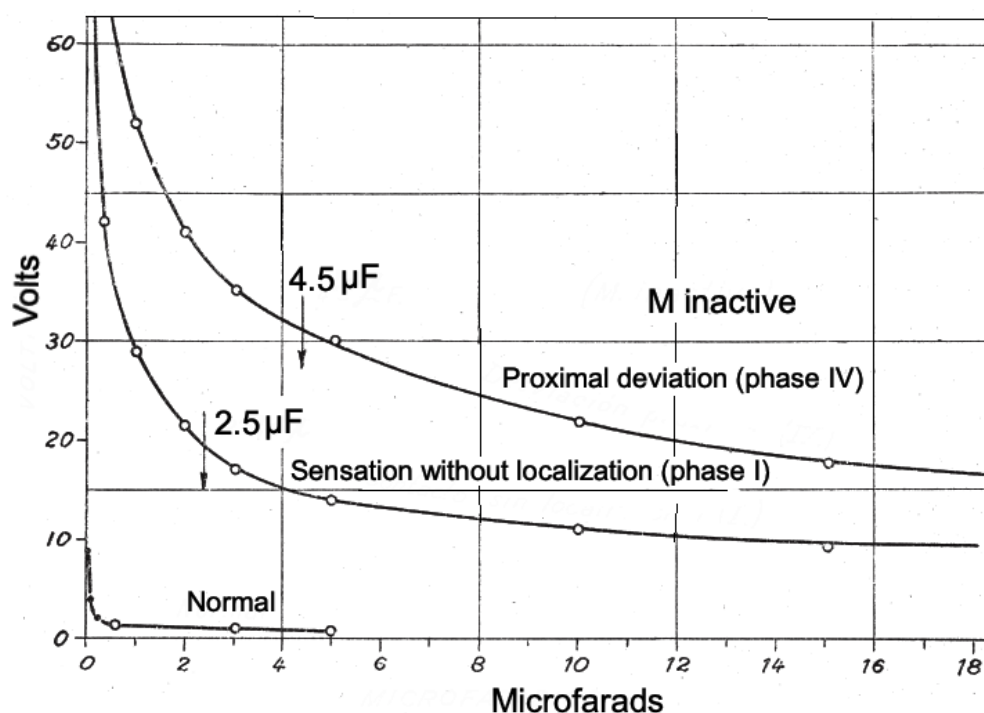
From the data in Table 26, it can be seen that the recruitment of localization by means of tuning forks is very small, which indicates that the frequencies, even being in the low range, are too high for the excitability characteristics of subject M (increased chronaxie and therefore increased refractory period); therefore, only under facilitation by maximum muscular effort and with the 32 Hz tuning fork, the proximal deviation is reduced to a minimum. For higher frequencies (i.e. very small interval), the proximal deviation is very large, especially in the inactive state. The decrease in sensory recruitment should then be understood as an effect of the refractory period, which reduces or prevents iterative summation. At least for the inactive state, it is presumed that the tuning forks indicated

in the table act within the relative refractory period, and the last of them (435 Hz) even in the absolute refractory period. Thus, there can be no latent addition or temporal summation of any kind, and the sensory effect is null since not even phase I of spatial localization is reached.

Vibratory sensation appears at the onset of phase IV, the presence of sensation during phase III being questionable since a weak vibration seems to be initiated in the contralateral shoulder in inactive M at 256 Hz. In short, localization worsens as the vibration frequency increases, also increasing the perceived vibration speed which is always greatly overestimated (Fig. 89). Thus, the greater the tendency to fusion, the greater the alteration of spatial localization i.e. the greater the deviation.

## 22.2 Asynchrony in spatial localization

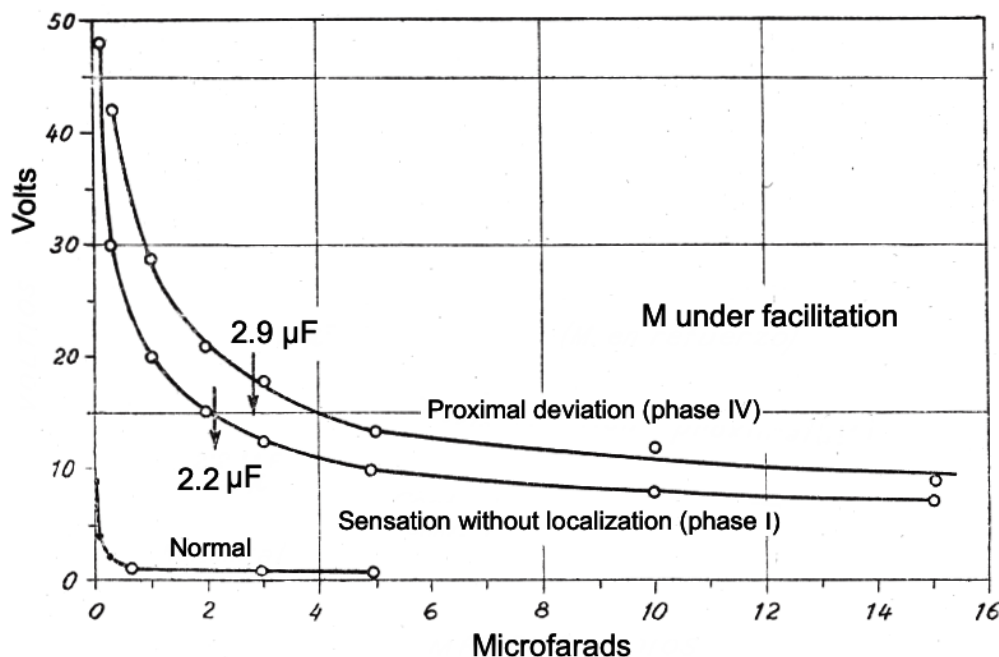
To complete the study of asynchrony, let us analyze the strength-duration curves referring to the most significant phases of the abnormal localization, i.e. the asynchronous bundle of curves corresponding to the different sensory levels. The determination of these curves is much easier with electrical stimulus than with mechanical stimulus, given the need to measure time.



**Figure 116.** Strength-duration curves for phase I of localization and for the onset of phase IV, in subject M inactive. Lip stimulation according to the characteristics of previous electrical tests (Figs. 82 and 83). Note the large excitability interval (asynchrony).



Figure 116 shows the strength-duration curves for two quite different spatial localization phases in subject M inactive: phase I, without localization, and phase IV of proximal deviation. Comparing these two curves with those corresponding to M under facilitation (Fig. 117), it can be seen that in the latter case the curves have lower values, i.e. the excitability for these phases has increased, and the interval between them is markedly reduced.

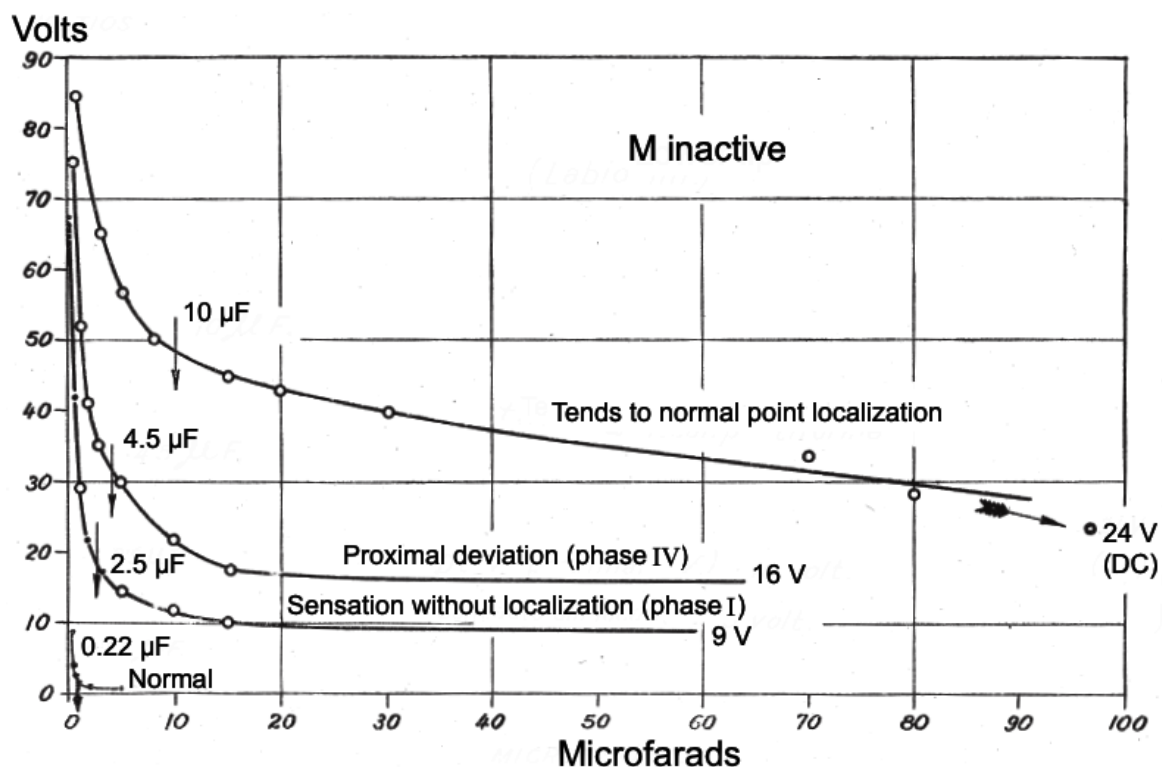


**Figure 117.** Strength-duration curves for phase I of localization and for the onset of phase IV, in subject M under facilitation by maximum muscular effort. Note the decrease in the chronaxie values of both curves, mainly of the upper one, which results in a remarkable closeness of the phases to each other reducing the interval between them. Test conditions similar to those in Fig. 116.

From the chronaxie values in Figs. 116 and 117, it can be noted that the chronaxie interval with facilitation ( $0.7 \mu\text{f}$ ) is one third of the chronaxie interval in the inactive state ( $2 \mu\text{f}$ ). We also see that the approximation of the levels (phases) to each other is obtained mainly at the expense of the increased excitability of the most out-of-phase sensory level (phase IV). It is important to insist on the action of facilitation on asynchrony, since it reveals the physiological mechanism of the nervous alteration, which consists in the fact that the higher levels become more altered than the lower levels as the excitability deficit increases. In both Figs. 116 and 117, as in most tactile stimulations by capacitor discharge, the lip has been used since it allows the best voltage utilization. The curves for simple contact (phase I) are therefore the same as those in Fig. 82 on general excitability. Likewise, the curves of the higher sensory level (phase IV) in both Figs. 116 and 117 correspond to the initiation of the tactile sensations, which show a tango-algic interval with respect to those of the lower level, being the same curves as those in Figs. 91 and 92.

As can be seen from Figs. 116 and 117, facilitation tends to convert the simple sensation curve (phase I) of the inactive state into a curve of proximal deviation (phase IV), as indicated in previous sections about the change of sensory level by facilitation. The sensory level only rises until the beginning of phase IV, with a wide margin of asynchrony still remaining and therefore being far from reaching normalization. The subjective localization corresponding to the described phases when the labial mucosa is electrically stimulated is as follows. For phase I, no localization as we already know; for phase IV, the localization is towards the temple or at most on the upper part of the cheek on the same side of the stimulation (stimulation on the labial commissure), therefore with marked deviation towards the vertex.

A more complete display of the asynchrony in case M is shown in Fig. 118, comprising from phase I to the end of phase IV, which is all that can be determined in M inactive. To the two curves of the phases previously studied, the highest and final curve is now added, which is very close to the normal localization, corresponding to the lower part of the cheek, close to the stimulation site on the lip and with a weak irradiation. The separation between this high curve and the one corresponding to the onset of phase IV determines the extent (amplitude) of phase IV.



**Figure 118.** Electrical stimulation on the lower lip of M in the inactive state. Strength-duration curves for the different phases of spatial localization. The separation between the lowest and highest curves indicates the degree of asynchrony. The separation between the two highest curves indicates the extent (amplitude) of phase IV. Note the diversity of values compared to the normal curve that includes all phases in itself.

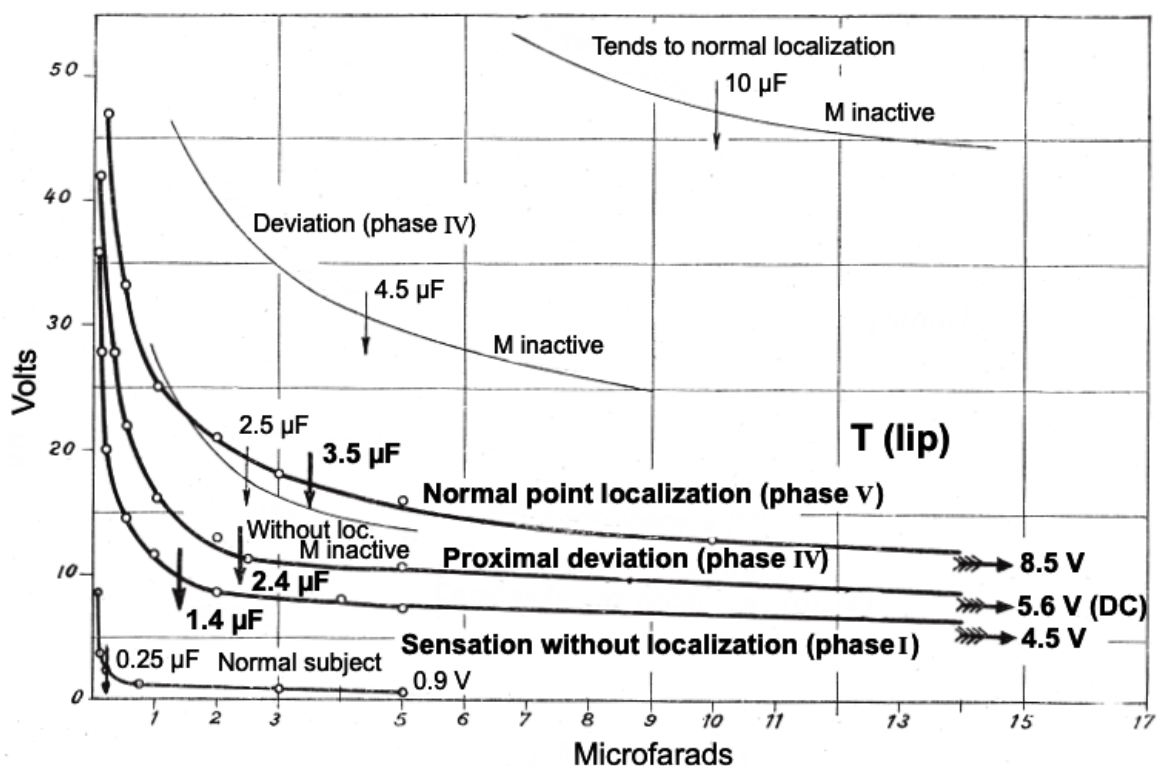
The *abnormal sensory interval* from phase I to point localization is of course much wider than that from phase I to the onset of phase IV. Phases II and III are not represented

in Fig. 118 since this figure as well as Figs. 116, 117 and also 119 for subject T were determined in the first half of 1944, when we were still unaware of the existence of phases II and III, whose values, differing little from the adjacent phases, made the identification of these intermediate levels difficult.

From the set of curves in Fig. 118, we note that whereas in a normal subject an all-or-none effect is obtained (localization without irradiation nor deviation, or total lack of sensation), in M inactive, the normal function is decomposed, according to the stimulation, into *partial reactions* (phases) with a marked deficit of excitability. This deficit is considerably accentuated for the partial phases of higher functional level, as already mentioned. Thus, with respect to the chronaxie for normal function, the chronaxie for simple contact (phase I) is about ten times greater, for the onset of phase IV it is twenty times greater, and for the end of phase IV about fifty times greater. As for phase V (total normalization), the increase would be indefinite since the asynchrony in M inactive cannot be completely overcome.

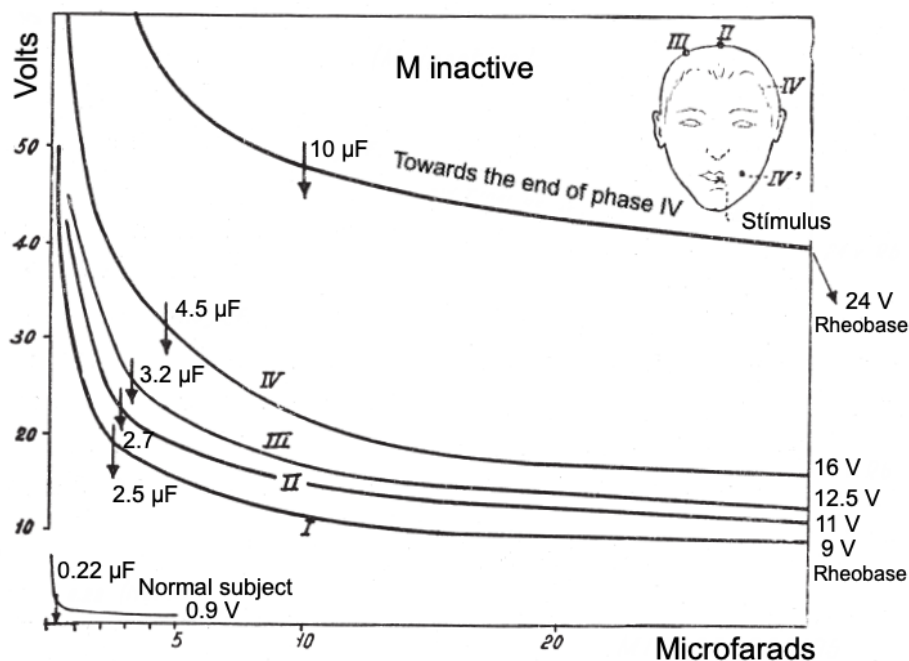
Although a direct comparison between Fig. 118 and Fig. 113 is not possible since the stimulation is at different locations and of different nature, the correspondence regarding the amplitude of the different phases is remarkable.

Subject T was also examined in this respect in early 1944, and the asynchronous curves of Fig. 119 were obtained. Although asynchrony in T is much less than in M inactive, it is sufficient to determine electrically different phases.



**Figure 119.** Strength-duration curves for the three existing spatial phases in subject T (thick lines). Experimental conditions similar to those of subject M. Compare with the curves for subject M inactive (thin lines), partially depicted.

In subject T, the contact phase I without localization can be individualized even though it is more excitable than in M with facilitation, and the separation between the phases is small in comparison with M inactive. In T, normal localization is easily achieved, as in M under facilitation. In M, reversed (contralateral) phase III is absent, at least with maximal facilitation (Fig. 113), and should also be absent with more reason in subject T although he was not examined in this respect. Phase II, present in M under facilitation, would also be excluded in T because of very rapid sensory variation. Therefore, only phases I, IV and V were determined in subject T. The arrangement of the curves is approximately the same as in M; that is, the excitability deficit is greater in the higher sensory levels. Likewise, the onset of the qualities takes place at the onset of phase IV (see Fig. 93). It should be noted that the distribution of the sensory defect is not completely symmetrical in T, the right side (contralateral to the lesion) being somewhat more affected than the left. The data in Fig. 119 refer to measurements at the midline of the lower lip (to take a midpoint). The action of facilitation in T, although much weaker than in M, reduces asynchrony and also allows passage from phase I to phase IV. Finally, it should be noted that subject T can show a much greater asynchrony as a consequence of epileptic seizures, as was indicated when studying vision. In such circumstances it was observed that his state of excitability was intermediate between M inactive and M under facilitation, tending to return to the usual state within a few days. Similarly, other modifications of excitability due to the effect of alcohol, or brain cooling, etc., already mentioned in relation to the tilt of the visual image, must also affect tactile functions for the same reasons as in the postictal state.



**Figure 120.** Strength-duration curves for all the phases that can be determined in subject M in the inactive state, stimulated on the left labial commissure. Note the great asynchrony and the deficit of excitability in relation to the curve of the normal subject, as well as the small differences

between the lower phases (I, II, III), which make their determination difficult. On the upper right a diagram shows the localization of the different phases with deviation towards the vertex.

To conclude, a more complete representation of all possible phases in subject M inactive is given in Fig. 120, where the curves for phases II and III, difficult to determine in their entirety because they show values very close to each other, have been added. These latter curves were obtained after 1944, and the others maintained the values in the repeated tests up to that time. The figure shows the respective localization deviations on the head of the different phases when stimulating on the left labial commissure.

The curves correspond to the thresholds of the respective phases, that is, to the onset of the phases. The localizations indicated on the head are as precise as possible in an experiment of this nature. They are: for phase II, on the vertex or upper part of the cranial calotte; for phase III, on one side of the calotte, contralateral to the stimulated labial commissure; for phase IV, on the homolateral temple, at the limit of hair growth; and for phase IV (the highest sensory level attainable), the contact is perceived towards the lower cheek, close to the point of stimulus application.

### 22.3 Temporal development

Some brief comments about time in both a) spatial sensory development and b) certain summation effects, will conclude the study on dynamic relationships.

a) Due to the slow reaction time, the development of a given sensory level can often be sensed through the different phases by the examined subject himself if he pays special attention to it. Thus, by using a weak but sufficiently intense stimulus to reach a well-developed phase IV, subject M inactive takes 1-2 seconds to reach phase I and a further 2 s to reach phase IV (a total of 4.5-5 s for phase IV). During this time, he can sense the change in the extent of irradiation and the change in localization. However, the transition from phase I to phase IV is abrupt, the intermediate phases being excluded. In order to obtain a more gradual development, it is necessary to resort to iterative stimulation and select the intermediate phases with a certain number of stimuli. For example, with mechanical stimulation by means of a piece of soft cotton, phase I is obtained after two or three successive contacts, phase II after the fifth or sixth contact, phase III after the tenth, etc. Undoubtedly, this type of development can be obtained more accurately and completely by electrical iteration, and we refer the reader to the corresponding curves presented in Section 6.1.2. Also, from the curves for the different phases (especially from Fig. 120), the *temporal development of the spatial process* can be inferred for a stimulus of sufficient intensity to reach the highest sensory level. The reaction speed of course increases in M with facilitation and even more so in the T case. For ease of understanding, this can be related to the time evolution of visual image orientation (Figs. 62 and 63).

A reverse development can also be obtained by studying *sensory degradation*, either by the fatigue process with permanent stimulation, or by residual persistence after removing the stimulus. In both cases the results are quite similar. Using the minimum stimulus necessary to obtain a well-developed phase IV, and keeping the stimulus

unchanged, it results that after approximately one minute of having reached phase IV, it disappears abruptly and leaves a sensation corresponding to phase I which is still maintained for a few minutes. Depending on the intensity of the high phase reached at the beginning of the test, the process during *fatigue* is different; sometimes the final phase I seems to be maintained indefinitely while other times it ends up disappearing, although much later. Fatigue progressively and gradually raises the excitation threshold, therefore the high functional level reached tends to present dynamic reduction through asynchrony.

The fading of the persistent sensory residue in the absence of stimulus occurs as follows. Once the stimulus that has allowed reaching a well-developed phase IV has been removed, the sensation persists only for a brief moment, passing immediately and abruptly to phase I, being impossible to notice inversions and intermediate phases. The persistence does not last in total more than 2 seconds at most, and is on average about 1.5 s for the two phases mentioned above. In this process of temporal degradation, a certain *sequence of phases* is obtained which, as in the temporal development, is limited to the most accessible phases I and IV, given the brevity and instability of the process.

b) It is now worth mentioning the action of facilitation on the residual sensation once the stimulus removed, which causes the remarkable phenomenon of *post-stimulus summation*, already studied in M since 1940 both in vision (effect on the inclination of the visual image) and in touch (spatial localization). The effect certainly takes place as long as the time interval between the removal of the stimulus and the application of facilitation (strong muscular effort) is very small (less than 2 s), otherwise the facilitation has no effect because the residual sensation has vanished.

Facilitation exerts a summative action on the persistent sensory residual and intensifies it for brief instants, raising it even to a sensory level higher than that produced by the stimulus. Of course, the degree of this increase is a function of the above-mentioned time interval, all other experimental conditions being constant. For example, when M inactive has a constant sensation of simple contact without localization (phase I) and then the stimulus is removed, the sensation lasts slightly more than 1.5 s. Thus, depending on the delay in the application of facilitation, different sensory levels are reached as shown in Table 27.

**Table 27.** Sensory levels reached according to the delay (in seconds) in applying facilitation after removing the stimulus. The stimulus is a 19 g permanent weight on a 0.7 mm diameter surface on the dorsum of a hand of M inactive, triggering phase I only. Simultaneous application of maximal facilitation would result in sensation at the homolateral elbow (advanced phase IV).

<i>Facilitation delay (s)</i>	<i>Sensory level</i>
0.1	Homolat. elbow (phase IV), same as no delay
0.2	Homolateral arm (onset of phase IV)
0.9	Contralateral shoulder (phase III)
1	Chest (phase II)
1.5	Only phase I
More than 1.5	No sensation

In this reactivation process, the time that the subject under examination takes from the moment he receives the order until he makes the muscular effort should also be taken into account; this time is usually fragments of a second and is somewhat variable according to the subject's degree of attention and other circumstances. Table 27 shows that the sensory level decreases as the time interval between stimulus removal and the application of facilitation increases, since the sensory residual of phase I decreases rapidly until it disappears completely shortly before 2 s. For long time intervals (1 s or a little more), facilitation acts on a subliminal residual that is below phase I and, therefore, facilitation only manages to elevate the residual to phase II, or even lower, restoring phase I. It is understandable that in all cases the level reached is of short duration, since the tactile stimulus has been removed.

The characteristics of this post-stimulus summation by facilitation are very similar formally to those of the experience in Fig. 115 (sensory level as a function of the interval between two identical electrical stimuli) although the experimental conditions are different. In both cases a certain sensory level is recruited from the residual trace of a previous stimulus. Such a residual trace is activated either by a second identical stimulus or by facilitation. In both cases, the maximum time interval for the summation effect to exist is of the same order (about 1.5 s), and even the degree of sensory enhancement is very similar, despite the different type of stimulation in each case. The fundamental difference lies therefore only in the type of summation, direct for iteration (*temporal summation*) and indirect for facilitation (*spatial summation*), the effects of both being equivalent. Whereas in temporal summation the two stimuli are of the same nature and travel to the nervous centers using the same pathway, in spatial summation the two actions (stimulus and facilitation) not only follow different pathways, but in addition, facilitation involves a nonspecific central action. In short, the residual imprint of a first tactile stimulus can be activated, and even raised above its corresponding level, by very different nervous actions.

Finally, some brief comments are pertinent about facilitation as a synchronizing action of desynchronized sensory levels. The transition to a higher tactile sensory level by facilitation due to muscular effort has been dealt with descriptively and graphically (Fig. 113), and the analogy with the behavior of the visual function with respect to facilitation (enlargement of the visual field, correction of the orientation of the visual image, etc.) can easily be admitted. For this reason, we do not add for the moment the corresponding graph of tactile sensory growth as a function of the weights held by subject M, since it is a great inconvenience (although not an impossibility) to check how the subject, holding weights up to 80 kg, perceives an improvement in the sensation of a constant weak tactile stimulus. It is also possible to state that recruitment as a function of facilitation (by muscular effort) has similar characteristics to recruitment as a function of stimulus intensity, noting that synchronization by maximum muscular effort is not complete and there is always an important residual that seems to be much greater than in the case of stimulus increase.

Concerning other non-muscular facilitations, namely cross-modal actions, only touch (bi-tactile effect) is of some importance. Thus, a pronounced mechanical pressure on the skin, or brushing gently over the skin, reinforces the sensation awakened by a weak

tactile stimulus elsewhere, raising its level, reducing the irradiation, etc. This fact is of particular interest when studying spatial discrimination between two stimuli, as will be seen further on. On the other hand, it is difficult to demonstrate the summation of visual and auditory facilitations on touch, although perhaps auditory facilitation is somewhat clearer, being issues that still need to be investigated

## 22.4 Theory of tactile localization. Spatial organization

Although the theoretical issues will be developed in depth in the last part of this research, we cannot fail now to make some comments on tactile localization, taking into account all that has been exposed about our cases, and also considering the Schneider case of [Goldstein and Gelb \(1919\)](#), according to the interpretations of these authors and the theories of others. All this will serve to establish as soon as possible the properties of spatial *organization* that can be deduced from our research.

Clinical neurology has contributed little to the elucidation of this issue, i.e., to the physiological mechanism of tactile localization. At most it is said ([Head and Riddoch 1920](#), [Head et. al 1920](#)) that the diffuse or incorrect localization provided by the nerve fibers of protopathic sensitivity is improved and refined by the special nerve fibers of epicritical sensitivity. The issue is thus focused on the anatomical specificity of the conducting fibers which, besides being a problematic hypothesis, leaves the problem of the mechanism in question untouched.

It is in the domain of theoretical psychophysiology that this problem develops and where the theories most in conflict with each other are conceived, namely ‘empiricism’ or ‘genetism’, and on the other hand ‘nativism’. For the founders of empiricism ([Lotze 1852](#), [Wundt 1862, 1874](#), [Helmholtz 1896](#)) the “local sign” arises *secondarily* through the association of cutaneous sensations with muscular movements in the course of life; and also, the images and visual representations of the body collaborate to it. This interpretation, more or less modified, reaches [Henri \(1898\)](#) and from there reaches [Goldstein and Gelb \(1919\)](#) who apply it to solve the problem posed by their Schneider case. In contrast to them, the nativist trend appears with [Hering \(1874/1878, 1880\)](#) and especially with [Stumpf \(1873\)](#), arguing that the spatial structure is *primarily* inherent to the sensation. Later, it became very significant the development and acceptance of the concept of extensity as an immediate phenomenic datum, considering it an attribute of sensation ([Külpe 1893/1909](#), [Titchener 1910](#), [James 1890](#)). Likewise, on the part of the Gestalt theory, [Wertheimer \(1912\)](#) and [Koffka \(1919/1935\)](#) clearly argue that extensity is as primary as quality, and that shape is a mere mental organization given in the extensity. As these orientations become more and more prevalent, there is a tendency to admit that the perceived space is in correspondence with the spatial relationships established in the nervous system independently of learning.

Turning our attention now to the concrete examination of the facts, we will first deal with the controversy about the Schneider case in relation to the pathophysiological problem of spatial localization, following the attempted interpretation of [Goldstein and Gelb \(1919\)](#) and other authors. The characteristics of this case are already known to us, and now we shall only refer to the way of achieving localization by means of the particular



“muscular twitches”, according to these authors. These authors distinguish between normal or “voluntary” localization and “automatic” or reflex localization, following [Henri \(1898\)](#). According to this author, in the first type (voluntary), secondary auxiliary means come into play such as the visual representation of the touched place, which is never lacking, and a special attention to the quality of touch [“genaue Beachtung der Tastqualität” [Henri \(1898\)](#)]. The second type involves localization movements of a finger probing, and contact sensations when probing with the finger. It is admitted that these localization movements have a congenital reflex character, seemingly of a spinal cord nature, providing only a very rough localization, corrected in part by the referred contact sensations which by trial and error guide the localization. [Goldstein and Gelb \(1919\)](#), without judging the obscure statement of [Henri \(1898\)](#), take advantage of the automatic localization to attempt an explanation of their case, in addition to the general conceptions (visual influence on tactile space, etc.) on which they base the tactile disorder. Thus, as soon as subject Schneider feels contact (perceives that he is touched), he automatically performs movements or small muscular twitches or jerks of very short amplitude, all over his body, reaching a moment in which, by colliding with the applied stimulus, the referred movements are more specifically directed towards it. The authors then think that a kind of “kinematic quality”, completely hypothetical, must intervene, leading to a certain sensation of location, or rather a kind of guidance of the body, according to the authors. In fact, the authors state that, even in the case of achieving localization, the tactile spatial structure is still absolutely absent; and likewise in vision, during movements following the contours of objects, a true sensation of form would not take place. Thus, in both touch and vision, the function occurs indirectly but without being achieved in reality. Without going into details, it is clear that the authors' interpretation seems non-transparent or very arbitrary (qualitative specificity of the movements), and even contradictory (the patient can localize perfectly but lacking a spatial notion), as recognized without exception by all authors, psychologists, neurologists, etc. who have commented on this issue.

[Goldstein and Gelb \(1919\)](#) admit that tactile space takes place only through visual representations of the body supported in turn by movement sensations. This is the genetic point of view defended and most extensively developed by [Henri \(1898\)](#), whose origin goes back to [Lotze \(1852\)](#), [Wundt \(1862, 1874\)](#), etc. [Goldstein and Gelb](#) think that given the lack of visual space in Schneider, touch suffers in this subject a “transcortical alteration of sensitivity” which they claim to have demonstrated for the first time. Finally, ascribing a character of generality to their conclusions, they maintain that there is no space other than visual space. This opinion was formerly held by [Platner \(1793\)](#), revived by [Hagen \(1846\)](#) and defended again after the Schneider case. However, the consequences of this opinion for the congenitally blind, to whom all spatial perception would have to be denied, were impossible to be maintained even by [Goldstein and Gelb](#) who have attempted to establish a certain parallelism with their Schneider case. A fully recognized fact in this type of blind people is precisely the ability to sense tactile space, therefore independent of any visual influence. Regarding the formation of tactile space in a normal subject, [Goldstein and Gelb](#) accept a pronounced empiricism, and thus, in children, only automatic localization would be present, which would gradually give way to voluntary

localization. Therefore, during the first stage of their development humans would have a tactile state similar to that of the Schneider subject.

In the Schneider patient, it has been rightly reached to distinguish his different behavior in the two different states, that of rest (inactive) and that of muscular twitching. On our part, we know from our research that this can be explained, for vision as well as for touch and for any other activity, by simple excitability relations, and that muscular twitching is a central summative facilitation. This summative action can equally well be achieved by immobile muscular tension or by any other type of facilitation. On the other hand, the authors have lacked knowledge of the phenomenology of localization (stages according to irradiation and proximal deviation) which provides essential data to address the spatial problem. Regardless of our findings, we have followed the course of the ideas of [Goldstein and Gelb \(1919\)](#) in their attempt to explain the Schneider case, to show that their explanation is not possible. Paying attention to certain particularities, the contradictions become even clearer. Thus, it seems that in the supposed automatic localization, spatial guidance is possible thanks to the hypothetical kinesthetic quality mentioned above, namely, to muscular movements that are guided towards the stimulus, which is casually found by the sensation of increased pressure. However, it is impossible that, normal localization being abolished, the ability to perceive joint movements and others still exists, since it is known ([Head et al. 1920](#)) that joint movements are much more severely impaired than any other spatial function. The specific features of motion in touch will be studied later, but in Sec. 21.2 we saw that motion, either on the skin or involving joints, appears only weakly in phase III (inversion). Some peculiarities are also indicated in Sec. 21.1. If we apply this criticism on the perception of joint movement to the contouring movement around objects for visual perception of them, the explanation given by [Goldstein and Gelb \(1918\)](#) for vision is also problematic. As for the hypothesis about the specificity of those movements that seem to have the property of spatial localization, no fundament or effective proof has been provided. Concerning the way of achieving localization (or as it should be called according to the authors' thought), it is not understandable. For instance, the kinematic quality would awaken in the subject the indication 'arm'; he does not know *where* the arm is but abstractly (?) he knows that it has something to do with the arm.

[Goldstein and Gelb \(1919\)](#) incorporate normal localization into automatic localization, probably impressed both by the spasmodic muscular twitches of subject Schneider in order to localize and by the ideas of [Henri \(1898\)](#) which, in principle, seem to fit perfectly with the interpretation of the subject's behavior. However, they feel forced to postulate in addition the existence of a "kinematic quality" in the aforementioned muscular twitches, so that some movements would be qualitatively different from others, that is, they would carry in themselves significance of location. This means admitting local sign for the muscular twitching. But then, why not admit in a primary way the cutaneous local sign, i.e. the static quality, which is much simpler? According to [Henri \(1898\)](#), from automatic localization (a mere spinal reflex of a rough spatial character), there would be a transition to voluntary localization, more perfect, thanks to learning through experience by means of a functional transition that does not seem at all clear and, on the contrary, involves new requests of principle. In order to solve the situation, [Goldstein and Gelb \(1919\)](#) are forced to admit the new kinematic quality factor, as already mentioned. The importance of movements for the perception of sensory space seems to them so decisive that they apply it also to visual recognition, although in this case it has to be done with tactile movements,

i.e., by means of the tactile space which the subject in question lacks. All this entails many complications and new hypotheses at every step, to leave the theoretical construction finally unresolved.

In relation to the above, we shall briefly examine the matter of automatic localization. This is well known in animals (frog, dog, etc.) brainless or spinal. In the spinal frog, the stimulus of an acid placed on one side of the lumbar region provokes defensive contractions of the hind leg on that side, as if to get rid of the excitant. If the reflex is prevented by holding the referred leg, the defense reflex is produced in the hind leg on the opposite side. Similar behavior occurs in the spinal dog with respect to the scratching reflex elicited by a tactile stimulus on the animal's flanks (Sherrington 1906). Likewise, defense reflexes similar to the mentioned scratching reflex can be observed in humans with central affectations (Guillan 1905). Such automatisms in humans and animals are localized reflexes in conformity with the nervous connections in the CNS. They are therefore entirely native mechanisms, and thus independent of previous experience. In some brain-injured patient with acute disorders in tactile localization, we have been able to observe similar synergies when stimulating a severely hypoesthetic area, or in any case devoid of the sense of location; but without having made a sufficiently complete study, it is difficult to draw conclusions of any importance on this issue. In such cases a number of factors may come into play (allochiria, automatosis, diversity of response according to the intensity of the stimulus, etc.) which cannot be assessed in a superficial examination. The best studied example is the spinal automatisms, either in spinal man or in physiological preparations of animals. Such automatisms constitute segmental reflex activities that depend on pre-existing structures in the nervous system. They lack any sensory effect, and it does not seem possible to establish a transit towards specific or normal tactile localization. Already in case M we have had occasion to observe that cutaneous stimuli that do not go beyond phase I of primitive projection (without localization in the body), may trigger reflex responses such as eye closure (see Sec. 21.2.1).

In our opinion, automatic (reflex) localization and normal specific tactile localization are clearly completely different activities that have no relationship or transition of any kind between them. Even rejecting any relationship between the two types of localization, it could be assumed that, if reflex responses are localized in a primary due to the corresponding neural structures and connections, the same could be true for cutaneous localizations at a higher level of nervous organization. However, the structure of the sensory field is generated by the brain, both by the action of the nervous connections or cortical projections and by the action of the central mass which, by influencing the physiological level, determines the value of the spatial sensory dimension. Thus, in the central syndrome, spatial localization, whether tactile or otherwise, will have several stages with a sensory field reduced according to the amount of the lesion and the action of the pre-existing nervous connections. Therefore, the tactile field (body schema) will be more or less wide, and will be subjected to the spatial inversion effect due to the inverted cortical projection. The brain lesion gives rise to a spatial dedifferentiation until a point is reached where progressive loss of structure results in complete destruction of spatial localization, corresponding to phase I of spatial localization, as shown in our cases, especially subject M.

Turning now to other approaches, we must consider the theory of Stein (1928, 1930) and Stein and Weizsäcker (1926), which, in addition, is closely related to the studies on the Schneider case. These authors postulate that perceptions (sensations of spatial character) arise through the action of certain motor activities. It is clear that Stein (1928, 1930) seems to refer especially to what he calls, in a vague way, "virtual movements", and in such a case these movements correspond to the phenomena of Wertheimer (1912) and Benussi (1913, 1916) on apparent movement, which are rather an effect of the spatial structure and not its cause, as Stein thinks. If they are admitted as a support of spatial perception, they are an unnecessary complication, if not a theoretical misrepresentation, at least in our opinion. Apart from all this, we strongly disagree when he states that the Schneider case is explained according to his theory by a dissociation of movements and

sensations. He also thinks that the most interesting finding of Goldstein and Gelb (1919) lies in the observation of the auxiliary movements that the patient uses to achieve visual and tactile perceptions. He therefore thinks that there must exist a certain primary relationship between virtual and real movements, and he even admits a genetic dependence between them. The child would start with real movements, then their virtual imprints would remain. Pathologically, there would be the corresponding regression.

Note that Stein (1928, 1930) seems to admit that Schneider's auxiliary movements do indeed allow him spatial perception, which Goldstein and Gelb (1919) reject since according to them the subject, with or without movements, would lack all visual and tactile space, and furthermore, they admit no space other than visual space.

The interpretation of Stein (1928, 1930) on sensory space differs in part from the previous ones, since he does not admit visual influx over touch either, but it is not satisfactory. The supports and proofs he takes from the Schneider case, slightly changing the interpretation of the case to make it somewhat simpler, are understandably rebutted from all that has been exposed of our research. Stein has been right in assuming the physiological basis of nervous excitability for sensory organization, but his questionable motor theory of perception, referring to and relying on the Schneider case, does not work. The same must be said, as far as the spatial question is concerned, of all the authors [Schilder (1923/1935) among others] who have been influenced by the conclusions of the Schneider case, or of this case and Stein at the same time, such as Buscaino (1946).

In summary, after reviewing the ideas on spatial theory, particularly tactile, we must reject, for being inconsistent, the theories of Henri (1898), Goldstein and Gelb (1919) and Stein (1928, 1930), who in one way or another have tried to interpret the Schneider case. All these theories are genetic, empiricist or associationist. For tactile sensations, Henri, and Goldstein and Gelb admit muscular movements and vision influence; and Stein admits movements and sensations for space in general. With regard to a hypothetical visual influence, it is a well-known fact that congenitally blind people have a perfect sense of tactile space, and even deaf-blind people have it too (such as Laura Bridgman and Helen Keller), therefore any sense can develop higher structures *directly* without any help from other senses.

The solution to the problem of the structure of the tactile space and localization of stimuli lies in the phenomena described above concerning asynchrony in tactile localization. The existing abnormal sensory interval gives rise, depending on the intensity of stimulation, to a series of spatial stages, completely ignored in the long study of the Schneider case carried out by Goldstein and Gelb (1919).

We already know that the fundamental phenomena of irradiation, proximal deviation, narrowing and inversion, are manifested through the different stages (phases), and such spatial phenomena must be admitted as *primary* or innate, i.e., inherent to the activity of the nervous centers, independently of genetic or learning mechanisms. Thus, irradiation means a reduction in the specificity of nervous reaction, being an expression of loosening of the local sign in the sensory field. The proximal deviation or concentric reduction of the field indicates a reduction in the sensory dimension, and the sensory inversion is a reflection or copy of the inverted projection of the sensory pathways in the cerebral cortex. In short, if the lesion causes a reduced brain activity, this results in a

*residual function field* (irradiation, narrowing and inversion). It is not the time now to examine in detail the psychophysical basis of these phenomena; suffice it to point out that the sensory organization in the brain centers depends both on the connections (inverted projection) and on the action of the central mass (excitability, cohesion, sensory dimension, etc.). Thus, it is clear that the nature of the spatial phases is in *correspondence* with the pre-existing central nervous substrate, so we could easily connect with the psychophysiological views of the authors already mentioned at the beginning of this section, stating that the perceived space is in agreement with the spatial relations contained in the nervous system, independently of learning. The same can be said about certain very singular spatial alterations, such as monocular polyopia, polyesthesia, and also the formation of a pseudofovea in hemianopsia.

The phenomena studied reveal that the local sign (the spatial value), far from being an easily identifiable and isolable invariable entity, is subject to gradations that depend on the state of sensory organization. That is to say, a functional entity, whether of space, or of sensory qualities, or of any other kind, does not exist by itself but its *individualization is due to sensory organization*. In any case, such functional entities are not primary elements, but on the contrary, functions that derive from a degree of complexity (structural development), and that starting from what is homogeneous and continuous, evolve towards heterogeneity and discontinuity, thus generating distinctness and individuality, as shown throughout the five phases of tactile localization.

A similar situation is found in the phenomenon of irradiation of conditioned reflexes, according to [Anrep \(1923\)](#). At the beginning of the development of a tactile conditioned reflex, a response can be elicited from cutaneous sites other than the point in question. There is then a certain area of irradiation, but as the development of the reflex is consolidated, the area of irradiation narrows, being almost reduced to the first stimulated site. In this case it can be said that the reflex is practically specific for a same receptor. In view of such behavior, it is possible to conceive that a stimulus influences the whole tactile field, since an area of irradiation arises which may cover the whole field; but as the central organization of the reflex progresses, the excited site tends to become specific for the reaction. It is thus clear that localization depends solely on the state of central organization, with total exclusion of any other factor.

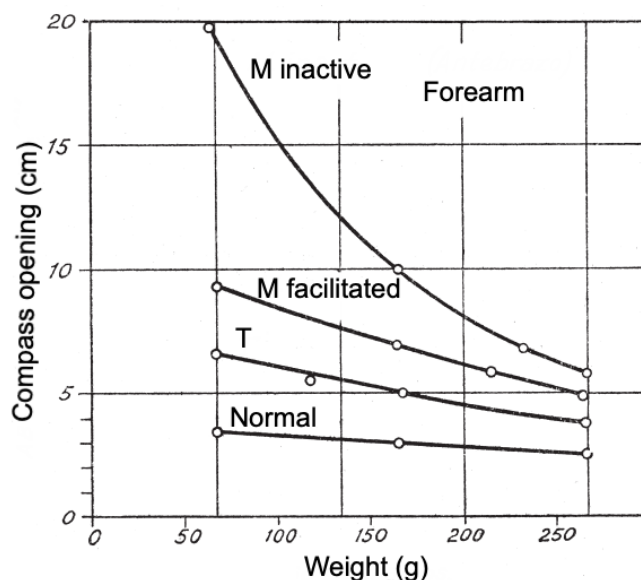
In conclusion, spatial localization (spatial organization) presents very different phases in relation to the nervous state of central organization, that is, of the sensory field. This organization is intrinsic (innate), and therefore, alien to external circumstances.

The above is sufficient to give an overview of the thesis defended here, which will be studied more extensively in the third part of this research.

## 23 Spatial discrimination

### 23.1 Spatial acuity (Weber)

The spatial discrimination of two points on the skin constitutes the tactile spatial acuity, similar to the minimum separable in vision. Such discrimination is, of course, conditioned in our subjects by the functional level of the of the phases of spatial localization (irradiation, deviation, etc.), maintaining close correlation with them (see Sec. 21.2.4 and 5.2.5). The characteristics of the stimulation are always decisive, as well as the central factor (facilitation).



**Figure 121.** Spatial threshold (Weber's compass opening) as a function of stimulation intensity (compass pressure) on the middle part of the dorsal forearm, for M inactive, M under facilitation by strong muscular effort, subject T and normal subject. Note that the threshold hardly varies in the normal subject, whereas the effect is very pronounced in the other cases, mainly in the one with the highest deficit, i.e. subject M in the inactive state. Even under high pressure, the pathological cases show a threshold much higher than normal. Compare with Fig. 47 concerning vision.

In phase I there is absolutely no sensation of duplicity whatever the spatial distance between two stimuli. In phase II, duplicity may be possible, but in such special circumstances that it is not comparable to its usual character; in addition to the great instability it presents, it is necessary to stimulate regions of different medial representation (see Sec. 21.2.2). A more normal-like discrimination, but with a greatly

increased threshold, corresponds to phase III, but the duplicity of stimuli is perceived very centripetally deviated and contralaterally inverted. For example, a stimulus on one hand and another on the elbow of the same side, 18-20 cm apart, are perceived as two contacts almost together towards the shoulder of the opposite side. It can be said that spatial discrimination appears towards phase III, in which, as we know, there is a certain rudiment of organization in the corresponding sensation (irradiation band with a weak central point).

Figure 121 shows the relationship between spatial threshold and stimulation intensity for subject M in his two main states, for subject T and for the normal subject. For the two extreme curves in the figure (M inactive and normal subject) the different behavior is clearly shown, i.e., when increasing the pressure of the two points of the Weber compass (Weber 1834/1846), there is a marked reduction of the threshold in M, and very little variation in the normal subject (see Fig. 47 concerning vision).

The compass pressure is then decisive in the pathological cases and especially in subject M, the most affected. Even using pressures that produce unbearable pain, the reduction of the threshold in pathological cases presents a limit much higher than normal values. The thresholds, deviations, phases and localization area reached by simultaneously applying the two tips of the compass are shown for subject M in Tables 28 and 29.

**Table 28.** Subject M in the inactive state, stimulated in the middle of the forearm. Simultaneous Weber threshold (in cm) as a function of intensity (in grams).

<i>Stimulus Intensity</i>	<i>Spatial threshold</i>	<i>Phases,</i>	<i>Area of localization</i>
65 g	18 cm	III,	Contralateral duplicity toward the shoulder
115 g	14.5 cm	IV,	Above the homolateral elbow
165 g	11 cm	IV,	Toward the homolateral elbow
215 g	8 cm	IV,	Toward the homolateral elbow
265 g	7 cm	IV,	Below elbow, upper third forearm

**Table 29.** Subject M under facilitation by strong muscular effort, stimulated in the middle of the forearm. Simultaneous Weber threshold (in cm) as a function of intensity (in grams).

<i>Stimulus Intensity</i>	<i>Spatial threshold</i>	<i>Phase,</i>	<i>Area of localization</i>
65 g.	8-9 cm.	IV,	Upper third forearm
165 g.	6.5 cm.	IV,	More distally
265 g.	5 cm.	IV,	Between the prongs of the Weber compass

As shown in Table 28, in M inactive, spatial duplicity with the Weber test begins in phase III (inverted), since with less pressure only phase II (medial deviation) is obtained, which only gives a single sensation. It is also observed that each spatial phase corresponds to a very different spatial threshold (minimum separation between the points of the

compass to perceive duplicity). In phase III, with a separation of 18-20 cm between the points of the compass, the perception is two cutaneous sensations almost together; however, at the end of phase IV in M inactive, a separation of 7 cm is sufficient for this same result. This shows the different *tactile spatial shrinkage* according to the functional level reached. Thus, the small subjective separation in phase III must be greatly increased if the corresponding pressure is applied to reach the end of phase IV, resulting in a widening of the tactile space. The same occurs when, at a threshold, in the inactive state, facilitation is applied; the points are no longer perceived almost together but widely separated, especially if the stimulus is weak (see Fig. 121).

In the *simultaneous Weber* test, the spatial threshold values in different skin areas of subject M are given in Table 30.

**Table 30.** Spatial threshold (cm) in different areas for *simultaneous* Weber in subject M and a normal subject.

<i>Areas</i>	<i>M inactive</i>	<i>M under facilitation</i>	<i>Normal (after Weber)</i>
Forearm	7-8 cm	5 cm	4 cm
Hand	6 cm	3 cm	1.5 cm
Finger	3 cm	1 cm	0.2 cm
Tongue tip	1.5 cm	0.65 cm	0.1 cm

The thresholds for the M case correspond to the maximum pressure with the two points of the Weber compass. By comparing the inactive state, the state under facilitation by strong muscular effort and that of the normal subject, it is observed that facilitation reduces the threshold to approximately half that of the inactive state. Both inactive and facilitated states present a threshold well above the normal one, this difference being much more marked for the cutaneous areas with higher spatial sensitivity. For example, in a fingertip, the threshold in the inactive state is about ten times that of normal despite the considerable pressure of the compass, whereas in the normal subject, a gentle contact is sufficient. As with other functions, by means of stimulus intensification, or facilitation, the less fine sensory levels (case of the forearm) can be recruited almost completely, but the more differentiated ones are impossible to reach even by combining facilitation with very intense stimulation.

In all these tests, it is necessary to pay attention, in order to be more precise, to the temporal development of the sensation of duplicity, which is quite rapid under facilitation, but slow, up to four seconds, in the inactive state. There is an evolution through the various phases among which it is easy to distinguish simple contact, single point localization and finally duplicity. In general, in all pathological cases, the perception of duplicity does not occur immediately after applying the two points of the compass, and it is perfectly possible to perceive at least the interval between the sensation of a single point and that of two.

As for the *successive Weber* test (successive application of the compass points), the threshold in the normal subject is one third or less of the threshold for the simultaneous Weber. In pathological cases, a significant reduction in threshold is also obtained with



respect to that of the simultaneous case, although somewhat less than the reduction in the normal subject. For example, the threshold for the successive case on the back of the hand of subject M is 3.5 cm in the inactive state and 1.8 cm with facilitation. Comparing these values with the respective ones for Weber simultaneous shown in Table 30, we see that the reduction is almost 50%. Therefore, even within the notable reduction of functional capacity, and using very intense stimuli, the diversity of thresholds according to the types of tests tends to be maintained. But it is important to note that, given the slow reaction time of subject M, a considerable time interval is necessary between successive applications of the two points of the compass. Thus, whereas in the normal subject the time interval between the successive application of the two points must be between 1/10 and 1/5 s to be perceived, in M, even with very intense stimuli, the interval must be at least 0.5 to 1 s, otherwise he will perceive fusion of stimuli (remember the fusion in vibratory sensation). A consequence of all this is that as the temporal interval increases, the successive spatial threshold is clearly reduced. Thus, with an interval of about 2 s, the threshold on the back of the hand in the inactive state can drop to 2.2 cm, which is smaller than the value indicated above (3.5 cm) corresponding to an interval of 1 s or less. Likewise, under facilitation and an interval of 1 s, the successive spatial threshold is 1 cm on the back of the hand.

Clinically, a dissociation between simultaneous Weber and successive Weber has often been suggested, preserving only the latter, as if they were independent functions. However, according to the criterion of dynamic reduction, it is understandable that simultaneous Weber having a higher threshold than successive Weber, is more affected, being difficult to achieve it in extreme cases. Instead, successive Weber, with a lower threshold, would still be possible, especially if the time interval in the application of the compass points is relatively long. If the conditions of stimulation in both types of spatial testing are carefully studied, the alleged dissociation or independence cannot be accepted, just as for any other type of functional activity. Apparent dissociations due to the effect of functional diversity exist, but an effective and absolute dissociation is only an observation or judgment error.

Other types of phenomena are of interest because of their dynamic characteristics of the spatial threshold. Thus, when the second point of the Weber compass is applied while keeping the first point in its place, the subject perceives a virtual motion of the first point towards the second. This motion effect can be considered an *induction* of the second stimulus on the first one, and the intensity of the effect is related to the distance and the intensity of the second stimulus. This induction seems to exist in a normal subject according to Frey (1910, 1913, 1916/1917, 1928), but in pathological states, such action is much more clearly manifested because it is increased. In subject M, it can easily be shown that the induction is much more pronounced in the inactive state than in the state under facilitation, and seems to be related to the instability of the sensory field.

In the inactive state, for two stimuli producing duplicity with a distance of 7 cm between them on the back of the hand or on the lower part of the forearm, the above-mentioned induction of the second stimulus on the first (which decreases with increasing distance) seems to disappear when the separation between stimuli is about 40 cm. With facilitation, it is much less, about 16-20 cm. The maximum induction effect in the inactive

state corresponds to a distance below the duplicity threshold. For example, by placing the second stimulus 6 cm from the first stimulus, this one can undergo a virtual motion of about 3 cm toward the second stimulus. This sensation is only a single one, since it does not reach the threshold of duplicity, and also seems to gain in intensity, which should be understood as a “bi” effect varying with distance. In addition, due to the strong induction, the proximal deviation of the localization disorder can be substantially corrected, the single sensation tending to be localized between the two points of the compass. According to the above, it is understandable that when facilitation is applied, the single point sensation becomes double, i.e. duplicity appears as a result of reaching a more favorable functional level.

With respect to the action of facilitation it should be added that if facilitation is applied at the spatial threshold of the simultaneous Weber for the inactive state, the small separation perceived between the two points is clearly enlarged, i.e., a spatial dilation occurs.

A similar modification occurs when, the subject being always in the inactive state and first applying simultaneously the two tips of the Weber compass, they are then applied successively while maintaining the same aperture. In the latter case a greater separation is perceived. Here we could attribute the change to the fact that in the simultaneous modality there is a mutual attraction or induction of the stimuli, whereas in the successive modality, such induction is non-existent or much weaker, due to the split in the application of the two stimuli.

According to all the above, the disorder in tactile acuity (spatial threshold) must be explained mainly by the spatial narrowing or reduction, and perhaps partially by an increase in the mutual spatial induction of stimuli due to the spatial instability of the sensory field. All this leads to a *reduction of spatial acuity*, i.e., to an increase in the spatial threshold, according to the functional level of the nervous centers which depends on the magnitude of the central lesion, facilitation, degree of recruitment by the stimulus, etc. The correlation with tactile localization is clear, and it has already been seen how Weber's threshold evolves in parallel with the localization phases, i.e. according to the degree of sensory organization. Thus, the increase in threshold is an immediate result of the *reduction of the body schema* (see Fig. 110).

## 23.2 Motion on the skin surface

The motion perception of a mechanical stimulus sliding over the skin is closely related to the state of spatial discrimination (tactile acuity). In addition to the spatial alteration, time disorder has to be considered. Both perfectly explain the different phenomena of motion disorder in touch, entirely similar to those occurring in visual motion perception.

In subject M inactive, motion on the skin begins to be perceived, as in Weber's test, in spatial phase III, although in a very rudimentary form and disturbed to the maximum, i.e., contralateral localization with strong proximal deviation, inverted direction of motion, maximum speed and considerable shortening of the trajectory. As for all types of functions, the sensation depends on the intensity of stimulation and other characteristics such as asynchrony, facilitation, etc.

In a mobile stimulus (e.g. point sliding on the skin), the pressure on the skin, trajectory and speed, i.e. the intensity and duration of the stimulus, must be considered. Very fast motions need a long trajectory or a high intensity to awaken the sensation of displacement, as is understandable due to the characteristics of the subject (M). If a mobile stimulus on the skin does not produce a certain level of excitation, only the static phase is obtained, as in vision, either phase I or II. As said, the first rudiment of motion perception corresponds to phase III, although still very far from normal sensation. As the stimulation intensity increases, the motion sensation tends to resemble, in phase IV, that of the normal subject (the trajectory increases, the speed decreases, contralateral localization and reversal direction disappear although some tilt remains), being impossible in the inactive state to reach complete normality no matter how much the stimulation is increased. By letting a large drop of cold water fall along the arm of subject M inactive, he can perceive during phase III conditions (when hardly any thermal sensation is perceived) a certain motion sensation on the skin. But if the drop is either very cold or very hot, or the stimulation conditions are such (long trajectory) that a first drop already awakens a clear thermal sensation, the perception of motion has the characteristics of phase IV, the more intense the temperature sensation, the more accentuated motion perception. It can be seen that in spatial phase III a series of functional differentiations appear, albeit weakly, such as lateral localization -although contralateral to the stimulus-, two-point discrimination, tactile quality and motion, thus establishing a certain functional correlation between these diverse activities (see Sec. 21.2).

Under facilitation, phase III is not obtained and there is a sudden transition from static phases I and II to motion phase IV. Moreover, it is sufficient to stimulate with a piece of hard paper, whereas in the inactive state a pin is needed for the same purpose. Thus, there is a major change, and if there was already some perception of motion in the inactive state, the perception approaches normality.

Examining in more detail the characteristics of the altered motion perception, and leaving aside the changes in localization and direction, which will be studied in Sec. 24, we shall now consider only the *seeming acceleration* and the *shortening of the trajectory*. Both factors appear in inverse ratio to the degree of sensory development. Thus, at the onset of motion perception in phase III, the subjective speed becomes the maximum that the subject is able to feel, the true speed of the stimulus being greatly overestimated, and at the same time the perceived trajectory is the minimum in which the subject can feel translation of a moving object. The length of the trajectory in such circumstances is difficult to assess accurately, but can be estimated at one tenth of the true trajectory of the stimulus, i.e., if the true trajectory is about 10 cm, it is perceived as little more than 1 cm. For a given trajectory and speed of the moving object, as the pressure of the object on the skin increases, the perceived trajectory increases and the perceived speed decreases, although both are always out of normal by defect and excess respectively. The study of this type of alteration is easily done by the subject's description of his perception, since in extreme cases of alteration the perceived motion differs considerably from the real one. It can also be studied by observing the effects of facilitation in the inactive state under a constant stimulation.

The emergence of motion perception is in relation to the degree of tactile irradiation. In the static phase, only a uniform and widespread sensation is obtained (spatial phase II). In phase III, there is some irradiation, but a central point of more marked intensity already emerges which determines the motion sensation within the surrounding irradiation. Finally, in phase IV, as the spatial character is better recognized, the irradiation decreases, and the motion is perceived with a longer trajectory and lower speed, getting closer to normal. There is thus a joint evolution for several factors.

To analyze the genesis of the disorder it is necessary to consider some essential factors in both trajectory length and speed. The former depends on the degree of spatial reduction revealed by the increased spatial threshold in the Weber test. The overestimation of speed results directly from the alteration of excitability over time. Chronaxie is increased, i.e., more time of excitation is required, which means that time is appreciated in less than in the normal subject, therefore, the motion seems faster, as well as any other time perception process (e.g., vibratory sensation). In other words, there is a *contraction in space and time*, the latter being the one that mainly disturbs the perception of motion on the skin, or other kind of motion perception (e.g. visual). It follows from the above that the higher the sensory chronaxie, the greater the overestimation of speed.

Finally, we should highlight the perfect similarity between motion disorder in touch and in vision, as summarized for subject M inactive in Table 31.

**Table 31.** Similarity between motion perception disorder in touch and in vision for subject M in inactive

<i>Phase</i>	<i>Spatial irradiation</i>	<i>Quality</i>	<i>Motion</i>		
			<i>Speed</i>	<i>Trajectory</i>	<i>Direction</i>
I & II	Homogeneous irradiation ----- Achromatic fog				
III	Irradiation with central point ----- Spatial color	Initiated	Maximum	Minimum	Inverted
IV	Point, weak irradiation ----- Flat color	More defined ----- Green chromat.	Lower	Longer	Tilted
V Normal	Point localization ----- Surface color	Normal ----- White is possible	Normal	Normal	Normal

### 23.3 Shapes on the skin surface

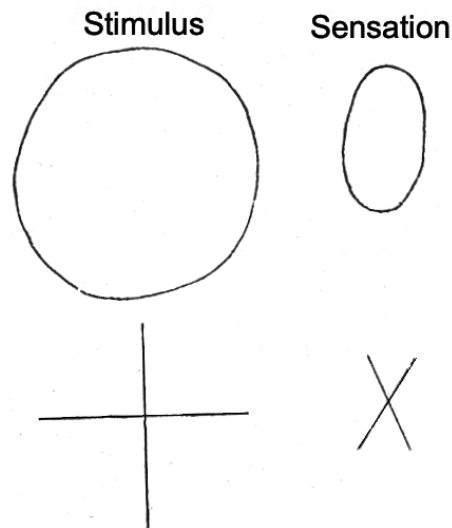
The perception of shapes and figures either applied or drawn on the skin is related to all that has been said about tactile acuity (Weber) and motion. *Static shapes*, i.e., those applied to the skin in one single action, have little relevance even in the normal subject. They are very difficult to be perceived by subject M in the inactive state even when resorting to high-pressure stimuli. It is understandable considering his remarkable alteration in the simultaneous Weber. The blade of a knife applied by its cutting edge or by its blunt edge needs to be pressed rather hard on the back of the hand to elicit the sensation of a static line. When it is perceived, it always seems to be shorter than the stimulus, apart from other alterations of position and orientation which will be studied further on. The perceived length is related to the intensity (pressure) of the stimulus, in a similar way to that described for motion perception. What can be studied by means of static shapes is very elementary, but it is of great importance for studying orientation disorder (orientation of a line).

The interest of tactile shapes lies therefore in *kinematic shapes*, i.e. shapes drawn by means of a point on the skin. Kinematic shapes are much easier to be perceived, requiring much less pressure. This difference with static shapes corresponds to that between simultaneous threshold and successive threshold in Weber's compass test. Kinematic shapes present the characteristics studied above on the perception of motion on the skin. Most of the somewhat complex shapes need a rather advanced spatial phase, so they belong to phase IV (see Sec. 21.2), where spatial contraction and speed overestimation are less pronounced than in the onset of motion sensation in phase III. The subjective speed may give the sensation that shapes are drawn quickly, but it is not the cause of an alteration of the shapes. Instead, spatial contraction leads to a reduction in the size of the lines, which is more pronounced at the beginning of phase IV.

In addition to the decrease in size, the shapes show a peculiar disorder that results in dysmorphism, originated by various causes. In phase IV there is still an alteration of the orientation that causes inclinations of different degree depending on the stimulus, giving rise to changes in the shape. Moreover, the lines are deformed due both to the particular conditions of excitability causing that the beginning of the lines can be excluded due to a slow reaction time, and to the instability of the tactile sensory field giving rise to certain dominant balances in the sensory field which imprint their peculiarity to the drawn shapes. All this will be studied below.

These different factors often act together in the transformation of the figure perceived, but it is not difficult to analyze them separately by appropriate tests. One of the most remarkable effects consists of a deformation that could be called *longitudinal dysmorphism*, caused by an abnormal spatial balance in the sensory field. When drawing, with a toothpick, the largest possible circle on the back of either of the two hands of subject M, in a continuous way, rather slowly and with enough pressure, he perceives an oval of half the size of the circle, oriented in the direction of the axis of the limb. If instead of the circle, a cross 4-5 cm long is drawn, the sensation is of an X-like shape shortened on the sides, more or less oriented also in the direction of the axis of the limb. To better know the sensation perceived by the subject, he afterwards draws the perceived figure, as

shown in Fig. 122. A verification test consists of drawing on the back of the hand an oval and an X-shaped cross, transversal to the axis of the limb; they are perceived as a circle and a cross respectively, although smaller than the original model. The figures are perceived with a proximal deviation that, given the strength of the stimulus, is of little importance. The deformation practically disappears by the action of facilitation, at least using a stimulus of energy similar to that of the previous test in the inactive state. Under facilitation there is also no size reduction.

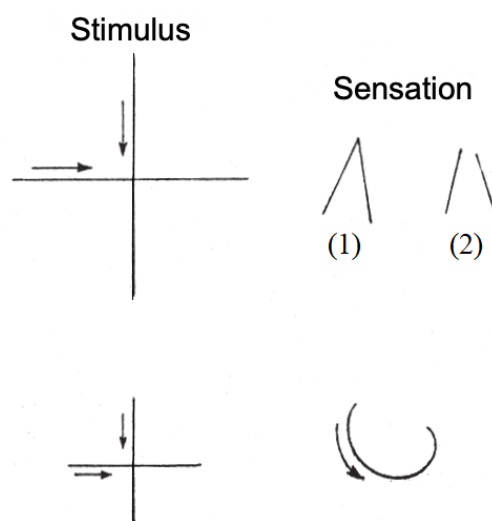


**Figure 122.** Longitudinal dysmorphism in subject M in the inactive state. On the left, figures of about 4-5 cm drawn on the back of the hand. On the right, perceived figures showing deformation in the longitudinal direction of the limb, as well as reduction in size.

As for the interpretation of this type of dysmorphism, there may be some relation with the longitudinal irradiation (irradiation band) in the localization disorder, and most probably with an increased effect of the longitudinal spatial predominance already observed in the normal subject with the circles of Weber. It would thus result, as indicated above, in a heterogeneous spatial balance, i.e. an anisotropy of spatial values in the sensory field. This same type of deformation is also present in vision (dismorphopsia), and we have been able to observe it clearly and with precision in many wounded with anomalies in the visual field, before the study of the two present cases who also present this deformation under special conditions of experimentation. This visual disorder has been deliberately omitted in this work because it is not essential for our present purpose, leaving it for a special publication on various very singular optical phenomena (polyopia, pseudofovea, etc.). We should point out that this visual dysmorphism was first described by [Gelb and Goldstein \(1923\)](#) in a series of brain-injured patients with visual field alteration.

Other types of deformations can be produced by the action of factors of different nature, either in conjunction with the effect described above or independently. A couple of examples are shown in Figure 123. A large cross may become an acute angle if the drawing is faster than in the previous test, then the initial line strokes do not have time to

produce sensation, the longitudinal deformation subsisting for the remainder of the lines. The result is then a longitudinally oriented acute angle, and if the residual is even smaller, only two more or less parallel lines remain. A more considerable transformation occurs when the figures drawn are small (2 cm). In such a case, a cross usually becomes a more or less closed curve. Here it is necessary to appeal to various effects to explain it. Short line strokes mean less stimulation, preventing a differentiated perception. In addition, due to the orientation disorder, the line stroke is perceived as tilted. Above all, as this process corresponds to a low functional level and to a great instability of the sensory field, the different line strokes tend to be joined and curved by the action of pregnancy of the form (in gestaltist terms).



**Figure 123.** Different dysmorphisms on the back of the hand of subject M in the inactive state. At the top, a cross is perceived in a first test (1) as an acute angle by elimination of the initial line strokes (excitability deficit), and lateral narrowing due to longitudinal dysmorphism. Repeating the test, it is perceived as two more or less parallel lines (2). At the bottom, a smaller cross is perceived as a more or less closed curve due to a complex effect involving excitability disorder, orientation disorder, form's pregnancy (in gestaltist terms), etc.

Similar deformations were described by [Stein and Weizsäcker \(1927, 1928\)](#) in central tactile lesions. A simple drawing that is repeated at the same skin site elicits different perceived shapes each time because of sensory fatigue and lability, until a point at which the drawing stimulus no longer elicits any sensation due to a continuous increase of the excitation threshold. It should be emphasized that the dysmorphism in all the cases indicated obeys perfectly regular and easily explainable laws; therefore, we are not dealing with errors of interpretation of the forms but rather with a certain modification of the forms, as already studied in visual perception of forms (see Sec. 11.3 and 15.1 in Vol. 1).

As for the action of facilitation, it suffices to add that in small figures it eliminates deformation and also increases their size.

Tests of greater complexity are performed with letters and numbers, showing the instability of the figures even under optimal stimulation conditions. The lines should be

made with great pressure and a certain slowness, the figures should be written on the skin (back of the hand) in a correct and regular way, and the size should be as large as possible (5 to 6 cm). Thus subject M in the inactive state can recognize a `0`, an `8` or an `A`, even though the perceived size is half or even less than the true size. However, recognition alterations are very frequent, generally due to shape simplification and similar effects. Thus, many numbers are taken for a `1`, a `2` is perceived as an `8`, a `3` as a `2`, etc. The shapes of `6` and `9` are well perceived by him but he confuses them mutually, correcting under facilitation. For the perception of numbers, it makes no difference whether they are written normally or as a mirror image; the result is the same.

By contrast, deformations or changes in meaning are markedly reduced under facilitation. Errors tend to be scarce, the size of the numbers is two-thirds or more of the true size, and drawing on the skin can be faster and with much less pressure. Also, subject M notices if the numbers are abnormally oriented. However, some remnant of abnormality persists (small decrease in size, some errors, etc.).

### 23.4 Sensitivity to joint movement

The perception of joint movement shares the general characteristics of the perception of motion on the surface of the skin, presenting in addition peculiar aspects determined by the examination method. In the case of motion on the skin, when a moderate pressure stimulus does not elicit motion sensation in a short path, the path can be prolonged until the effect is obtained. However, in joint movement, the amplitude for stimulation of movement is limited by joint excursion. If the examination is restricted to the simple test of passively moving an articular segment, it could be erroneously concluded that the perception of articular movement is completely abolished in somewhat intense alterations. However, in this function, as in all other functions, the impairment occurs in a gradual manner and the aim is to determine the *quantitative value of the functional reduction*.

In what follows we shall deal with the perception of the so-called *passive joint movement*. The active movement, more proper to another chapter, will be dealt with briefly only as a complement. As for the two subjects studied here, it should be noted, as always, that subject T in the inactive state is approximately similar to subject M with facilitation. Subject M in the inactive state presents at first sight a considerable loss of perception of joint movement; an arm can be passively raised from the vertical to the horizontal position without him perceiving any movement. The same result is obtained in any other joint. With facilitation, the sensation of movement is lacking when the displacement is small. In general, there are many possibilities for examination or stimulation. Mild and repeated stimulation is possible as well as single forced stimulation. The subjective experience is much more complex than is commonly recognized. Suffice it to say that, as in motion on the skin, intrinsic motion disorder (shrinking and acceleration) is present together with spatial localization disorder (proximal deviation and inversion).



We shall begin with the most common joint movements in ordinary activity. In our subjects, these movements must be *repeated* to elicit the sensation of movement. The following experiment gives a fairly complete picture of the state of this function. While subject M is in the inactive state, the tip of his index *finger* is held by its nail and alternate flexion and extension movements of the finger are imparted. With two oscillations of medium speed and amplitude, he feels only a simple contact of spatial phase I type. With four or five oscillations he reaches phase II, thus continuing to feel no movement, and localizes the contact toward the chest, as expected. With ten or twelve oscillations (these numbers are approximate depending on the stimulation method), a joint movement is perceived in the contralateral elbow, but only two or three very brief movements are felt (corresponding to the multiple oscillations of the index finger). Here, phase III has been reached, in which movement with contralateral localization is initiated. If the oscillating movement of the finger is maintained, the joint movement can be localized in the homolateral wrist joint. For this to occur, it is necessary to make the finger oscillate widely and rapidly. Phase IV is then reached, and the movement is perceived a little slower and wider than in phase III. Finally, no matter how long the oscillation of the finger is maintained, a new change is not achieved. Thus, it is impossible to bring the sensation of movement to the metacarpophalangeal joint (origin of the true movement). Table 32 summarizes the perception process.

**Table 32.** Iterative joint sensitivity in subject M in the inactive state. Passive oscillatory movement of the index finger.

<i>No. oscillations</i>	<i>Joint sensation</i>	<i>Localization</i>
1	None	None
2	Static	None (phase I)
4	Static	Medial (phase II)
10	Signs of movement	Contralat. elbow (phase III)
15-20 very strong	Mov. wider and slower than before	Homolat. wrist (phase IV)
Indefinite	Idem	Idem

From all this it can be inferred that in this type of stimulation the recruitment of the spatial level is obtained by *iteration*, similar to what happens in visual perception of motion in the metronome test (see Sec. 11.2). Recruitment is incomplete and normality is not reached. The movement is intrinsically altered in both speed and amplitude, is spatially deviated and undergoes the same spatial asynchrony studied in other tactile excitations.

Given the large number of oscillations necessary to obtain a sensation of movement even in signs, and also the strong proximal deviation and contralateral inversion, it is understandable that one can easily make the mistake of assuming a complete abolition of the sensation of joint movement. Such a mistake may be favored by the subject who knows that the test consists of the movement of the finger (even if during the experience he remains with his eyes closed), since, not feeling sensation of movement in that finger,

he tends to exclude any other perception of movement, which in fact is very weak. This shows how necessary it is to pay close attention to all kinds of circumstances, both to the factors of excitation and to the sensory experience of the subject, in order to get a true picture of his functional condition.

It should also be noted that joint movement can elicit a sensation of simple contact, of phase I, fully identical to that produced by a weak mechanical pressure on the skin, or by a thermal stimulus that does not arouse a sensation of temperature, etc. Thus, stimuli of a very different nature produce the same sensory effect within certain limits, the specific response being lost. Along with the decomposition of movement into static and kinematic phases (the latter of varying speed), there is no doubt that one of the major novelties in the disturbance of the perception of joint movement lies in its *localization* anomalies, which shows an identical process to that of the perception of a stimulus on the skin whether mobile or static (see Fig. 100). Given that both localization and the sensation of movement evolve in a completely parallel way, it is easy to understand how unacceptable it is to consider dissociations between the different activities, an issue that arises throughout the different chapters of this book. It follows from all the above that the so-called dissociations have arisen from the partial and incomplete analysis of a set of phenomena; and in reality, there is only a functional reduction in the series of activities of a given sensory system.

Tests in other parts of the body show characteristics analogous to those indicated for the finger, but it is convenient to examine the particularities of each case. Regarding the *elbow* joint, different phases are obtained with increasing number of oscillations; first phases I and II, then the sensation of movement appears in the contralateral shoulder (phase III), and finally the sensation shifts to the moving elbow, with the already described subjective change of speed and amplitude. As can be seen, the change of localization from the contralateral side to the homolateral one occurs with a certain inclination. In the case of the finger, from the elbow to the wrist; and in the elbow test, from the shoulder to the elbow. In the latter case there is no final proximal deviation because the stimulation is not at an extreme distal location. In the first case of the finger, it is useless to pretend that by stimulating all the fingers (opening and closing the hand passively with force and quickly), the perception of movement descends below the wrist; but the change of side from the elbow to the wrist can be made easier than with the stimulation of a single finger, a change that is sometimes difficult to achieve.

For the case of *wrist* joint movement, in addition to the non-movement perception phases, the perception of movement moves from the opposite shoulder to the homolateral elbow, and finally to the stimulated wrist, although the latter occurs with considerable difficulty since very intense stimulation is necessary. In this case, localization at the stimulation site is achieved because the latter is not very distal, although the difficulty is greater than in the case of the elbow.

For the *shoulder*, the localization goes from the contralateral shoulder, where the perception of movement is initiated, to the stimulated shoulder, with the corresponding difference in speed and amplitude. In this test, the transit from one side to the other is at the same level because there is no other possibility. For the lower limb, the same behavior is found as described for the upper limb in all cases.

Finally, the case of the *head* is even simpler, without any transit from one side to the other. After phases I and II with no perception of movement in the medial region of the head (vertex), the movement is perceived in the neck or occiput. No new change of localization of the movement occurs here, since it is impossible, but a phase III with reversal of the direction of the joint movement can be found, as will be seen in another chapter. Also, phase III can be perfectly distinguished from the more developed (slower and wider) movement of phase IV.

*Facilitation* causes in all the above tests a change as remarkable as in any other function. Subject M in the inactive state under continuous stimulation of the finger, does not achieve localization beyond the wrist. With facilitation, however, the perception of movement is shifted to the actual site of stimulation (metacarpophalangeal joint); at the same time, a doubling or tripling of the trajectory and a significant decrease in speed are felt. A greater number of oscillations is also recognized. Therefore, there is much more agreement between the stimulus and the subjective sensation.

If the above tests are performed from the beginning with subject M under facilitation by maximal muscular effort, the significant functional improvement results in reducing the excitation threshold, suppressing intermediate phases (especially phase III) and greatly reducing abnormal residuals (proximal deviation), as well as reducing movement impairment. Thus, the following is obtained for the finger test. A slight shaking is enough to obtain a sensation of movement without the need for repetition. If the stimulation is properly regulated, it is possible to obtain asynchrony and to find phases I or II, but phase III is never possible. From phase II, localization goes directly to the homolateral wrist, but if the intensity of stimulation is increased a little, the sensation of movement arises in the finger. Thus, there is no residual proximal deviation, and it should be noted that a wide finger oscillation of about  $40^\circ$  is sufficient for the sensation of movement to arise. However, two or three oscillations of the finger are necessary if the amplitude of the oscillation is small.

When the passive movement takes place in the interphalangeal joints of the finger, an intense muscular effort enables the movement to be felt in the same location of the motor origin, but several oscillations (7-8), somewhat intense, are necessary. Therefore, when it is an extremely distal movement there is a tendency for proximal deviation, even with facilitation, although the deviation can be overcome by reiteration. In contrast, in the inactive state the localization cannot go beyond the wrist.

For the other joints of the upper limb, facilitation makes it possible that a single small oscillation is sufficient to obtain the perception of movement in the examined joint (wrist, elbow, shoulder). These are large joints and more proximal (to the center of the body), factors that facilitate excitation. In tests with facilitation by generalized muscular contraction, the joint segment whose passive movement is being explored must, of course, be left free. When the area under examination is also part of the strong facilitating muscular contraction, the threshold of sensitivity to movement decreases even more than in the previous case, but here it is not a sensitivity to purely passive movement since the provoked displacement of the limb must overcome the strong muscular contraction.

Subject T showed at the beginning of being examined (1938) the anomaly of perceiving passive joint movement as simple pressure (static phase), together with a

certain difficulty in localizing stimuli of medium intensity. It was also evident that he did not perceive slow movement even of a certain amplitude. However, these disorders disappeared through involuntary muscular twitching (spontaneous facilitation). Later, we observed that this subject behaves in everything like subject M under maximum facilitation. Thus, passive finger movements of very small amplitude are not perceived, but when the amplitude is increased a little, the sensation of movement arises, although modified since it is clearly felt to be of smaller amplitude than the real one.

So far we have seen that the sensation of joint movement is only possible by prolonged repetition of moderate oscillations, especially in subject M. However, by stimulating vigorously it is possible to achieve the perception of movement in the inactive state with a *single oscillation*. Of course, this oscillation has very different characteristics from the previous ones. For example, a single oscillation with all possible amplitude of the metacarpophalangeal joint and with great speed can result in a sensation of movement at the wrist. For this to occur, it is very important to forcefully reach the point of maximum excursion of the joint. Otherwise, the proximal deviation is stronger and the localization results in the homolateral elbow or the contralateral shoulder. It is therefore possible to regulate the test and obtain different localizations. In short, with a single oscillation the same result can be achieved as with 15-20 moderate oscillations, but the single oscillation must be very strong, especially to achieve the maximum effect that is possible in the inactive state.

The conditions under which the sensation of movement is elicited by single stimulation deserve close examination. A necessary characteristic, common to both single and repeated oscillations, is the *rapidity of the oscillation*. In this respect there is a great difference with visual perception of motion and tactile perception of motion on the skin, since in these cases an excess of speed of the mobile stimulus tends to impede the perception of its motion. By contrast, in the multiple tests performed over the years on our subjects, it has been found that a rapid joint movement is necessary to cause any effect. The fact that the sensation of movement is better when the speed of the movement is higher was pointed out by [Goldscheider \(1889\)](#), who proved that at low speeds the threshold value to perceive joint movement is somewhat increased. The *range of movement* and the *pressure on the joint end-stop* must also be considered. The importance of the former is clearly seen in the single oscillation test where it is desirable to involve the entire joint excursion; it should also be noted that in the less disturbed cases (subject T inactive and M with facilitation) there is a greater sensitivity for large joints than for small ones. As for the pressure on the joint end-stop, it seems to play a decisive role in generating the sensation of joint movement. In the theory of joint movement, there are two opposing hypotheses: the *purely articular* or articular surfaces theory ([Goldscheider 1889](#)), and the *para-articular* theory ([Frey 1918](#)) which attributes the process to the changes and tractions performed by the muscles, tendons, articular capsules and even other deep and superficial soft parts including the skin. In any case, the articular movement would be the result of an organic complex. In our cases, the fact that the pressure on the joint end-stop is important, forcing the joint capsules, ligaments, etc. to the maximum, seems to support the para-articular theory of Frey. The influence of the speed and the range of movement could also support it; high speed would cause an abrupt

change in muscle tension, and a wide range of motion would work in the same way. Thus, if the passive movement is the effect of the activity of ligaments, tendons and muscles, and not of the simple displacement of the articular surfaces, the sensation of movement would develop in a very indirect way in comparison with the case of an object moving on the skin or with the case of visual perception of motion.

The pure articular theory (displacement of the articular surfaces) seems to have a little less support in our tests. The artificial increase of the articular pressure (of one surface against the other) which, according to experiments by [Lewinski \(1879\)](#), would notably favor the sensitivity to movement, has given us irregular but rather confirmatory results. However, it is questionable whether the facilitation action is due to pressure between the articular surfaces or perhaps to an indirect summative effect due to the pressure exerted on the skin when gripping tightly the finger being tested, since this gripping may result in localization at the wrist even if there is no actual movement. Without being able to completely attribute the perception of joint movement to certain factors by excluding others, it should be noted that the important effect of pressure at the joint limit and also the effect of the speed of movement seem to directly support the para-articular theory of ligament tensions. However, the importance of the amplitude of movement can be a support for both para-articular theory and purely articular theory. In short, the process is complex and a very considerable part seems to involve para-articular organs, in accordance with the predominant orientation at present.

Finally, we should briefly indicate some characteristics about perception of *active* joint movement, i.e. voluntary movement. This shows in general the same disturbances as the passive movement, although somewhat reduced. For subject M to be able to make a voluntary movement, he needs a certain degree of facilitation to bring out the body schema, and then he can make use of his own movements (as will be seen in Sec. 11). When he is asked to move any finger of his hands, he needs a significant muscular effort to orient himself on his own body and choose the requested movement. But, if the movement of his finger has not been very vigorous, the sensation differs considerably from the real movement made. If the movement is slight, it may not be perceived as such movement (phase I) or in any case be very deviated, or contralateral, since the facilitation is not maximal. If the movement is more intense, it may be localized in the homolateral wrist, etc. Of course, the greater the deviation, the greater the speed and brevity of the perceived movement, as in passive movement. Also, under repetition of the movement, changes in the localization and amplitude of the movement are obtained by increasing the number of oscillations.

It is therefore understandable that there can be considerable incongruities between the voluntary action and the corresponding proprioceptive sensation. Different questions of great importance will be studied in depth in the section on orientation in touch (Sec. 26) and in the section on the body schema (Sec. 27), where the issue now initiated will be continued.

## 23.5 Body perception and manual touch

Body perception, and in part manual touch, are issues of a more complex order than those discussed above, and already belong to the function 'tactile schema' which will be dealt with in the last part of this book. However, as is always the case, there are transitions between the different categories. Here, both the *schema of the own body* and manual touch (*active touch*) will be briefly discussed in relation to tactile space, thus completing the study of this topic.

### 23.5.1 Reduction in the schema of the own body

Body perception derived from the set of tactile sensations, both cutaneous and deep, presents remarkable phenomena due to asynchrony. Among the most characteristic phenomena are: fragmentation of the body schema, abnormal flexibility of the body, sensation of lightness (loss of weight), reduction of size, etc.; of which a brief description will be made although many of these phenomena are difficult to be fully described.

As the coherence of the tactile spatial functions is impaired, a certain *fragmentation of the body schema* arises, that is, a kind of independence of its parts, which acquire a singular degree of lightness or flexibility, all similar to the phenomena described by [Beringer \(1923, 1927\)](#) in experimental mescaline intoxication. This author describes that the body continuity is lost, and when shaking hands, the affected person has the feeling that the hand is detached from the rest of the body, the body is felt to be smaller, as if concentrated in a homunculus, etc. When a limb is moved passively and the inversion phase is obtained, the subject feels the movement of the limb as something more or less disconnected from the body and says that it seems to be a little loose; whereas in the next phase (IV), this disconnection tends to disappear quickly. From the examination of the patient's perceptions it seems that in the first case the moving limb has no references with respect to the rest of the body, especially the trunk; but as the organization improves by increasing stimulation, the rest of the body schema tends to emerge, although it is a process difficult to express clearly. The less developed phases (II and I) give an even more blurred sensation of the body schema, which is reduced to spatial references that are always static and highly ill-defined.

Subjective *flexibility* of the body is another aspect of body schema disorder. When a limb moves, either passively or actively, it gives rise, within certain functional level limits, to the sensation of being somewhat diffuse or swollen, soft or malleable; in part, somewhat similar to the sensation a normal subject has when a limb falls "asleep" due to nerve compression. In relation to the decreased sensation of pressure, both *weight* and *strength* seem greatly diminished (see end of Sec. 18.1), the body is felt much lighter and without resistance to thrust, thus contributing to the lack of body definition. When rising from the sitting position, in an inactive state or with the minimum possible facilitation, the weight of the body is perceived as very light compared to the sensation when performing the same movement under maximum facilitation. The same occurs when walking, the movements are felt light (or without force) as well as fast and short. If the

subject actively lifts a limb under maximum facilitation (by muscular effort), the subject feels it three times heavier than if he lifts it in an inactive state (but with minimal effort to produce a voluntary movement). Under facilitation, a hand is felt as heavy as the whole leg in an almost inactive state.

The *size* of his own body is felt to be reduced due to spatial contraction (see Sec. 23.1), and in the usual state, the subject feels himself considerably smaller than under facilitation (by maximum muscular effort). In addition, and as an effect of proximal deviation, the more distal parts of the limbs are excluded, resulting in a further decrease in body size and volume. These distal parts need maximum facilitation to recover their presence (see Fig. 110).

All these anomalies are easily manifested in activities such as walking, getting up, sitting, etc. When rising from sitting, in the usual way (without maximum facilitation), the body is felt to be smaller, lighter and softer; but if then the subject starts to walk, the normal characteristics tend to become progressively established. In these subjects, walking is a very complex process from the proprioceptive sensory point of view, and will be studied in detail in the section on orientation. In the following, only some characteristics about corporeality during gait are indicated. In the first steps, the body is felt to be much smaller and lighter in weight, and the steps are felt to be much shorter and faster. At the same time, the footstep feels soft “like on a carpet”. A great change occurs when the subject is under the action of maximum facilitation since the functions are considerably normalized. As for the resistance of the floor when treading, the subject in the inactive state is unable to distinguish between treading on a floorboard and on a carpet. However, with facilitation he immediately notifies when he treads on a carpet or on a hard floor (always without seeing the floor). When he stands and rests his body on one leg, this leg seems to him longer than the other; the same effect is obtained when, standing on both legs equally, he moves one of them or contracts its muscles.

Finally, to be complete in our description, we should mention the recognition of body postures or attitudes, especially of the limbs; but this issue requires an in-depth study of the body schema in order to be sufficiently clarified, which will be done in the sections on tactile schema. Suffice it to point out now that the perception of body attitudes requires a highly developed body schema, and therefore they are usually suppressed unless a fairly intense facilitation intervenes, and even then there are noticeable reductions, also in the less impaired subject T. In addition, there is a special tendency to ignore different postures or attitudes, which are perceived as similar to the usual posture of the body and limbs (pseudoagnosia).

In short, the perception of the body is closely related to all kinds of tactile space functions, and its study will be completed when dealing with orientation, and mainly with tactile schema.

### **23.5.2 Reduction in manual (active) touch**

As a result of the various alterations of the tactile space, superficial and deep (in joints), active touch with the fingers can be quite affected even under the usual conditions of the subjects. The disturbances are, as for body perception and other sensory functions, of the

type described by [Beringer \(1923, 1927\)](#) in relation to mescaline (the author says: “hard objects feel as if they were rubbery, soft and ill-defined in shape”).

These alterations of active touch with the fingers due to the spatial disorder of touch could be called *flat touch*, by analogy with flat colors. Thus, due to the thickness of the tactile irradiation that dulls the cutaneous surface, there is a certain sensation of penetrability or flexibility of hard objects and a reduction in the appreciation of microstructures. Hard objects, such as a glass jar, do not provide to the hand's touch a strong sensation of hardness, but are perceived rather as something malleable that could be squashed with the fingers, but not like a rubber ball but like a solid rubber. Similar sensation may be elicited by a wooden table top, seeming less hard, or as if lined with a padded tablecloth. All these abnormal sensations are greatly diminished with a fairly intense facilitation (by muscular effort), but they are quite evident to the testing subject when facilitation is weak, and even if he exerts strong pressure with the fingertips when touching objects. It also happens that all touched objects give the sensation of having a smaller size. This must be caused by the contraction of tactile space (superficial and articular) already studied, in such a way that the size of an object becomes equal to that of the hand. As tactile acuity is reduced, roughness and other characteristics of the microstructure of objects are erased, giving the sensation of being smooth to the touch. In all these tests, the subject is free to touch objects with his fingers by moving the fingers as he wishes and pressing the objects at will; and although this involves some muscular effort (facilitation), the above-mentioned anomalies are clearly present. Only with facilitation by vigorous muscular effort as well as active touch with strong pressure, a noticeable change towards normal sensation occurs, regarding hardness, roughness, size, etc.

All these phenomena show us that stereognosis is very impaired, although not abolished, but reduced and altered by ‘flat touch’. It is then understandable that tactile recognition of objects is severely altered. When the subject slides his hand over a brush, he only feels a soft surface that deforms greatly, comparable to a rubber ball. He feels the same when he sinks his fingers into the brush. When he slides his hand repeatedly over the brush or his hand is rubbed with the brush, he perceives at most a slight roughness but as coming from a compact mass. When the subject is in an inactive state, the rubbing of the brush on the cheek gives him the sensation of a rough cloth, but on repeating the action, he ends up having a normal perception. Also, a somewhat thick pocket chain is perceived as a cord, i.e., more compact and softer than it really is. However, under facilitation all these anomalies disappear.

It should be noted that touch must be combined with movement since in this way the advantages of the successive Weber test are exploited, in addition to other facilities provided by joint movement. Thus, even if the tips of a fork are strongly applied to a finger, the subject feels only one tip and must move the fingertip over the tips to perceive two or three of the four tips. But this active touch (kinetic or successive) is still very diminished in the ordinary conditions of the subject since if he touches the tines of a fork lying on a table with the tips downwards, not upwards as before, he says he perceives only “two things, two thin sticks”; and this is achieved with very active movements and a strong pressure exerted with his fingers. Only with facilitation by maximum muscular



effort does he recognize several times and immediately understands that it is a fork. The test is performed, of course, with the eyes closed.

When many ordinary manual activities are examined by allowing the subject only a weak facilitating action, such activities present great difficulties. Thus, when buttoning a shirt or any other article of clothing, he finds great difficulty in locating the buttonholes, distinguishing them from the rest of the cloth and, above all, in channeling the buttons through the buttonholes, etc., all effects of the reduction of tactile space.

Impairment of tactile object recognition involves several anomalies, namely the so-called 'flat touch' anomaly that influences the loss of acuity, other intrinsic anomalies in shapes on the skin surface and in joint movements, and in a very important way the alteration in body perception (e.g. distal exclusion). The disorder of tactile object recognition is therefore the result of a global disorder of the tactile system, and will be dealt with in the part devoted to the schema.

# Perceived orientation

## 24 Dynamic disorder of orientation in touch

### 24.1 General aspects

The phenomenon of inverted vision was discovered in subject M by chance in 1938, and only one or two years later could it be rationally understood within the framework of this brain dynamics research. However, the *inversion of tactile space* was not found until much later (1945-1946). Its finding, also partly by chance, was a consequence of certain theoretical assumptions but mainly of very meticulous examinations of various tactile functions such as spatial localization and motion perception of a stimulus moving on the skin. With the discovery of the dynamic action phenomena (1939) it was shown that the nature of sensory disturbance was the same in intensity and manifestations for the three most important sensory systems, vision, touch and hearing. An exception, however, was inverted or tilted vision, unparalleled at that time in touch and hearing. From a theoretical point of view, if pathological inverted vision was to be linked to the normal inversion of the visual image on the *visual retina* and thus transferred to the “cerebral retina” (sensory projection), it is also true that the inversion of sensory projections is not a particular case of vision, since an analogous anatomical arrangement exists for touch and hearing. Precisely in the case of touch, it is elementary knowledge that the projection of each half of the body is contralateral and upside down. Therefore, it seemed that an even greater parallelism in the alterations of the mentioned sensory systems should be expected. In principle, both a tactile and an auditory inversion were expected, but it was difficult to predict what modality they should adopt.

The long-standing and persistent debate on how vision can be right (normal), knowing that the visual image is inverted on the retina, has already been pointed out in the appropriate place (Secs. 12.1 and 14.1 of Vol. 1). In contrast, no trace is found about touch, although a very similar problem can be posed if one considers the inversion of the sensory projection in the cortex. But it must be taken into account that the knowledge of the contralateral and inverted projection of the tactile space (or of the body) belongs to a time (end of the 19th century) when all these questions of the sensualist philosophy about the orientation of space and other related questions had fallen into the most absolute

oblivion among scientific concerns; and moreover, these issues had never given rise to significant manifestations in brain lesions. As already mentioned, the long dispute about the mechanism for a correct orientation of the perceived visual image has not had any impact on brain pathology either despite the existence, since very ancient times, of some clinical reports about inversion and tilt of the perceived visual image, although without objective verification or detailed study of the phenomenon, always described in a few lines.

When dealing with visual image orientation, we have mentioned some clinical precedents on pathological inverted vision, whereas it is almost impossible to find them for tactile inversion, except perhaps in the brief, vague and unexplained observations of what is called *allochiria* (tactile localization contralateral to the stimulus), which we have been able to observe in a brain-injured patient in 1948. In accordance with what will be expounded on the theory of orientation in Sec. 10, it seems certain that in many of the known cases, *allochiria* correspond to phenomena of a very different kind from that now being studied on tactile inversion. If some cases could perhaps show some relationship, it is clear that they would constitute a minimal and much less direct precedent than what we have found in inverted vision.

Within the general difficulty of investigating the different abnormal manifestations, the fact that in subject M tactile spatial inversion is more difficult to be detected than visual inversion deserves a comment. As [Katz \(1920/1925\)](#) points out, already under normal circumstances, visual sensations and functions are more objective than tactile phenomena, since the visual ones refer to the external space, outside the body and the eyes. However, in tactile phenomena there is inevitably a subjective factor in relation to both our own body and the object; this is why Katz calls tactile phenomena “bipolar”.

We can admit that such tactile circumstances make it difficult to clearly perceive pathological phenomena which fit poorly into the normal routine, and which subjectively would be suppressed or diminished. But moreover, there is a general tendency, not only in touch, to exclude any defect both by the singular and inscrutable phenomenon of anosognosia and by the summative mechanism of facilitation by muscular effort exerted unconsciously. In view of all these characteristics, it could perhaps be said that tactile inversion has been more difficult to discover due to the lesser objectivation of tactile perception. Probably, other peculiarities of tactile space are also involved, such as its greater heterogeneity compared to visual space. In short, tactile inversion is a very hidden disorder despite the multiple and important manifestations to which it gives rise. When inverted vision was found in subject M, he said spontaneously that he had on occasions before seen more or less marked inversion of the visual scene. In the case of touch, however, he was not able to say something similar, and the complex and diverse phenomena of tactile inversion, painstakingly brought to light, seemed to him as surprising and strange as the phenomenon of facilitation by muscular effort at the beginning of being examined.

*Tactile inversion* is linked to the finding of phase III in the localization of a stimulus, a phase that arises due to nervous asynchrony as an extreme consequence of the disorder of orientation in space, as with vision. However, depending on the level of recruitment, the orientation disorder appears differently pronounced, with different degrees of tilt,

belonging to phase IV. These tilts can be easily appreciated when the stimulus is a straight line whose orientation on the skin is possible to perceive. This is obviously not possible with a point stimulus. Therefore, the orientation disorder is shown through phases III and IV until phase V (normal) is reached. The less developed the spatial phase is, the greater the disorder, although the first two phases (I and II) do not count at all in the study of orientation because they have a too elementary sensory organization. It should be noted that this disorder presents a very broad course. Phase III only corresponds to the extreme alteration with practically complete inversion of the orientation and contralateral localization, whereas in phase IV (very broad and with homolateral localization of the stimulus as we know), the orientation of a straight line can show from very large tilts exceeding  $90^\circ$ , to very small or practically null tilts. Therefore, the exact localization of a line also involves its orientation, so the following must be taken into account: the side of the body, the proximal deviation and the orientation of the line. Moreover, as the line is perceived in different sizes depending on the phases, the localization process is very complex, evolving in its entirety as is always the case in the dynamic reduction.

All the processes of orientation in touch were studied only in subject M since subject T was not available to be examined after 1944, and the disorder of tactile inversion was glimpsed toward the end of 1945 and extensively investigated in 1946. However, given that two quantitatively very different sensory levels can be studied in subject M, inactive and under facilitation by maximal muscular effort, and given that subject T is very similar to M under maximal facilitation, it can be presupposed the degree of the disorder in tactile orientation that subject T should suffer from. Subject M presents a tactile inversion of the same intensity as that of the visual inversion, in accordance with the characteristics of the central syndrome. This allows us to conjecture the degree of the tactile orientation disorder in subject T since it must also be equal to the degree of the visual orientation disorder that we already know well. Hoping to be more complete on a more favorable occasion, we now stick to the experimental data of subject M. Because of the two extreme states of excitation of subject M, it is possible to make very broad generalizations to other cases with less brain impairment. We know that subject M in the inactive state presents almost complete inversion in vision and touch, whereas under maximal facilitation he only presents tilts of moderate intensity, phase III (in touch) resulting then excluded. These moderate tilts, such as that of subject T in vision, are manifestations of incomplete or frustrated inversions because the asynchrony is not sufficiently intense, and are therefore included in the same type of disorder. Thus, the Schneider patient of [Goldstein and Gelb \(1918, 1919\)](#), of whom nothing is mentioned about visual or tactile inversion or tilt, should present a tilt of 80 to 90 degrees in both sensory systems. It should be noted that since the sensory disorder is always of an overall type, as already exposed in this research, the alteration in tactile orientation should be a quite frequent phenomenon (at least in the moderate form of small inclinations) even in cases of a not very pronounced brain tactile disorder, in the same way as we stated about visual image orientation.

The *general laws* of the inversion or tilt of the perceived orientation in touch are the same as in vision, but the study in touch is much more difficult. This is due to the fact that in addition to the aforementioned difficulty in being objective, the heterogeneity of the tactile field both in texture and form, highly irregular in comparison with the visual

field, greatly hinders accurate determinations. The position of a straight line in space can be perceived visually with remarkable accuracy; but the orientation of a straight line on the skin surface can only be recognized approximately even by a normal subject. It is also not easy in touch to perform a wide variety of tests and quantitative determinations such as those related to the study of visual image orientation, for example, those related to the “bi” effect (Sec. 13.3 of Vol.1), the sensory state of the receptor (Sec. 13.4 of Vol. 1), sensory adaptation, etc. In touch, the tests have to be restricted to the recruitment of orientation by intensifying the stimulus for the two sensory states, inactive and under facilitation. However, this experimental restriction is compensated by the complexity of tactile space, namely, spatiality on surface, spatiality in deep sensitivity (joints), and processes in the orientation of the body schema, all this offering a very wide field for the investigation of tactile orientation, whose phenomena can be considered among the most novel ones in this research.

Regarding the theoretical development of this research on brain dynamics, we should note the important progress made by finding in the central syndrome the spatial inversion for all the sensory systems that allow localization and orientation in space (vision, touch and hearing). In fact, the discovery of tactile inversion made us adequately search for the possible auditory inversion (localization in the contralateral ear) and confirm it. Thus, it is no longer possible to consider reversed vision as an isolated process without parallel in other sensory systems. Within the perfect concordance between the pathological manifestations of the various sensory systems, the privilege that vision seemed to have in altering orientation seemed strange indeed. Therefore, the generalization of spatial inversion to the remaining senses was enormously gratifying, especially because of finding such a remarkable regularity in sensory organization. The material basis of the stimulus for each sense is specific for each of them (light, touch, sound), but the functional architecture is identical for all of them. In the central syndrome studied here, the reduction of functions is similar for all of them, showing a perfect parallelism between the different sensory systems involved.

Finally, we must emphasize the great importance of the finding of tactile inversion and the generalization of the phenomenon of spatial inversion for the development of this research on brain dynamics. It means not only complementing the study of the disorders following what has already been established in vision, bringing a greater homogeneity to the central syndrome, but mainly it establishes a new theoretical stage of great interest for this research. One of the reasons for the interest is that the investigation of tactile inversion has led to a deeper understanding of the phenomenon of inversion (also in vision) according to a *spiral development*. Another reason for the interest, perhaps more important, is that the topic of spatial inversion acquires a great scope by being linked to general issues of remarkable theoretical importance, such as that of the anatomical crossings of the long pathways, and the *psychophysical correspondence* involved, as will be seen in the theory of tactile inversion and mainly in the third part of this research. As a result, this research does not change its characteristics but undergoes an important extension of its dynamic postulates.

## 24.2 Perceived orientation in cutaneous stimulation

In the case of cutaneous stimulation, it would have to be a pure chance that the orientation disorder might be perceived by the subject spontaneously, outside experimental determinations. This is because for phase III (inverted) to appear, a stimulus of very mild and adequate intensity is necessary. If the intensity is slightly higher or lower than the adequate one, this phase does not appear. This refers mainly to point stimuli, since rectilinear stimuli, whether static or kinetic, very easily present very diverse tilts (rotations), at least within phase IV, which is a very broad phase. However, these rotations cannot be appreciated as directly as in the case of vision, requiring a certain amount of attention which is not usually paid to tactile perceptions, apart from the fact that isolated straight lines are not usual tactile stimuli in ordinary life. Thus, the disorder remains largely hidden and is only revealed by very thorough and varied special tests.

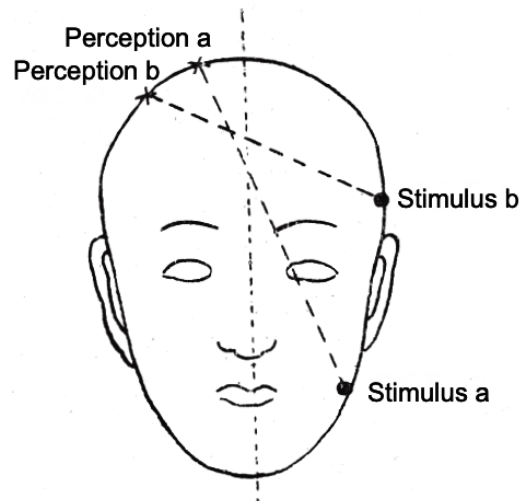
The general empirical conditions are the same as for vision, i.e., the sharper the tactile perception, the less the orientation disorder; and vice versa, as the perception becomes diffuse (irradiation, proximal deviation, etc.) there is a greater tendency for the inversion of the perceived spatial orientation of a tactile stimulus over the body. We shall now study, in increasing order of complexity, the inverted perception of a point stimulus, of a rectilinear stimulus and of motion on the skin.

### 24.2.1 Inverted perception of a point stimulus

Given the close relationship between phenomena, the study of inversion and localization in point stimulation over the skin is entirely the same as that presented on localization phases III and IV. Therefore, we shall now only complete it with some particularities, especially regarding the head.

As we know, the *head* behaves in localization tests as an independent element (see Sec. 21.2.2), and tends to deviate the stimuli towards the vertex or cranial vault (proximal sector) in medial phase II, the face acting as a distal sector. Let us recall that the sagittal midline belongs to phase II, a small parasagittal area on the cranial vault belongs to phase III, and finally the rest of the head and face belong to phase IV. In phase III, the stimuli are localized contralaterally, but always within a narrow area of the cranial vault. The characteristics of the contralateral localization can be seen in Fig. 124. A stimulus on the lower cheek is perceived contralaterally on the cranial vault and very close to the midline; but if the stimulus is on the temple, the localization is also contralateral on the cranial vault and less close to the midline than in the previous case, i.e., a little below, the distance between the two inverted points being less than the distance between the referred stimuli (see Fig. 124). All this refers to the vertical direction, and as for the horizontal, the disposition is analogous: very anterior stimuli in the head are localized contralaterally backwards in the cranial vault, and vice versa. Thus, the tendency is towards contralateral localization and with inversion of all positions, although due to the strong proximal deviation towards the cranial vault, stimuli at the upper part are not localized at the opposite bottom part. In general, it can be stated that the point of inverted localization

corresponds to the opposite end of the straight line that starts from the stimulus site and passes through the center of the head, considering in addition the strong deviation towards the vertex. Thus, there is no possibility for the inverted perception to be localized on the face, since the *inversion area* is much smaller than that of the stimulus application, and is restricted to the area most proximal to the midline of the head.

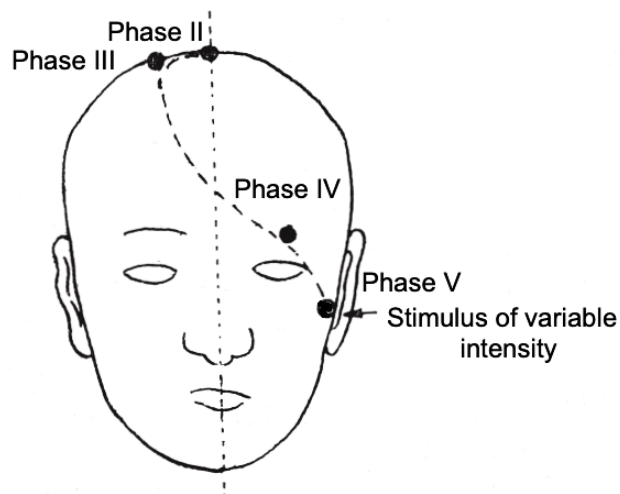


**Figure 124.** Schematic drawing of the inverted perception of stimuli in the head of subject M in the inactive state. In phase III there is a contralateral localization and always on the cranial vault, showing a certain inverted symmetry: stimuli on the lower part are localized on the upper part and vice versa. The inverted symmetry is rather distorted by the strong proximal deviation towards the vertex.

As for the transition from inverted to homolateral localization with increasing stimulus intensity, it suffices to refer to what has already been said about stimulus localization. The tests referred to in Fig. 120 show the asynchrony between the different phases of deviated localization and these tests are also valid to show the stages in orientation. In the case of a point stimulus, it is only possible to study the position of the felt point, whereas if the stimulus is a straight line it is also possible to study its orientation. Let us now analyze in more detail the trajectory that links the various phases of stimulus localization as stimulus intensity varies. When the stimulus intensity decreases, localization tends towards the medial zone (centripetal trajectory) and if it increases, the opposite occurs (centrifugal). For example, if we consider a stimulus in the center of the left cheek (see Fig. 125), as the intensity of the stimulus increases, we obtain after phase III (contralateral localization on the cranial vault about two fingers from the midline) a forward translation on the upper part of the forehead; from here it crosses the midline toward the homolateral temple (or external angle of the left eye), and finally, with painful stimulation, the localization tends to move down the cheek approaching the stimulation site.

It should be noted that it is very difficult for the tested subject to describe verbally the exact trajectory followed by the localizations deviated from the stimulus site. This is because these are lower stages of poorly differentiated sensations that change rapidly, and also because of the difficult spatial discrimination in the cranial vault even in normal subjects. Therefore, we have to stick to a few data that give us a rough indication of the

general path: upper contralateral zone, somewhat lower mid-anterior, mid-height homolateral, and finally homolateral in lower zone. All this, including phase II of medial localization, results in a *spiral development* of the sensation from the vertex to the stimulated cheek because of the combination of inversion and proximal deviation effects, with the spiral becoming more and more open. This spiral development can be considered valid within the imprecision of perception in this test since a similar trajectory is shown in other tests, thus constituting a general characteristic of spatial inversion. Moreover, this is valid for both touch and vision.



**Figure 125.** Trajectory followed by the perceived localizations when varying the intensity of a stationary point stimulus in subject M in the inactive state. Due to the contralateral translation and proximal deviation towards the cranial vault, the trajectory tends to be a spiral.

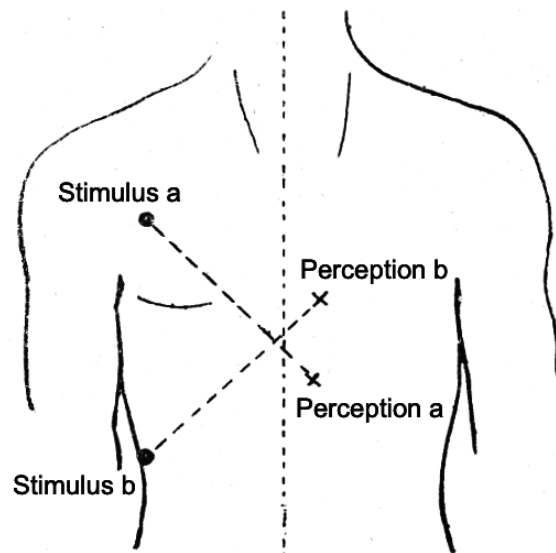
Under maximal facilitation, we already know that the complete inverted phase (contralateral localization) is not possible, and localization abruptly shifts from phase II to phase IV. Phase IV includes orientation changes that can only be demonstrated by a rectilinear stimulus.

As for the case of the *limbs*, there is nothing special to add to what was stated in Sec. 21.2.3 on the inverted phase of spatial localization, the process of inversion and deviation being illustrated in Fig. 100. In this figure it can be seen that, given the morphological circumstances, although it is not possible to clearly uncover a spiral-shaped trajectory, the two effects that determine it are still valid: contralateral inversion and proximal (centripetal) deviation. As will be seen, the study of orientation can be easily performed by means of a rectilinear stimulus on a limb.

Finally, the inverted perception of stimuli on the *trunk* is analogous to that on the head, although much simpler. Only the most anterior plane of the thorax corresponds to the inverted localization, since the lateral and the back already belong to phase IV, i.e., to the distal zone. The contralateral localizations are inverted with respect to the vertical, i.e. stimuli on the upper part are perceived on the lower part, and vice versa. Due to the special shrinkage of the inversion region, the distance between the perceived localizations is much smaller than the real distance between the stimulation points, as shown in Fig. 126. In this figure, the proximal deviation in phase III can also be seen, as in Fig. 101.



In short, as we already know from the study of tactile localization, we find *three independent zones for inverted perception*. One is the head, another is the trunk (thorax) and upper extremities, and the other is the abdomen and lower extremities. Thus, the body is segmented into these three zones in relation to tactile inversion. This is a notable difference with the visual field where the inversion occurs for the whole field (each retinal half). Therefore, a stimulus on the head cannot be perceived inverted on a lower limb or on the contralateral side of the trunk, but only on the head towards its proximal midline region, as we have seen.



**Figure 126.** Inverted perception of point stimuli on the trunk of subject M in the inactive state. During the inversion phase, contralateral localization is obtained with opposite localization (up-down) in the vertical axis. Inversion symmetry is reduced by the effect of proximal deviation and constriction.

In relation to the meticulous tests on the inversion of point tactile stimuli is the finding of *polyesthesia* (multiple localization of a single stimulus), analogous to monocular polyopia. As in the latter, polyesthesia requires a particular study and now only the most elementary features will be mentioned. In the inactive subject M, polyesthesia has only been demonstrated on the midline of the body including the cranial vault and trunk, especially on the chest. When a point stimulus is applied on the midline of the body, we know that the phase III sensation is in the form of a band of irradiation with a central point located on the midline, i.e. at the same place of application of the stimulus, without deviation. In this situation, another point usually appears, much weaker in intensity and always on the right side, about two fingers away from the first point, just at the lateral boundary of the irradiation band. If the stimulation is intensified, the sensation tends to change to an oval shape (always in the midline) and the double localization disappears. It has not been possible to observe this additional localization on the left side. There are strong analogies with polyopia since in the latter the polyopic deviation appears on the right in central vision and when the tilt of the image of the test arrow is about 30°-40°. Polyopia disappears in good vision and without image tilt perception, whereas on the contrary, it becomes triplopia (a virtual image on each side)

when vision worsens and the perceived tilt of the central image is greater but without reaching 90°. Whereas in the visual field polyopia is easily obtained in peripheral vision, in touch it has been impossible to obtain polyesthesia outside the referred midline. There are certain similarities between vision and touch in the way in which polyopia and polyesthesia appear, but the greater difficulty of examination and objectification of tactile phenomena may perhaps be the cause of an incomplete parallelism in this phenomenon of multiple localization. From the results obtained, this phenomenon seems to be less pronounced in touch than in vision.

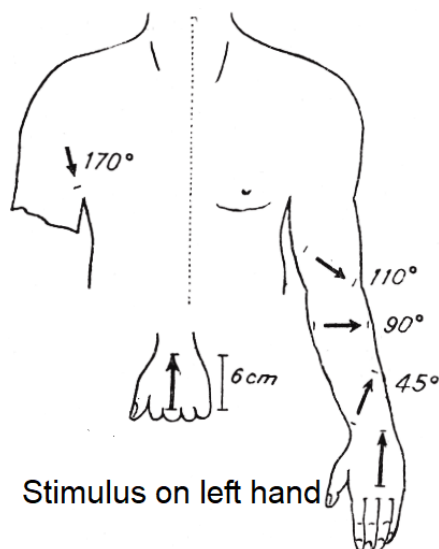
### **24.2.2 Inverted perception of a rectilinear stimulus**

If the stimulus has the form of a straight line and is applied statically on the skin, a deviated and contralateral perception is also obtained, and in addition, a tilted perception analogous to the already studied tilted visual perception of a vertical arrow. Thus, the study of perceived orientation in touch is more complete by means of a rectilinear stimulus.

This type of test can be performed in any region of the body. However, it is easier on the upper limbs since, as in the study of stimulus localization, they provide ample space to carefully follow the process if the stimulation is applied quite distally. In the test, the straight edge of a metal sheet is placed on the back of the left hand, being in contact about 6 cm along the hand (see Fig. 127). The intensity of the stimulus is determined by applying to the metal sheet weights ranging from 200 g to 3 kg. In some tests it is possible to use the compensation method analogous to that used in vision (tilting the test arrow in the opposite direction to be perceived vertically upward). But due to the normal tactile imperfection to perceive orientation accurately and, above all, due to the irregularities of the back of the hand (tendon prominences, etc.), such a procedure is much less useful than in vision. To overcome certain shortcomings and confirm some data, it is advisable to use a moving stimulus by tracing a straight line on the skin, which is easier and quicker to apply on most areas.

By varying the intensity of pressure on the skin, the corresponding perception evolves in a complex way, changing in unison the following features: localization of the perceived straight line, its size, tactile irradiation (diffuse perception of the edges), orientation, and the corresponding quality (diverse pain). The evolution of the orientation is roughly as follows as the intensity increases. In the inactive state, only the maximum inversion is contralateral (upper third of the contralateral arm), although homolaterally the rotation of the straight line is very considerable, probably at least 110°, until it straightens while proximal deviation decreases. Under maximum facilitation by muscular effort the straight line is always localized homolaterally, but the maximum limit of rotation is quite large, about 110°. Proceeding carefully, it is not difficult to obtain a large number of positions and orientations of the perceived line, which allows to get a very good idea of the development of the process. But due to the characteristics of the test, the tested subject must verbally report the situation reached, which entails obtaining less accurate data than in analogous experiences on visual image orientation, in which the

subject must only report when the arrow is seen in a vertical upward position (compensation method). However, repeated tests have shown us that it is possible to obtain sufficiently reliable data to evaluate the process quantitatively and represent it graphically. Figure 127 graphically illustrates the process schematically, and Table 33 shows the results obtained.



**Figure 127.** Perceived position of a 6-cm straight line stimulus applied with varying pressure on the back of the left hand of subject M in the inactive state. The localization and orientation of the straight line in the course of the process is indicated. The maximum rotation takes place contralaterally. The degree of rotation correlates with the degree of proximal deviation (see Table 33).

**Table 33.** Perception of a 6-cm straight line stimulus applied with varying pressure (applied weights in grams) on the back of the left hand of subject M in the inactive state. See Fig.127.

<i>Stimulus (grams)</i>	<i>Perceived orientation</i>	<i>Phase, Localization</i>	<i>Size</i>	<i>Quality</i>
280	Looks vertical	III, Contralat. arm close to shoulder	~0.7 cm?	Pressure
340	Tilted?	IV, Lower half homolat. arm	May be larger	Very slight pain?
500	Oblique	IV, Homolat. elbow or below	~1.5 cm?	Mild pain
850	Transversal	IV, To the half of the homolat. forearm	Larger	Painful
1000	Somewhat oblique	IV, More distal	Like previous	More painful?
1300	Very oblique	IV, To the wrist	~ 3 cm	Severe pain
2000	Close to vertical	IV, To the hand	Longer	Acute pain
3000	Almost or totally vertical	V, Hand dorsum	~ 4 cm	Unbearable pain

The following development is obtained when the pressure is increasing. At first, when a well-developed phase III is reached, something like a very short line in the center of a more elongated and diffuse irradiation can be perceived in the upper part of the contralateral arm. Its orientation is difficult to perceive accurately given the small length and faintness of the line, and may give the sensation that the line is oriented vertically, i.e. following the axis of the limb. However, by appealing to the inversion of motion on the skin when the stimulus has the intensity of phase III, it is shown that a motion on the dorsum of the hand from top to bottom (from proximal to distal) gives rise to a sensation of brief and rapid motion on the upper part of the contralateral arm and with practically inverted direction, i.e., from bottom to top. There is possibly, as in vision, a tilt limit with respect to the vertical, without a complete inversion of 180°, but it is technically difficult to prove this in touch. Phase III ends soon, followed by the transition to the homolateral side and the beginning of phase IV. As already noted in the tests of localization of a point stimulus, it is difficult to determine accurately at what level of the limb the change of side occurs when the stimulus intensity on the dorsum of the hand increases, but it most likely takes place somewhat above the elbow. Thus, a proximal part of the arm belongs to the contralateral localization, and the remaining distal part belongs to the side homolateral to the stimulus. The most noteworthy feature of phase IV now is the significant orientation disorder involved, perfectly demonstrated with the straight line test. It turns out that the localization defect (centripetal deviation) is always correlated with the orientation defect. Thus, in the case of maximum facilitation, which does not allow contralateral localization, it is possible to find a strong rotation of the straight line greater than 90°, which would allow to set the limit of orientation disturbance in homolateral localization.

A rotation of the perceived line that is easy to determine is that of 90°, since when the straight line stimulus is applied on the longitudinal direction of the limb, the altered sensation must correspond to a totally transversal orientation, without obliquities, and which is localized well below the homolateral elbow. It can be said that this transverse subjective line (midway of full inversion) is situated towards the middle of the limb, i.e. equidistant from the site of application of the stimulus and that of the onset of perception of phase III. As the intensity increases, the perceived length of the straight line increases, which is perceived more clearly from the transverse position onwards. Finally, very high intensities are necessary to try to suppress the centripetal deviation and the inversion residue. Unbearable pain is then produced which easily triggers defensive muscular contractions, impurifying the state of inactivity. When reaching the maximum recruitment (sensory growth), it is very difficult to say if there are abnormal remnants of deviation and tilt, but in analogous tests in vision, their existence is verified with relative ease, and here in touch they should also be admitted. Thus, at the end of the process, the perceived line probably does not exceed 4 cm (compared to 6 cm for the test line), to which must be added a weak centripetal deviation of about 2 cm and a rotation of about 10°, although all this has been deduced theoretically and according to other tests.

These tests are completed by considering the action of facilitation by maximum muscular effort. The results obtained are shown in Table 34.

**Table 34.** Same conditions as in the previous Table 33 except that subject M is under facilitation by maximum muscular effort.

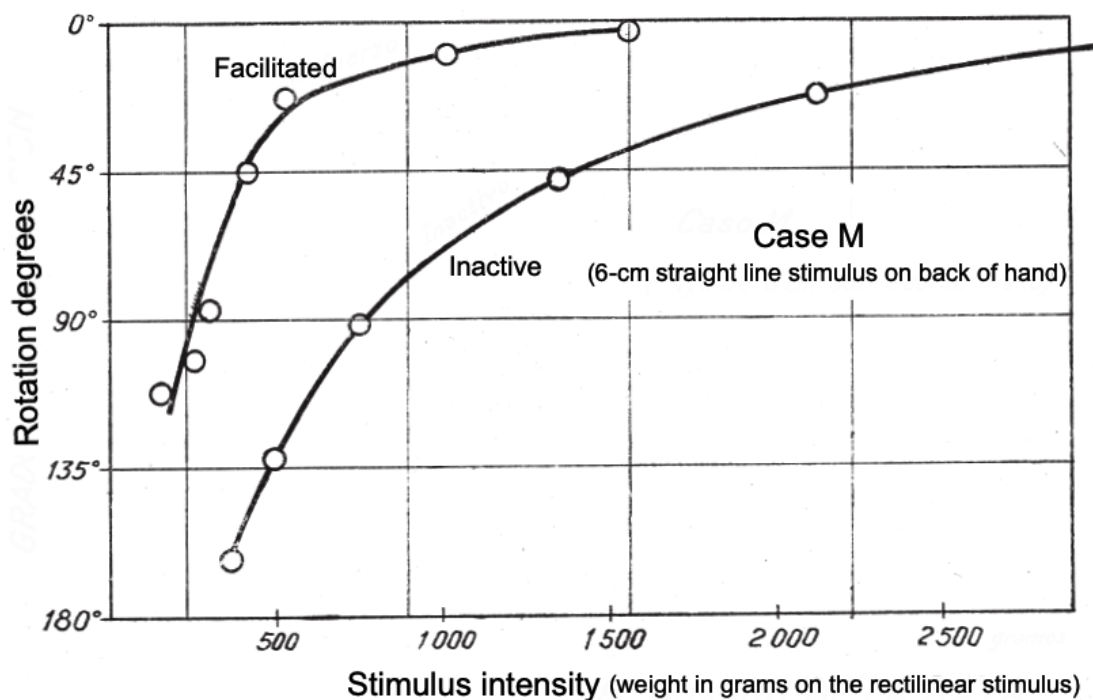
<i>Stimulus (grams)</i>	<i>Perceived orientation</i>	<i>Phase, Localization</i>
150	Somewhat tilted	IV, Lower third homolat. arm
200	About the same	IV, Homolat. elbow
300	Transversal	IV, Mid homolat. forearm
370	Oblique	IV, Toward homolat. wrist
500	More vertical	IV, Homolat. wrist
1000	Vertical?	V?, Hand
1500	Even more vertical	V, More central on hand

It was already indicated that under maximum facilitation the maximum rotation goes beyond  $90^\circ$ , i.e., a behavior analogous to that observed in vision, but the line does not pass to the other side and is positioned homolaterally, a little above the elbow. This maximum rotation is reached under facilitation with about 150 g on the stimulus, whereas about 500 g are required in the inactive state. Thus, facilitation saves about two-thirds of the stimulus intensity, this being valid for all other tests more or less. Apart from the difference in stimulation for the two states referred to (inactive and under facilitation), it can be stated that the correlation between the degree of rotation and the position on the limb is the same for both states; for example, the transversal orientation of  $90^\circ$  corresponds towards the middle of the homolateral forearm for the two states, and so on for the other orientations.

Under facilitation it is observed that not only are thresholds much lower but also the intervals between sensory levels are much smaller than in the inactive state. In short, there is an increase in excitability leading to greater sensitivity and faster variation in the process under consideration.

The considerable rotation of the perceived stimulus in a state of relatively moderate functional impairment such as that of subject M under maximum facilitation, points to the fact that orientation disorder in touch should be very frequent, as is also the case in vision, even if only as small tilts, less than  $30^\circ$  as should correspond to subject T. In subject T, the tilt should be strongly intensified after epileptic seizures, or more moderately intensified by the ingestion of alcohol, as occurs in other tests already described in the case of vision (see Sec. 13.1.1 in Vol. 1). In summary, whenever a proximal deviation in tactile localization occurs, an alteration of the perceived tactile orientation, which always accompanies such a deviation, can be presumed. Thus, it is shown that the localization disorder is more complex than it appears in a simple examination with a point stimulus, since spatial localization (exact localization of a sensation) involves both proximal deviation and rotation. The latter only revealed by a rectilinear stimulus as shown in Fig. 127. By plotting the data from the above tables, not forgetting that it is not possible to achieve as much precision as in vision, we obtain the curves of Fig. 128, which undoubtedly provide a valuable indication of the functional law of tactile orientation disorder.

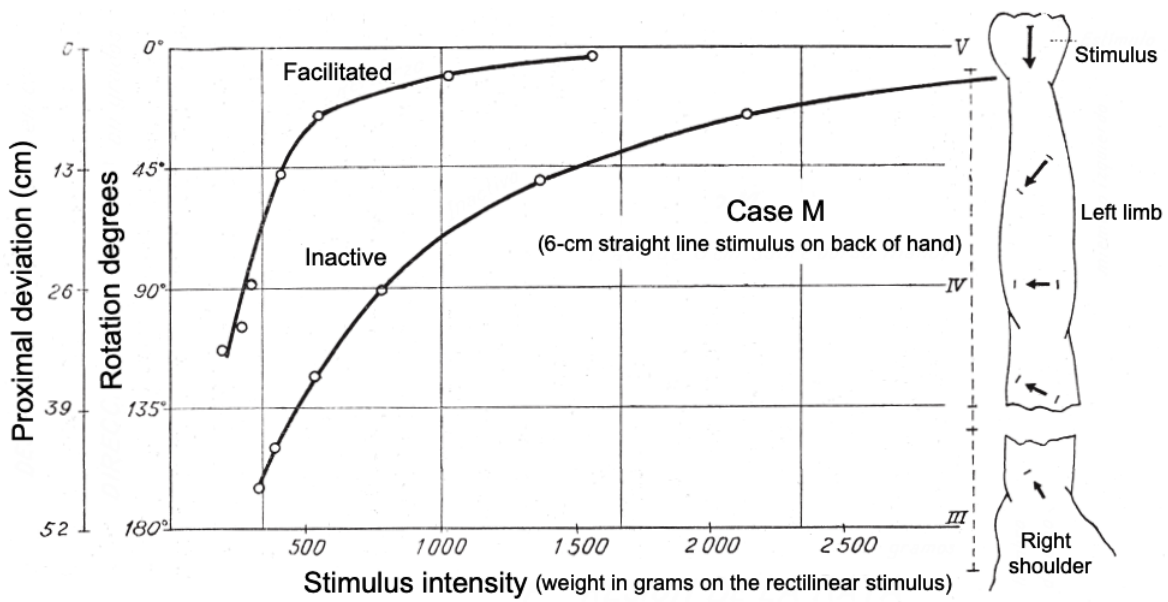
It is evident that these curves are similar to those of visual image orientation recruitment as a function of stimulus intensity (Fig. 56). It can be seen that at the beginning of the curves the sensory growth is fast and then becomes slower and slower following the general type of logarithmic growth according to the law of [Fechner \(1860\)](#). All that has been said about curves of a similar type, as those obtained in vision, is now applicable. Since it is difficult to use the compensation method with the same accuracy as in vision, it is impossible to determine exactly the maximum limit of rotation (inversion) in each of the two states studied. However, in view of the approximation that can be obtained, it is quite certain that it is of the same order as in vision, i.e., in the inactive state,  $160^\circ$  to  $170^\circ$ , and under facilitation, about  $110^\circ$ . When starting from the inactive state with very pronounced rotations, and changing to the state of maximum facilitation, the facilitation effect is very easily noticed, as can be seen in the curves of Fig. 128, the correction in the rotation being about  $80^\circ$  to  $90^\circ$ .



**Figure 128.** Perceived orientation (in rotation degrees) by subject M of a straight line stimulus as a function of its intensity (weight on the stimulus), in the inactive state and under facilitation by maximal muscular effort. Note the difference between the two curves, especially the different maximum rotation limit and the slope at that point (the lowest one) in the corresponding curve.

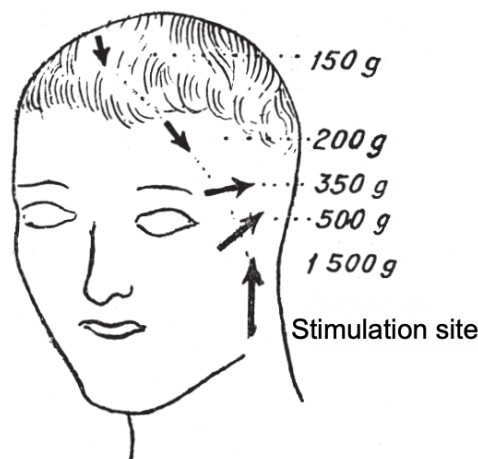
Since the development of normal orientation (re-inversion) with increasing stimulus intensity is correlated throughout its course with the growth of the body schema (correction of the deviation), both phenomena can be depicted simultaneously, as shown in Fig. 129. In fact, the development of localization (Fig. 113) and that of orientation (Fig. 128) are quantitatively similar. Such Fig. 129 provides us with a complete illustration of the actual process that takes place in the perception by subject M of a rectilinear stimulus.

As for the *sense of rotation*, the characteristics are also the same as in vision (see Sec. 12.3 in Vol. 1). Thus, rectilinear stimuli in both halves of the body rotate in opposite directions as the stimulus intensity decreases. On the right half of the body the rotation is counterclockwise and on the left half it is clockwise, i.e., outward in both cases. In the midline of the body, whether it be the forehead, chest, etc., the rotation is counterclockwise, i.e., toward the right side. As we have already seen, the rotations in each half of the retina of a single eye are in opposite senses. However, in the center, the rotation is outward in each eye. The sense of rotation can be easily determined on the different parts of the body by stimulating with a rectilinear motion over the skin and exerting a little facilitation so that only a moderate rotation of about 40° occurs.



**Figure 129.** Same as Fig. 128 but including proximal deviation (in cm). On the right, a schematic drawing of the size, deviation and corresponding perceived orientation on the right shoulder and left arm.

The pressure test on the rectilinear stimulus can be performed on any part of the body, although with varying experimental ease and accuracy, obtaining results of the same type as in the hand. On the cheek, the test can be performed with a much lower stimulus intensity than on the back of the hand, either because of thinner skin or greater nerve sensitivity. The subject must be appropriately positioned in the inactive state such that the edge of the metal sheet rests horizontally on the left cheek.



**Figure 130.** Test with a rectilinear stimulus on the left cheek of subject M in the inactive state. As in the upper extremity, most of the rotation corresponds to the homolateral side. Note the localization corresponding to 90°.

Figure 130 shows the stimulus location and the perceived localizations and orientations according to the weight applied on the rectilinear object. With a weight of

150 g, a weak localization is felt on the opposite cranial vault (right); with 200 g, a sensation of oblique line is obtained on the left forehead; with 350 g, the line is better felt, localized horizontally between the eye and the homolateral ear (left temple); finally, with 500 g, the line is felt as oblique (Fig. 130), more toward the cheek, without reaching the place of application of the stimulus; being necessary about 1500 g to reach this place. Thus, the values obtained correspond to those of the hand test under facilitation (see Table 34). Compare Fig. 130 with Fig. 125 with respect to the path followed by the localization.

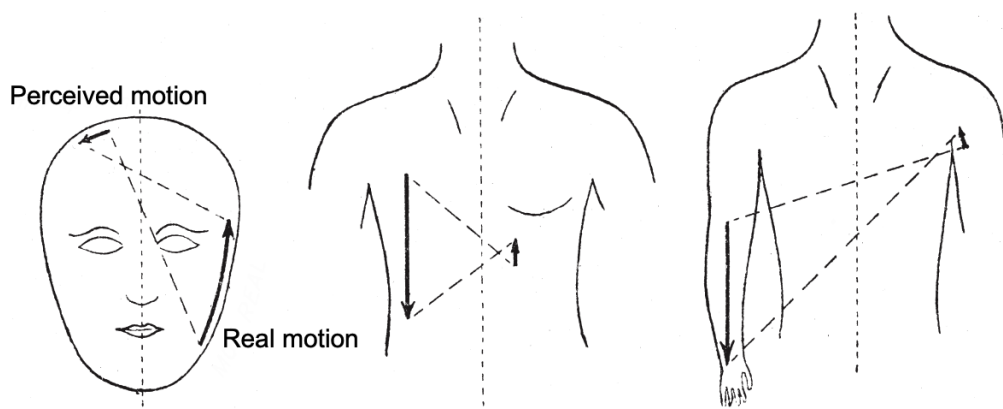
### 24.2.3 Inverted perception of motion on the skin

In the case of a moving stimulus on the skin, the same characteristics as in the static straight line case are given, but in addition, it provides other information such as the direction of the perceived motion and the alteration of the perceived trajectory, due to the variable inversion. It should also be considered that intrinsic changes in trajectory and velocity occur in parallel with the degree of inversion, as mentioned previously in Sec. 23.2.

The direction of motion and the corresponding localization are of course dependent on the stimulation conditions, namely stimulus intensity (cutaneous pressure of the moving stimulus) and its duration (path length and speed). Any variation of these factors influences the sensory recruitment (development) and thus the direction of motion. For a given motion on the skin with a fixed trajectory, constant intensity and moderate speed, capable of eliciting a very little altered perception (in localization, direction of motion, length and speed) in the inactive subject M, it happens that as the speed increases, the anomaly in orientation and other associated factors tends to be clearly shown as follows. When descending to the threshold of motion perception, which corresponds to phase III as we know, the localization becomes contralateral and very close to the midline, the speed felt is the maximum possible and the direction of motion is felt practically inverted (close to  $180^\circ$ ). The increase in stimulus speed certainly means a shortening of the stimulation time, thus preventing the development of the function which then shows asynchronous levels of different degrees of inversion. The result obtained is the same as in the tests on visual perception of the direction of motion of a moving object at different speeds (see Sec. 13.5 in Vol. 1). Obviously, any variation of the other stimulus parameters (intensity, path length) produces a similar effect. When the maximum inversion is obtained, the felt path is approximately 1/10 of the real path of the moving stimulus. Figure 131 shows the complete inversion of motion on different parts of the body. In addition to the sense of motion, the other concurring features are explained by the fact that this stage belongs to phase III. Thus there is an intense proximal deviation towards the midline, as well as a sensation of both strong path reduction and high speed, all as a result of the dynamic reduction in the *space and time sensory dimensions*, according to what has already been studied. The motions represented in Fig. 131 have vertical orientation, but any other orientation shows the same result, so a motion from front to back originates a sensation from back to front, and so on for other cases.



The static straight line test is particularly suitable for a more precise determination of the degrees of tilt (rotation) as a function of stimulus intensity, i.e., for the detailed study of the evolution of the process, whereas the test with moving stimuli is more appropriate for obtaining general data on the sense of rotation and inversion. The examination of the direction of the felt motion by means of moving stimuli offers an important complement to the static straight line test, both because of the ease of performing it and because it reveals more clearly the perceived orientation.

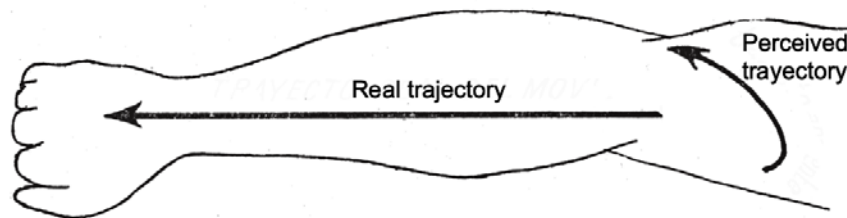


**Figure 131.** Total reversal of motion over the skin in different parts of the body of subject M in the inactive state. The large arrow corresponds to the moving stimulus and the small arrow to the perceived motion, which shows reversal of motion direction, intense centripetal deviation (phase III) and strong spatial and time shortening that produces a sensation of increased speed.

When the moving stimulus can promote a greater recruitment (sensory growth), there is no longer such a complete inversion nor, consequently, contralateral localization, but only different tilts on the side homolateral to the stimulus. An interesting test is the one performed by moving a stylus with moderate pressure on the skin, uniform moderate speed and very long trajectory over almost the entire upper limb of subject M in the inactive state. A homolateral, non-inverted trajectory that deviates considerably from the real trajectory is then obtained subjectively, as shown in Fig. 132.

Because of the great length of the real path and the moderate pressure, it results that at the beginning of the perception, the direction of the felt motion undergoes a great deviation, and subsequently, due to the continued development of the function, the alteration of the direction tends to be progressively corrected (see Fig. 132). There is undoubtedly a cumulative action on the nervous centers, with the result that the final effect differs from the initial one, even though the stimulation is uniform in all its aspects. The path length is about 35 cm, from the lower third of the arm to the middle of the back of the hand. However, the path felt by the subject is only about 10 cm, so it has been reduced to about 1/3 of the real value. At the beginning of the perception, the direction of motion is felt rather inverted, it could be evaluated at about  $120^\circ$ , but it lasts very short, and the path then becomes somewhat larger and transverse ( $90^\circ$ ), from which it gradually changes to less and less rotated and longer paths. Finally, a certain deviation of about  $10^\circ$  always remains. In short, the sensory level is always growing during the motion of the object, and since the length of the path and the degree of inversion evolve together (the

greater the inversion the greater the spatial shrinkage), the above mentioned deviated trajectory is obtained, which in reality would be the *spiral deviation* referred to above. Also, the uniform speed of the real motion should undergo a change similar to that of space, as we know, so a high speed of the moving stimulus is felt at first, and gradually decreases as the trajectory straightens out and increases in length. As for localization, a proximal deviation of the entire trajectory is obtained, which is located between the lower third of the arm and the upper forearm, as shown in Fig. 132.



**Figure 132.** Real trajectory and perceived trajectory of a stimulus moving over the skin of subject M in the inactive state. The perceived trajectory is deviated proximally, with a shorter path (1/3), and is perceived with higher speed, especially at the beginning.

This alteration of motion that involves so many aspects is a logical consequence of the impairment of various factors which come into play, and a great deal of experimental thoroughness is necessary to bring out such an alteration. If the subject under examination disregards the initial path due to its smallness and sensory weakness, he will only state to have felt a somewhat oblique motion on the end of the arm; but if he pays more attention, he will become aware of the initial path resulting from the initial inversion.

In relation to this alteration of the trajectory is the deformation of figures drawn on the skin (see Sec. 23.3), unless the drawing is of large size and is made with great pressure.

### 24.3 Perceived direction of joint movement. Deep sensitivity

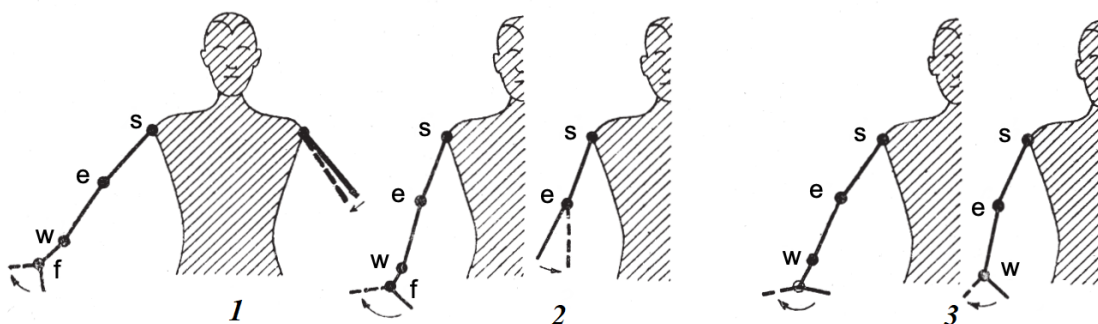
Here we study the perceived direction of joint movements produced both passively and actively. We already know that there is a parallelism between the perception of motion on the surface of the skin and the perception of deep (joint) movement. The most important phenomena are manifested in passive movements of the limbs, the head, and in the peculiarities of active (voluntary) movements.

#### 24.3.1 Inversion of passive movements in the extremities

For the study of the direction of movement, it is not possible to use repeated stimulation by means of an oscillatory movement of an extremity. It is indispensable to perform only a single movement in order to observe in a well-defined manner the anomaly in the direction of the perceived movement. Such movement must be of large amplitude and vigorous in order to elicit sensation of movement in the inactive subject M, as explained

in Sec. 23.4, which is now of great importance. Depending on the energy used, very large in all cases, the localization of the movement undergoes a more or less strong proximal deviation. In conjunction with this deviation there is a certain degree of inversion in the direction of the perceived movement as well as intrinsic modifications of it, analogous to what occurs with a stimulus moving over the skin.

When testing the index finger (metacarpophalangeal joint) of subject M in the inactive state, the following is obtained. For a given energy applied to the finger in a passive movement, the felt movement is localized in the contralateral shoulder joint in such a way that when the finger performs an extension, a flexion of the arm is perceived as shown in Fig. 133, 1. This movement is felt to be very rapid and very short in its path compared to the real movement of the finger. If, for example, the amplitude of the finger movement is about  $100^\circ$ , the amplitude of the perceived movement in the arm would be about  $10^\circ$ , just enough to be perceived by the patient. The direction of the movement perceived is practically inverted, being impossible, due to the experimental conditions, to appreciate exactly what may be missing to reach the absolute inversion.



**Figure 133.** Schematic drawing of inversion and proximal deviation in passive joint movement of a finger in subject M in the inactive state. *1*: A vigorous finger extension produces a sensation of weak flexion of the arm at the contralateral shoulder. *2*: A more vigorous extension of the finger causes sensation of forearm flexion at the homolateral elbow. *3*: Much greater amplitude of the finger extension and with more energy produces a sensation of extension in the homolateral wrist.

If the passive extension of the finger is performed more strongly, the sensation of movement is perceived as a flexion of the homolateral elbow (Fig. 133, 2), that is, there is a considerable, albeit incomplete, reversal of the direction of movement. Finally, a finger extension with the maximum possible amplitude and energy (it is convenient to press on the articular limit) produces the sensation of a deep movement at the wrist, with approximately the same direction of the finger movement (extension). We already know that no matter how strong and large the movement is, it is not possible for subject M to localize it at the real stimulus site (metacarpophalangeal joint).

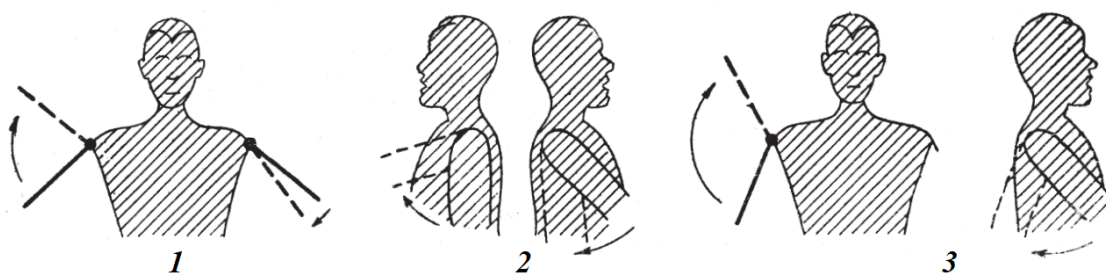
These abnormal localizations (contralateral shoulder, homolateral elbow, wrist) and their respective directions of movement are in perfect agreement with the results of the straight line test in which the tilt felt correlates with the proximal deviation. Thus, at the level of the elbow, the degree of inversion of the straight line is still very pronounced, whereas at the wrist, the line feels rotated only about  $30^\circ$ . In joint movement, it is difficult

to obtain further data from the patient on the degree of inversion and whether it is clockwise or counterclockwise according to the intensity of the stimulus, so we must be satisfied with a rough indication of the joint extension and flexion. However, it is very evident that the perceived movement on the homolateral side is much larger and slower than on the contralateral side. At the wrist, the movement is felt at least three times larger and slower than when felt at the shoulder.

The same test performed under facilitation by maximal muscular effort excludes contralateral inversion at the shoulder, as we know. Depending on the stimulation energy, flexion is felt at the elbow, or extension at the wrist and even the finger, the latter movement being possible to be perceived under facilitation. When the localization is at the wrist, in addition to extension, possible rotation is felt, whereas at the finger, the perceived extension is more correct. When the localization is felt in the finger, the perceived movement is the largest amplitude and slowest of all those that can be felt in the different joints with proximal deviation.

Further tests in the inactive state using passive motion of major joints serve to complete the data on movement direction. Thus, by passively abducting the right arm about  $90^\circ$ , a sensation of adduction of the contralateral arm of about  $10^\circ$  and with very rapid movement is obtained (Fig. 134, 1). The subject cannot say whether he also feels the arm deviates a little backwards or forwards, but in some tests he has felt it, and in that case it would not be a complete inversion of  $180^\circ$ . If the passive movement of the arm is forward, the felt movement is backward in the contralateral arm as shown in Fig. 134, 2. But if the passive abduction movement of the right arm is much more intense than in case 1, then there is neither contralateral localization nor reversal of movement direction. The perceived movement is localized in the same shoulder and undergoes a partial inversion of only about  $90^\circ$  in such a way that the abduction is felt as a backward movement, as shown in Fig. 134, 3.

The same happens for the other joints, both of the upper and lower extremities. No further details seem to be necessary.



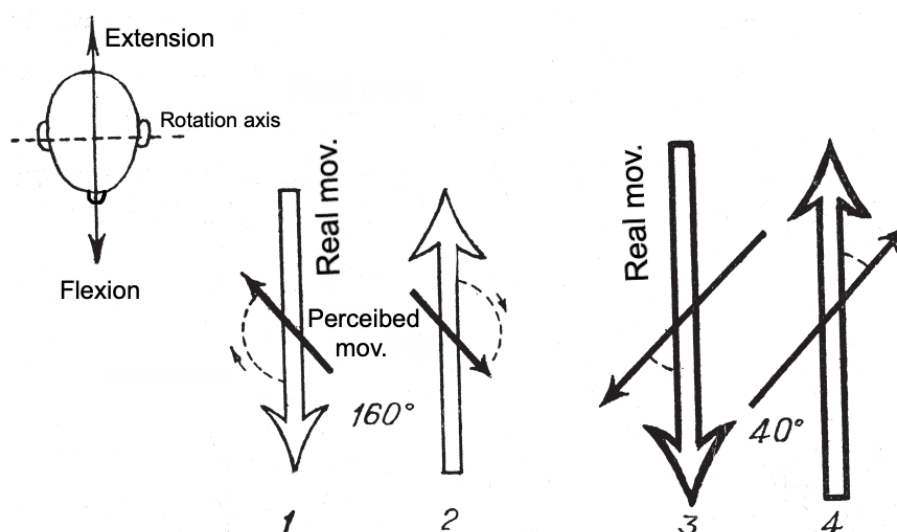
**Figure 134.** Schematic drawings of the reversal of direction of passive articular movement in the arm of subject M in the inactive state. **1:** Abduction of the arm is perceived as adduction of the opposite arm (complete reversal of direction in the contralateral arm) and in one tenth of amplitude. **2:** Arm forward is perceived as a weak and rapid backward movement of the other arm (complete reversal of direction in the contralateral arm). **3:** Very vigorous abduction is perceived as a backward movement of the same arm (partial reversal of movement in the same arm).

### 24.3.2 Inversion of passive movements of the head

The direction of the articular movements of the head can be studied more easily and accurately than in the case of the extremities, and because of its importance, it deserves a new subsection.

As we already know, in this case no deviation of localization is possible, and in all tests the movement remains referred to the same site (neck joint). Therefore, it is necessary to pay attention only to the direction of the joint movement according to the intensity of the stimulation.

In subject M in the inactive state, a passive head movement forward (flexion), or backward (extension), with moderate intensity but sufficient to produce excitation, is perceived as follows. If the movement is forward, the perceived movement is reversed although not completely, so that the exact direction of the perceived movement is backward and to his right as shown by the black arrow in Fig. 135, 1. If the movement is backward, the perceived movement is forward and to his left as shown by the black arrow in Fig. 135, 2. Therefore, the rotation that takes place is always clockwise, the head behaving as an organ of the midline of the body.



**Figure 135.** Diagram on the inversion of the passive movement of flexion and extension of the head of subject M in the inactive state. If the movement is large but not vigorous, the perceived movement is rather inverted (small black arrows in 1 and 2). If the movement is very large and very energetic, the change in direction of the perceived movement is much smaller, as indicated in 3 and 4. Note that the rotation perceived is always clockwise, and that the different perceived length of the path is correlated with the degree of inversion.

A very large (to the limit) and extremely intense movement is necessary for there not to be such an accentuated inversion, and then the perceived direction is rotated about  $50^\circ$  with respect to the direction of the real movement. Thus, the flexion movement is felt in

the forward direction and to his right (thin arrow in Fig. 135, 3), and the extension movement is felt in the backward direction and to his left (thin arrow in Fig. 135, 4). These perceived directions are accompanied by a longer and slower felt movement than when there is an almost complete inversion. When the passive movement is of intermediate intensity between the two previous types, the direction of the perceived movement is rotated about  $90^\circ$ , and therefore the flexion-extension movement causes a sensation of movement with a transverse direction from side to side. Flexion causes a sensation of movement to the right, and extension, to the left.

Under facilitation by muscular effort, if the head movement is very weak, some inversion can still be obtained, although to a lesser degree (about  $100^\circ$ ) than in the inactive state. If the amplitude and speed of the movement is moderate, the perceived direction of the movement usually changes only by about  $40^\circ$ . If in addition the facilitation is very strong, the perceived direction changes only very slightly, and if the passive movement is a little larger, there is practically no subjective change in the direction of the movement. All this is verified by means of the compensation method, that is, by performing a real passive movement of flexion or extension with a certain inclination that the subject perceives as straightened.

Other tests by means of passive head movement in other directions provide similar results. Thus, in the movements of rotation from side to side in the frontal plane, it is observed that a movement to the left is felt as a movement with some forward flexion and rotation to the right, i.e., there is almost complete inversion. It should be noted that for the subject to be able to describe the felt movement with ease, the most appropriate movement is flexion or extension.

Aside from these directional disorder in passive head movements, this region has been especially significant for the remarkable phenomena of sensory-motor incongruence in active (voluntary) head movements, which will be studied below.

### **24.3.3 Inversion in active (voluntary) movements. Incongruences**

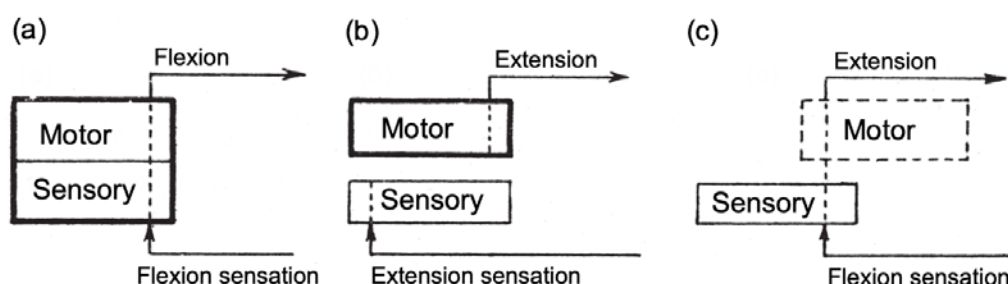
When studying the disorder of the direction of movement through passive movements, the patient could remain indefinitely ignorant of his disorder. This is because with his eyes closed, ignoring the type of stimulus and limiting himself only to the perceived sensation, it is impossible for him to notice discrepancy between cause and effect. A very different situation occurs when the patient takes an active part in the stimulus by producing it himself. Thus, when examining the alteration of the perceived direction of voluntary movements, it is observed that, in the range of stimulation intensity that causes subjective inversion, a significant disparity appears between the direction of the executed movement and that of the perceived movement, opposite to each other due to the effect of inversion. This is a *sensory-motor incongruence of the movement direction*, and can be noticed by the patient himself. It is clear that, from the above, this disparity extends to the intrinsic character of the movement, since it is perceived as having a shorter path and higher speed than the movement performed.

The mechanism of execution of a voluntary movement by subject M will be studied in detail in a separate section. Suffice it to say now that if this subject is in the inactive state, any voluntary movement is impossible for him, since he needs a certain degree of facilitation for the body schema to be activated and thus make it possible to guide the motor impulse adequately. If the facilitation is weak (sustained muscle contraction of moderate intensity), the movement short and of a single joint excursion (adequate to arouse a kinetic sensation in the initial phase), then a reversal of the direction of movement is perceived along with a proximal deviation and a contralateral localization. For example, closing one hand produces the sensation of abduction of the opposite arm. It should be noted that in the above circumstances, facilitation is used rather to "find" and perform the movement that the subject is asked to execute. The facilitating muscular effort, being weak, seems to modify very little the movement in question, allowing a very pronounced inversion to be perceived.

A discordance occurs between the motor command, usually well executed, and the sensory response indicating a completely different location and direction of the movement. Let us analyze such a curious phenomenon in order to determine what kind of independence exists between the motor function and the sensory function involved in the voluntary action, and what states of adaptation can be achieved. The order to perform the movement is carried out by the subject with a certain delay due to the time spent in activating the body schema and selecting the movement. This movement, being very simple, does not necessarily present pathological deficits (ataxia, etc.), although the proprioceptive control is not normal. In such conditions, the subject may become aware of the aforementioned sensory-motor incongruence. In fact, if asked if he is sure he performed the assigned command, or if he is confused by the disparity, he responds that he knows he moved his hand as commanded, even though he felt the movement in the contralateral shoulder. He seems to be sure of the specific movement he performs, but various tests attest that this conviction is very shaky, as seen in the following test. Instead of telling him to close a certain hand, he is commanded to move an arm (without specifying the side) so that the verbal command is nonspecific. He perceives an opposite movement in the contralateral arm, but spontaneously tends to believe that the moved arm is the arm where he perceives the movement. This is because the executed movement has been improvised at random without taking on a definite character, and it seems that only the sensory effect acquires prominence. Thus, when he is asked about the movement performed, he answers with total certainty referring to the sensory sensation and not to the motor action. This means that he ends up ignoring his own voluntary movement because a *change* of reference is established to overcome the aforementioned sensory-motor incongruence which is thus suppressed, and a certain functional balance appears. In this test there are special conditions, but even in the case of ordering the subject to execute a well-specified movement that he succeeds in carrying out, it always happens that finally, when continuing the test, the movement felt "absorbs" the one executed voluntarily. Thus, it may happen that the subject believes that he has made a mistake in executing the order, or that he does not remember which side was indicated to him.

This process is studied in a more complete and simple way by means of *active head movements*. In particular, the aforementioned change of reference with the consequent

functional balance excluding incongruences becomes clearer. Of course, all these phenomena, like many others, refer to tests within certain limits, and outside them, in the ordinary life of the subject, these phenomena do not arise, or go unnoticed, not compromising his activity. Since the subject must already apply some facilitation to promote the action of voluntarily moving the head, the movement commanded to him must be of small amplitude to obtain an inversion of about  $150^\circ$ . Thus, when the subject, complying with the order, performs a small flexion of the head, he feels an extension. The motor impulse has been well guided, but if he continues to pay attention to the order, he must feel an incongruence due to the sensation of inverted direction when the impulse of the movement performed almost disappears. He then tries to rectify by himself to fulfill the command, in the following way. He is spontaneously guided by the sensory information, and automatically performs the opposite movement to the one ordered to achieve concordance between the order and the sensation, disregarding the motor engram. When he remains sufficiently attentive to the course of the process, he is often seen to first move his head in the direction ordered; then when he notices sensory incongruence with the order, he makes small trial-and-error movements to obtain articular information, and finally initiates the opposite movement to that ordered, which suppresses the aforementioned incongruence. In such a situation, an automatic change of reference has occurred, i.e. the motor order ceases to prevail, and the action takes as reference the sensory factor which is the only one that controls it. The patient does not really know what voluntary movement he has executed, since he always responds according to the sensory information to which he is totally adapted. At this point, if the patient continues to receive orders indefinitely, he will constantly have the sensation of fulfilling them correctly by performing a movement opposite to the one ordered. The sensory-motor incongruence is thus overcome, and a balance is reestablished by disregarding the motor element, since it is not possible otherwise in such circumstances. This leads to the remarkable consequence that the active (voluntary) movement has become, through this change of reference, a *pseudo-active movement* bordering on passive movement. This whole singular process is illustrated by the diagram of Fig. 136.



**Figure 136.** Diagram to illustrate the incongruences and the change of reference in active (voluntary) movement of the head. (a) Normal subject: an active flexion movement produces a sensation of flexion. (b) Sensory inversion stage in subject M: an active flexion movement produces a sensation of extension. There is a sensory-motor incongruence due to the sensory inversion. (c) Subject M, who undergoes inversion, continues to receive the command to perform a flexion. However, once the sensory factor is manifested, it ends up absorbing (dominating) the sensory-motor complex. The motor system then suppresses its own direction and takes as reference the sensory perception which guides the action.



In short, it can be stated that the motor action can run by itself, but then it is very unstable and diffuse since it lacks the support of the epicritical proprioceptive sensitivity, and in addition, it is interpreted in a contrary way due to the sensory inversion. Thus, an articular adaptation (sensory predominance) is automatically established as the only resource to achieve congruence. At that point the active action becomes a quasi-passive action since the character of the executed movement is ignored. The impulse is “promoted” by the motor system but is “guided” by the inverted sensory cue.

This automatic change of reference is not an isolated fact in the series of anomalies of our patient M. It could be compared to the visual phenomenon that occurs in tests on “induced motion”, i.e., seeming movement of the visual scene, which is fixed, as the subject's body rotates (Sec. 16.2 in Vol. 1). This latter phenomenon is due to a failure of tactile information in the process. Such a phenomenon may also occur in a normal subject, for example, in the well-known seeming motion of the moon through the clouds, among many other cases. Thus, a new type of perception appears due to a reorganization in the set of factors involved in the process.

This “accommodation” to the sensory factor is maintained as long as the inversion is sufficiently pronounced, but it can easily be destroyed as soon as the subject is asked to make, instead of a small head movement, a large movement that no longer provides any inversion or only produces a small tilt. In this situation, incongruence appears for a moment because the patient still continues with an abnormal reference system that is no longer necessary for congruency. The return to normality, i.e. the complete reestablishment of the motor reference, does not occur suddenly but through trial-and-error movements and confusions, as in the abnormal process of sensory adaptation. Thus, at first, he does not easily notice the incongruence that appears when the inversion is suppressed, due to the fact that the exclusion of the motor system has been very strong. All these tests are performed with eyes closed, but even with eyes open the result does not change much since he does not pay attention to his surroundings nor would it benefit him in orientation, given his visual image orientation disorder and other disorders.

Although the patient gets used to the unexpected and paradoxical phenomena he experiences, all these tests on active movement make a strong impression on him. This is because the confusion in which he is immersed due to changes of reference, inconsistencies, etc., in an activity as immediate as voluntary actions, probably affects him more than many other disorders that manifest more indirectly.

Other types of sensory-motor incongruences in active movement can still be mentioned as a consequence of sensory deformation of the direction of very large movements, especially in the extremities. A curve-shaped deformation appears subjectively, similar to that studied in motion over the skin surface (Fig. 132). As an example the following test is worth mentioning. The patient (M) is asked to raise an arm from the natural hanging position of rest, forward to the horizontal, without deviating from the lateral sagittal plane of the body, lifting the entire limb in a uniform and non-violent manner. The movement perceived by the patient, standing and with moderate muscular effort, is as follows. He first feels a short backward and outward movement,

then a forward and upward curve finally falling out of the sagittal plane. The amplitude of the perceived movement (about  $30^\circ$ ) is much smaller than that of the real movement ( $90^\circ$ ).

In addition to the *change of shape* of the movement trajectory, its shortening and the change of speed (higher at the beginning) entail new disparity factors. The cause of the deformation of the active movement of the limb is the one exposed in the case of an object moving on the skin. The cause is the different degree of inversion according to the cumulative action of the stimulus on the nervous centers, etc., and we will not insist on this any further.

Finally, we must mention the disorder of orientation in mandibular movements, whether passive or active. Already in the normal subject it is very difficult to obtain a complete relaxation of the mandible that makes possible a proper examination of its passive movements. Therefore, we will refer mainly to active movements, which allows us to mention this case in this section. When the patient (M) opens his mouth voluntarily, i.e., makes a depression movement of the mandible, he can feel a movement in the head, towards the neck, due to the proximal deviation that accompanies the phenomenon, and in a quite inverted direction (backwards and to his right). Under moderate facilitation by muscular effort, the movement is usually localized in the mandible, and when he opens his mouth he feels a very incomplete inversion (sensation of lateral movement of the mandible). For all this to occur, it is necessary to properly instruct the subject on the type and strength of the movement to be performed, and to urge him to pay attention to the sensations he perceives from his voluntary movements.

## 24.4 Perceived orientation of the body schema

The alteration of deep (joint) sensitivity due to a defect in the direction of movement, among other disorders, affects the perception of the spatial orientation of the body schema (subjective position of the body in space). Moreover, as all sensory manifestations are closely linked to each other, this disturbance of the direction of movement is associated with a certain sensory level of the body schema, determined by body shrinkage, fragmentation, etc. (see section 7.5.1). If this sensory level is very low, any sense of spatial orientation of the body is suppressed, mainly due to the loss of body coherence.

If subject M is sitting on a soft chair, he does not feel his body at all if he is immobile and inactive. Only at the first moment of sitting down he has a diffuse non-localizable sensation (spatial phase I). In that situation it is impossible for him to determine the spatial orientation of his body. It is necessary that the subject (seated and in a natural upright position) apply a certain degree of facilitation by means of a moderate and sustained muscular contraction, especially of the trunk, for some rudiment of body schema to emerge. When the body schema is initiated under such conditions, he feels it quite deviated from the vertical, tilted to the right about  $60^\circ$ . If the subject is standing, well upright, requiring of course some muscular action to maintain that attitude, the subjective deviation of the body schema can be quite smaller, around  $40^\circ$ . Therefore, such a position involves a greater facilitation than that required to diffusely bring out the schema when seated.

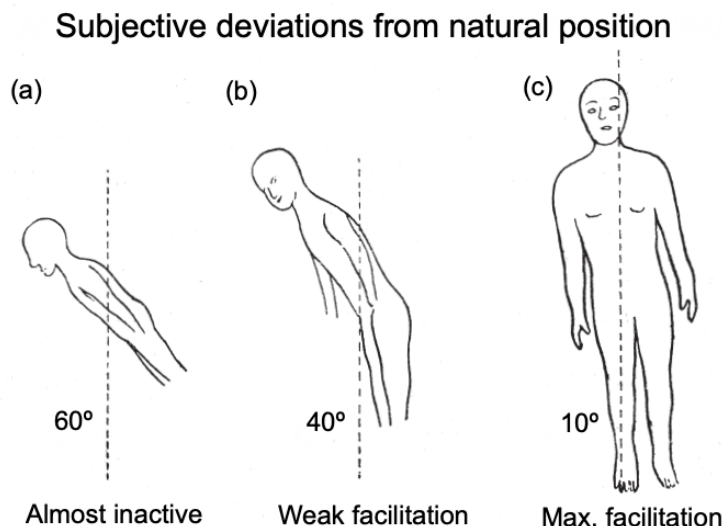
These results show how easily the *subjective tilt limit* is reached in the body schema. If in the sitting position the subject reduces the facilitation a little, all sensation of spatial orientation is lost because the body schema fades away. The maximum limit of subjective inclination could reach about  $70^\circ$ . These facts correspond to the general behavior in the perception of figures of all types (including the body figure), which require a fairly developed sensory level in order to be perceived, and at that level the spatial orientation function is only moderately affected. We have already seen this during sensory phase IV in the case of figures drawn on the skin (see Sec. 23.3), and also in the case of visual image inversion (Sec. 13, Table 14 in Vol. 1). In the latter case we saw that figures (e.g., optotypes used in visual acuity tests) already become blurred when the perceived image is tilted about  $40^\circ$ , all details disappearing before reaching  $90^\circ$ . If the degree of inversion is higher, only a diffuse shape is perceived (see case of the test arrow in Sec. 13 in Vol. 1).

From all that has been said, it can be inferred that for the subject to perceive a certain orientation of the body schema, he must reach a sensory development of at least spatial phase IV, to which corresponds a tilt of less than  $90^\circ$ . The rudiments of the body schema in more elementary phases are not enough to arouse a coherent sensation of the schema, which besides being very small and diffuse, tends to be fragmented (independence of its parts).

Examining the deviation of the body schema more precisely, it results that in addition to the rightward tilt, there is a certain backward rotation, and at the same time it seems that the whole perceptible body is somewhat tilted forward. It is therefore a complex deviation in several spatial directions that should be interpreted as a residue of the inversion of the whole body according to a *spiral turn*. The patient senses that the tilt is for the body as a whole, i.e. the body does not bend at the waist forming an angle, but rotates through the center of the trunk changing the whole body's orientation in space. The spiral deviation (a backward turn on himself) is better perceived when the tilt is very pronounced, about  $60^\circ$ - $70^\circ$ , but it is less evident at tilts of about  $40^\circ$ . As for the correction by means of facilitation, it should be noted that when standing and with maximum facilitation, the body schema is much more clear, but nevertheless there remains a small residual deviation to the right that may be about  $10^\circ$  or a little more. The different deviations according to the subject's states of activity are described by the subject, either verbally in an approximate way, or by indicating the position of an arrow rotating on a graduated circle.

As said, together with the alteration of the felt orientation of the body, the body schema evolves in shape and size. In a seated position and with weak facilitation by muscular effort but sufficient to awaken the body schema, the deviation felt is  $60^\circ$ - $70^\circ$  with respect to the vertical. In this case the schema is at the beginning of spatial phase IV (Fig. 110); the trunk is what takes on most relevance, the head and extremities probably less, the latter being only diffusely perceptible up to their middle, as shown in Fig. 137 (a). The *size of the body* is felt to be greatly reduced, both by the exclusion of distal parts and by the general spatial shrinkage (recall the reduction of the spatial threshold). As for these data, it should be noted that the perception of the body in a normal immobile subject is already somewhat diffuse, and this feature is even more pronounced in pathological

cases, being difficult a precise description. The data must be adequately “extracted” from the patient, since, as will be seen when studying postures, the defect in the perception of the body and postural alterations in the body schema tend to be ignored by a mechanism of pseudoagnosia that is very frequent in all types of pathological manifestations.



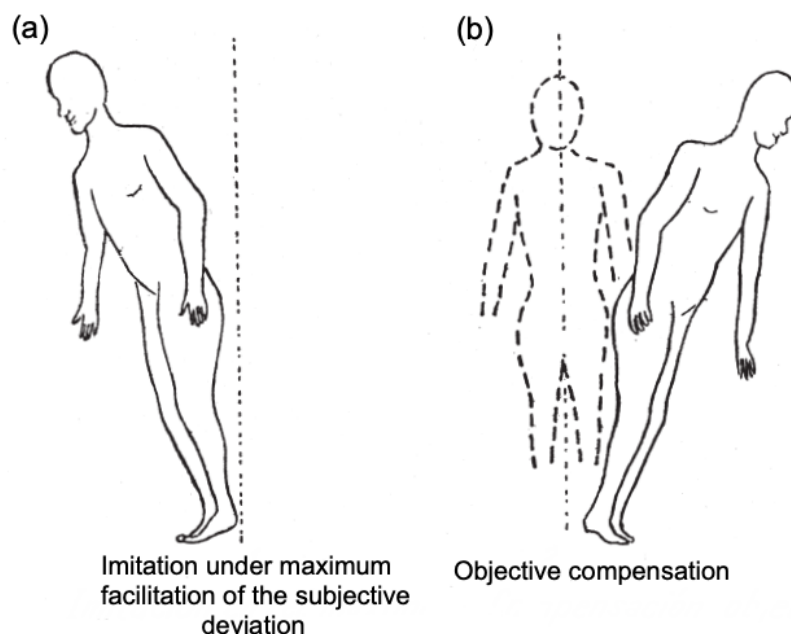
**Figure 137.** Subjective orientations of the body schema in patient M. (a) M seated and almost inactive: body tilted to the right  $60^{\circ}$ - $70^{\circ}$  and notable reduction in size corresponding to the onset of phase IV. It is the maximum deviation that can be felt at the beginning of the sensation of the body as a whole. (b) M standing and with more facilitation through muscular effort:  $40^{\circ}$  of deviation, phase IV more developed. (c) M standing and with maximum facilitation by muscular effort: residual deviation of  $10^{\circ}$  -  $15^{\circ}$ .

Note in the figures a tendency to spiral rotation (head turned).

For a deviation of about  $40^{\circ}$ , the size of the body schema increases a little more, the head and limbs are felt more clearly although the hands and feet are probably still missing. Finally, with maximum facilitation by muscular effort, there is only a subjective tilt of the body of about  $10^{\circ}$ , the size increases and tends to normal; although if the subject does not move, the most distal part of his fingers may be excluded. When standing, the difference in subjective height between the state of moderate facilitation and that of maximum facilitation can be about 20-25 cm. Sometimes, however, there may be a greater variation, since if he is standing and as inactive as this attitude allows, it may seem to him that he is as reduced in height as if he were kneeling. This reduction probably corresponds more to the sitting position, which is even more inactive [Fig. 137 (a)].

We can get a more precise idea of the nature of the deviation of the body schema by resorting to more objective methods. One of them consists of the subject under maximum facilitation imitating the deviation he feels in an almost inactive state. Another method is that of compensation of the subjective deviation by a real tilt in the opposite sense when the subject is almost inactive. Although the maximum facilitation by muscular contraction does not completely suppress the deviation, it is so small that it can be disregarded in these objective methods. The patient is then able to imitate the posture he feels when he is inactive, as shown in Fig. 138 (a): he turns his head to his right and a little backward,

the right ear is farther back and lower than the left, the trunk and upper extremities adopt a similar position, and the whole body leans forward. If the compensation method is used, the standing patient, as inactive as possible, should be positioned tilted about  $40^\circ$  or more to his left and backward in order to give him the sensation that his body is upright and in a normal frontal position, as shown in Fig. 138 (b).



**Figure 138.** Tests for body orientation disorder in subject M. (a) Patient M under maximum facilitation imitates the posture felt in the inactive standing state. He tilts objectively  $40^\circ$  to the right, with some backward rotation. (b) Subject M, in an almost inactive state, is objectively tilted  $40^\circ$  to his left to obtain the sensation of verticality of the body, thus compensating the subjective deviation to his right.

As already stated, the origin of the tilts and turns felt by the patient is the disorder of inverted tactile orientation. The perceived degree of inversion is related to the perception of the body, and below a certain limit, this perception fades easily and prevents to feel a large tilt. Thus, a moderate tilt and rotation correspond to an inversion following a spiral that is partially developed here. It can be said that the sensation of deviation of the position of the body reflects the state of deep sensitivity and of other functions involved in the formation of the body schema. Spatial values are modified by shrinkage and inversion, due to asynchrony. Thus, proprioceptive excitation (*via* joints, muscular tension, etc.) corresponding to the static attitude of the body results in incomplete spatial recruitment, leading to an inversion of the body and a reduction of its size. The less the facilitation acts, the more pronounced these effects are.

As for the *sense of rotation* in the inversion, it is always to his right because the rotation concerns the body as a whole. It is like in the inversion tests of rectilinear stimuli on the skin in the midline of the body.

The issue of body posture deviation does not end here and we shall return to it further on under other also novel aspects.

## **25 Complex processes of orientation in the sense of touch**

### **25.1 Perceived inversion and deviation during walking**

Having finished describing the deviation of the orientation of the body schema in a static situation, we must now consider the disorder kinetically, which can be studied in a very adequate and complete way when the subject is walking. Very remarkable phenomena of great importance appear which clearly illustrate the process of the so complex tactile inversion. The main interest lies in the phenomena that occur when the patient starts walking, i.e. during the first ten steps at the most, under certain conditions. These phenomena are very numerous due to the multiple factors involved in the process. Not all the steps actively taken by the subject are equally perceived and valued, although objectively they are the same. The first steps are perceived as if they were much shorter, much faster, and with the direction completely inverted with respect to the objective direction of the walk. Nothing remains fixed in the course of walking: the step size tends to increase, the speed to decrease and the reversed direction tends to straighten out by a process of latent addition in the nervous centers as the subject continues to walk. In parallel, the body schema varies greatly. The movement of the lower extremities is localized in different ways, the perceived body size gradually increases and the body schema tends to emerge, evolving also the orientation of the body in space. At first, the phenomena are very abnormal and diffuse, but gradually a more complex and more normal organization emerges. Thus, there is a very extensive development of tactile orientation. As intricate as this set of phenomena may seem, examination of the process reveals a fairly simple mechanism that finds a logical explanation in matters already studied for the most part in previous pages.

For a thorough analysis of the process we shall study in the following subsections: iterative excitation by successive steps, subjective space and time in walking, subjective trajectory, subjective localization of the movement in the body, size of the body schema, etc., and finally the felt orientation of the body during walking.

#### **25.1.1 Iterative excitation by successive steps**

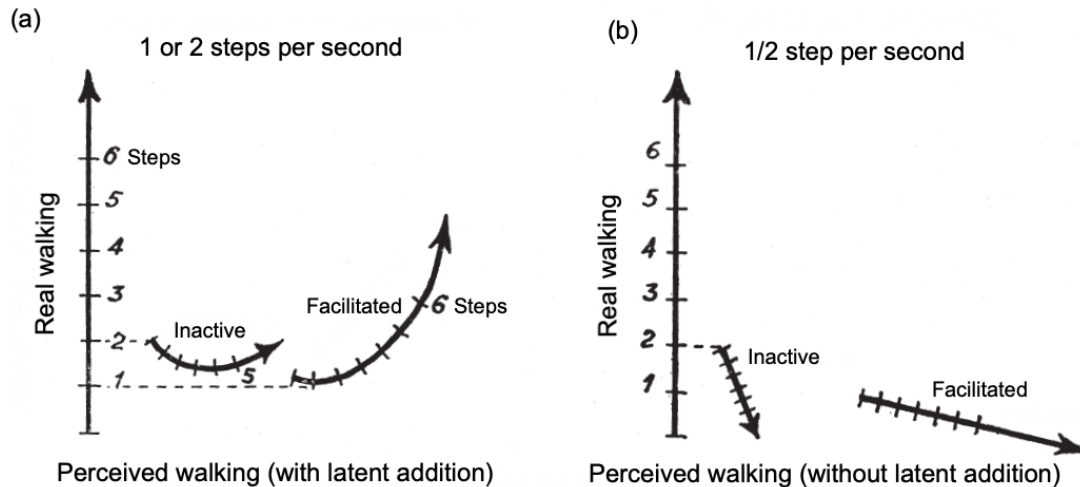
The process of walking is originated by the proprioceptive stimulus of the steps, so we must first analyze their characteristics and effects. Subject M, making no more effort than the usual effort to stand and walk (semi-inactive state for our purpose) takes a step or a

step and a half, i.e., advances one foot normally and then moves the other to meet the first, the length of the step being the usual one. Up to this point, the tactile sensation usually barely exceeds phase I, i.e., a diffuse pressure sensation without localization, or at most localized medially (phase II), but without arousing the sensation of movement. It is only after he has taken two and a half or three steps that he perceives signs of movement. Of the several steps taken, he only perceives the last one, and he senses it in the totally opposite direction, that is, backward, without being able to indicate whether there is some deviation to any side. This subjectively inverted step is felt as extremely fast and short (phase III), and the corresponding movement is localized only in the hips. It is worthwhile to compare the occurrence of the different phases here with the data in Table 32. The body schema presents certain deviations and reduction in size, which will be study further on. Continuing now with the process of walking, the patient is able to perceive two or three steps after four or five steps. After six or seven steps, he feels four steps as follows: the first one very short and totally backward, the next two also backward but somewhat tilted to the right and slower and longer than the first, and finally, the fourth step is felt as transversal, or something similar more or less towards the right. This happens successively, so that as the number of steps during uninterrupted walking increases, the subjective direction tends to straighten out and resemble the real gait (phase IV). At the same time, the steps are felt to be longer and slower than at the beginning. Since the initial steps are not perceived at all and the following steps are felt to be very short, the subjective path traveled must necessarily be much shorter than the real one (see below). If, in addition, the subject walks with his eyes closed, he will have no other reference for the path traveled than his steps reduced in length.

Performing the same test in a state of maximum facilitation, i.e., walking with the body under strong generalized muscular tension, results in fewer ignored steps, less inversion, and greater subjective distance traveled. Walking at the same speed as in the previous case, it may sometimes happen that he ignores the first step taken. This occurs when the step is short or weak, or when the facilitation by muscular effort is moderate. But in general, the first step is already perceived and is felt less inverted than in the previous inactive state. It is perceived as going backwards and to the right, i. e., rotated about  $110^\circ$  approximately. The second step may already be at  $90^\circ$ , and the successive steps quickly tend to be felt more towards the front. After seven or eight steps, the step is perceived in a direction very close to the one really executed. Therefore, the evolution of the subjective walking is quite different from that in the quasi-inactive state.

These two tests of walking, in the inactive state and under facilitation, correspond to a real rate of one step per second or slightly less. For example, the subject takes six steps in just over 4 seconds, or five, being steps of normal length, of about 40-50 cm (the subject is a tall individual), resulting in a rather slow walking pace. In such conditions we have seen that the sensation that the subject has tends to change continuously during the course of the gait, intervening, as already said, an action of accumulation (latent addition) in the nervous centers, due to the excitations involved in the successive steps. Thus, it results in a *recruitment of the direction of walking as a function of the number of steps*. This iterative character is demonstrated by studying the two main factors involved in this type

of stimulation: the energy of each step, and the iterative aspect related to the number and frequency of steps.



**Figure 139.** Diagram on the subjective perception of walking according to step frequency in patient M. (a) With an average of 1 to 2 steps per second, a latent addition effect and thus sensory recruitment is obtained. The steps vary subjectively in a continuous manner in direction and length. In the inactive state, recruitment is slower than under facilitation. In the former, only the second step is perceived, the direction felt is more inverted for the whole path, the length of which is felt to be shorter as well as the time spent. The subjective spatiotemporal shortening in the inactive state is one-third of the real value, and almost half of that perceived under facilitation. The tick marks for the steps indicate the spatial shortening. (b) At a frequency of half a step per second (one step every two seconds) there is no addition effect, and since there is no sensory recruitment, the subjective perception of walking remains unchanged as regards direction and spatiotemporal value, which are much more altered than in the previous case. There may be some trace of latent addition, especially in the inactive state, as the perception of movement occurs in the second step.

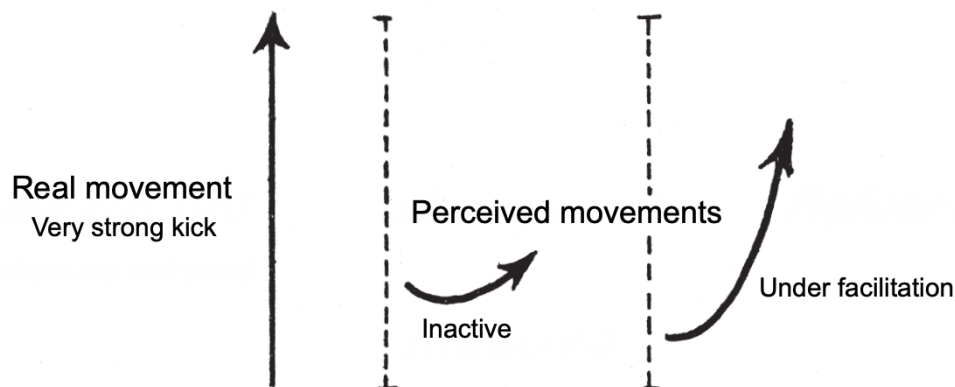
Note in the different cases the different subjective shortening of the step in relation to the degree of inversion, the difference between inactive state and under facilitation, and the different subjective trajectory with or without latent addition.

The action of the number of steps has already been seen. As for the frequency, if the subject walks very slowly, and there is a long time interval from one step to the next, it is not possible to achieve accumulation and the same result is obtained for both the first and the last step. Thus, the subjective direction of walking is approximately equally abnormal throughout the gait. Two different types of subjective gait can thus be obtained, of uniform direction if latent addition is not involved, and of variable direction if it is involved, as shown in Fig. 139. The latent addition tests described above correspond to a small frequency of about *one step per second*, since this was convenient for the initial analysis of the phenomena in subject M. But this frequency is too low to obtain full recruitment of the direction of walking. Much better recruitment is achieved with a double frequency of *two steps per second*, as will be seen later. Instead, to eliminate the central effect of accumulation (latent addition) of the steps, the subject, in an almost inactive state, should walk rather slowly. Tests performed on patient M show that a frequency of



one step in one and a half seconds results in a perception of continuously inverted walking direction, with the steps being felt as very short and fast. In the state under facilitation by muscular effort, the nervous reaction being more rapid and therefore the residue shorter, the frequency can be somewhat greater without giving rise to latent addition. Excluded the central action of latent addition, the recruitment obviously disappears and the subjective perception does not change no matter how long the walking lasts, or the change is so slow as to be negligible.

These step frequency values for the latent addition limit vary greatly in relation to the other fundamental factor involved in the process, the *step energy*, which depends on the length of the step and its strength. Thus, in the test under facilitation, no latent addition is obtained when the subject, at a rate of one step per second, walks with a very small step length (almost half of normal) and with very little energy (slow and not vigorous). In this case, the subject does not perceive a straightening of the gait and has to be led by the hand because he does not know how to walk so slowly on his own in this situation. On the contrary, if the subject under facilitation takes a single high-energy step, with great strength and force of the lower extremity, this single step reaches a considerable functional level, and the subjective direction deviates only about  $60^\circ$  from the real direction. Even less subjective deviation is obtained if the subject kicks hard in the air with as much force as possible. In this case he can achieve a practically normal direction. However, in the inactive or almost inactive state, no matter how strong the kick is, he only achieves  $90^\circ$  of subjective deviation, and there is probably a brief inverted path at the beginning of feeling movement. Figure 140 shows these results.



**Figure 140.** Subjective direction of a very energetic single step (with kick in the air) of subject M. Note that in the inactive state, the subject cannot straighten the subjective direction beyond  $90^\circ$ , whereas under facilitation, the subjective deviation is very small. Note also the different subjective path in the inactive state and in the state under facilitation.

In the case of the strong kick, it turns out that with a single step the same effect is obtained as with many. It is clear that in the kick, the joint movement comes into play in all its range, and also abruptly, which means a great excitation which results in a high sensory level, within the current asynchrony. In contrast, during ordinary walking, joint

movement is quite moderate in range and velocity, so recruitment has to be developed slowly and successively through iteration.

In short, the disorder of direction during walking is consistent with what has been stated above (Sec. 24.3) about perception and direction in deep (joint) movement, whether passive or active, and for both repeated stimulation and single strong excitation. A single step must be very long and vigorous to awaken the sensation of movement, and above all, to overcome the inversion of direction. This is partially achieved in the inactive state, and much better under facilitation. However, under ordinary walking conditions, the joint excitations are too weak, and only the addition of steps produces a re-inversion within certain limits of step frequency. Thus, very slow walking (one step every one and a half or two seconds) always provides the same perceived direction, since the residue of each step is dissipated before the next one arrives. Instead, fast walking (two steps per second) allows the residue of the steps in the nervous centers to add up successively, and thus the perceived direction (sensory level) varies with each step. The interval between steps to produce summation is related to the level of excitation, and therefore should be somewhat shorter under facilitation than in the inactive state. All these circumstances on the subjective perception of walking are illustrated in Figs. 130 and 140.

### **25.1.2 Perceived space and time during walking**

The space and time of the perceived steps is changing during walking (due to latent addition) in parallel to the change occurring in the subjective direction. That is to say, the first perceived steps are very short and fast compared to their real value; then, as the direction of the gait is being straightened and re-inverted, the steps gradually resemble their real value, although without reaching it as long as the felt direction is not completely normal. These manifestations of space and time in the steps correspond to the intrinsic alteration of the perception of movement (path and speed) already studied in several chapters for both touch and vision. This alteration depends directly on the elementary disorder of nervous excitability in the nervous centers. Such manifestations are the result of a functional dynamic reduction, reflected here in a *spatiotemporal reduction*, i.e., a contraction of perceived space and time, which results in a reduction of the path and an overestimation of speed, giving rise to a sensation of walking with small and fast steps.

Due to this intrinsic modification of the perceived movement, it turns out that the path perceived by the subject after six steps is greatly reduced in relation to the real path. However, this remarkable difference between what is felt and what is executed tends to be initially disregarded by the subject, and he only recognizes it by means of appropriate tests.

Of course, the subjective reduction of the path is more clearly noticeable in the inactive state, but the subject first believes that the path has been reduced by only two-thirds of the real path. However, it is understandable that due to the steps lost initially (if he walks at one step per second he ignores the first two steps and if he walks at two steps per second he ignores the initial one) and the enormous contraction that most of the steps

undergo due to the strongly inverted direction (Fig. 139), the perceived decrease in the path should be much greater, as is indeed the case. What happens is that very abnormal sensations, being more diffuse, brief and of less frequent occurrence for the subject (M), tend to be excluded from perception and easily not taken into consideration. This happens both for strong inversion and for the shortening phenomena we are now dealing with.

If the patient in an almost inactive state walks through a room six meters long and crosses it by taking ten steps, he later believes that the perceived shortening of the path is not much, especially if he has kept his eyes open. But if he walks this path with his eyes closed, alone or led by the hand, and is asked to pay attention to his sensation of the length of the path, he will easily realize how short the felt path is. Since with closed eyes he has no information other than his altered touch, he has to stick only to that information. It is difficult to determine with certain accuracy the reduction of the path, and it can only be known in an approximate and indirect way: the subject, after having walked the six meters in ten steps with his eyes closed, must indicate on the ground, opening his eyes, what distance he thinks he has covered. It happens then that the six meters can be reduced to 1.80 meters, that is, less than one third. This value is by no means small and may even be too large if the following is taken into account: at the end of the run the perceived direction is about  $80^\circ$ , therefore the steps must have greatly reduced their perceived size; moreover, as two or three steps are felt strongly inverted, the perceived shortening must be even greater, apart from one or two first steps that are lost. It should be noted that total or partially inverted motion corresponds to motion perception only in hints, so that a normal 40 cm step is perhaps reduced to about 4 cm. The same test under facilitation results in a much smaller subjective reduction because the steps are not lost, and they are perceived to be longer as there is less inversion. In this case, the subject indicates on the ground a distance of about 4 or 5 meters, but it can probably also be excessive, although at the end of the path he feels a deviation of only about  $20^\circ$ .

It is understandable, after all that has been said above about iteration, that the step frequency has a significant influence on the spatial reduction. If the subject walks very slowly, there is no addition and the reduction is maximum; thus, in the inactive state, six meters should feel like half a meter. Figure 139 attempts to illustrate the spatial reduction roughly, as well as the close relationship between the degree of inversion and the spatial reduction of the steps. (These steps in the non-addition case are perhaps represented too large).

As for the temporal contraction (increase of the perceived speed in the sensory process), it is related to the spatial reduction, and is manifested in a significant increase in the perceived frequency of the steps. As the perceived gait changes continuously when there is a latent addition effect, the subjective rate cannot be uniform but progressively decreasing, without reaching the objective step frequency which is always lower than the subjective one. This is the same as in other rhythmic stimulation tests, for example in vibratory stimulation with tuning forks. For both vibration and footsteps, at the beginning of the perception, the subjective frequency is so high that what is perceived is a fusion (phases I and II). Subsequently, the perception becomes intermittent with a very short time interval that tends to enlarge progressively throughout the process. In the case of walking, if the first step is lost, it is due to the fusion effect that prevents the appreciation

of the time interval, indefinitely reduced when the development of the sensory process of walking begins.

The sensation of time contraction would mean a shorter time felt by the subject in walking the aforementioned path. The real path is six meters, traveled in ten steps, which at two steps per second results in about five meters in five seconds, i.e., a rate of one meter per second. Assuming that the contraction of time is of the same order as that of space, it follows that if 5 meters of real path are subjectively reduced to 1.80 meters (approximately one third), the perceived time will offer a similar reduction, i.e., five real seconds would be reduced to 1.80 subjective seconds. It could then be said that the speed has not changed, since space and time are altered equally, but it is clear that the subjective frequency or rhythm of the steps will have tripled, since the same number of steps will be felt in one third of the real time. Similarly, the corresponding reduction under facilitation would be obtained. Thus, the spatial contraction of the steps indicated in the different figures also represents a temporal contraction.

### 25.1.3 Perceived trajectory

We know that the *subjective trajectory* of walking depends on the conditions for latent addition of steps and on the spatiotemporal dimension, which determine the corresponding development of the process. In the following, we shall take a closer look at this development by paying attention to both the immediate manifestations of the subjective trajectory and the functional quantitative relations. It is also important to demonstrate the existence of phenomena of this type in other sensory systems, for example in the visual system, which means that they have a general character.

When patient M is walking and there is progressive recruitment according to the number of steps, the deviation felt by the subject is always to his right (Figs. 139 and 140), as is the rule in this individual for stimuli affecting the whole body schema. Regarding the course of the trajectory, as the sensation of step direction changes from an almost complete inversion to a very pronounced straightening while also changing the sensation of size, the perceived trajectory follows a more or less open spiral depending on the speed of recruitment, as we have already seen in other types of tests.

By repeating the tests and studying the meaning of the different factors, keeping the subject attentive and properly informed about the nature of the tests, it is possible to obtain valuable data despite the drawbacks and inaccuracies of the method. In the experiment, subject M walks in a straight line forward with a frequency of about two steps per second, a frequency almost double that in previous tests, thus resulting in an efficient latent addition, well suited for our current objective. The step length is 40-50 cm, i.e. a common length. Naturally, it is necessary to maintain maximum regularity in all these factors during the test. The results are shown in Tables 35 and 36.

**Table 35.** Walking perceived by subject M in an inactive state, according to number of steps. Frequency of 2 steps per second. Step length 40-50 cm. (See Figs. 141 and 142).

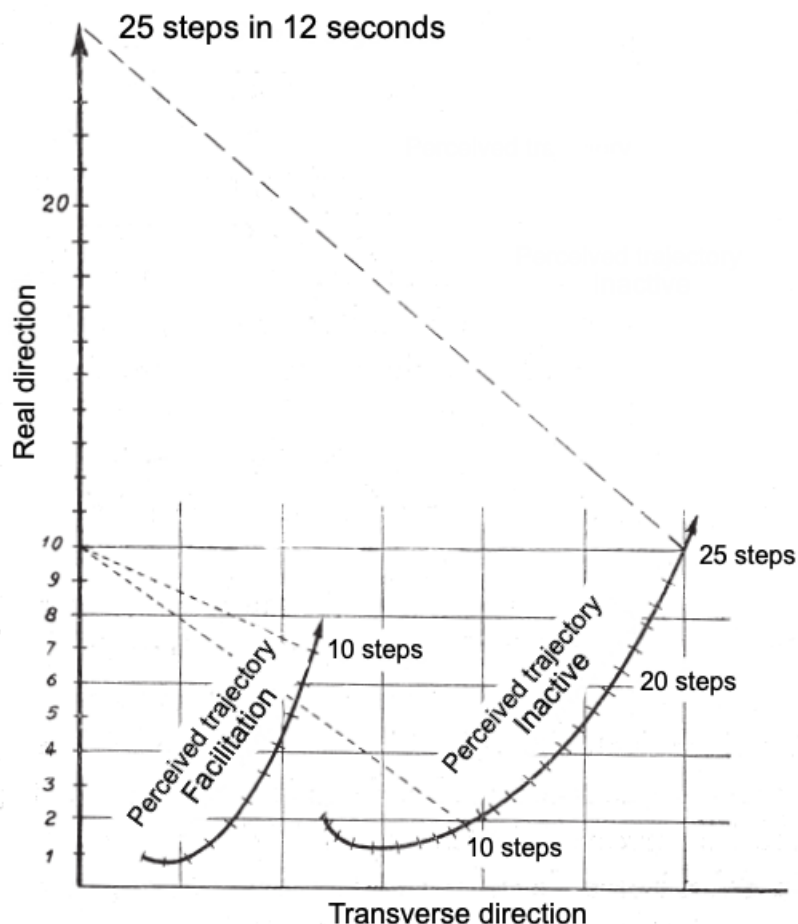
<i>No. of steps</i>	<i>Perceived direction, angle rotated</i>	<i>Movement localization</i>
1		Not perceived
2	Backward, 160°?	Signs in contralateral hip
3	Idem	Idem
4	Backward and to the right, 110°?	Idem homolateral
6	To the right, 90°	Idem
8	Weakly forward, 80°	Idem
12	Almost oblique, 70°?	Idem and signs in the knees
15	Oblique, 45°	Idem
20	35°?	Well in the knees
25	Almost forward, 20°?	Idem and signs in the feet

**Table 36.** Walking perceived by subject M under facilitation by strong muscular effort, according to number of steps. Frequency of 2 steps per second. Step length 40-50 cm. (See Figs. 141 and 142).

<i>No. of steps</i>	<i>Perceived direction, angle rotated</i>	<i>Movement localization</i>
1	Somewhat backward, 110°	Homolateral hip
2	Almost transverse, 80°	Idem and signs in the knees
3	Almost oblique, 50°	Idem
4	35°?	Well in the knees
5	30°?	Idem
6	Almost straightened, 20°?	Strong in the knees, signs in the feet
10	Very little to straighten out, 10°?	Hips, knees and well in the feet

Tables 35 and 36 show, for each number of steps, the corresponding sensory level expressed by the perceived direction of the steps and also by the localization of the movement. This localization initially undergoes strong proximal deviation, and after more steps it is more distal (knees and feet). In this way, the growth in the size of the body schema is also evidenced. Once the subjective gait deviation is known through global determinations, partial tests are performed to find out which subjective deviation corresponds to each step, in order to know more precisely the type of trajectory. After the subject has taken one or more steps, he must indicate the degree of deviation he feels at the end of this partial walk. Due to the iteration effect, it is not possible to resort to the compensation method because it would be too cumbersome to divert the real gait in various ways. Therefore, there is no choice but to stick to the verbal information of the subject. The disorder in step direction causes steps taken forward to be felt as deviating

in various ways, as if the step were taken with a certain lateral, transverse, backward deviation, etc., and the patient should indicate this type of deviation. Some directions can be indicated with some precision, for example in the case of a  $90^\circ$  deviation in which case the subject feels as if he has taken a step in a completely transverse direction. For other cases, the patient must be guided by the degree of backward or forward obliquity, and only approximate determinations can be obtained (indicated in the tables with a question mark) but very useful and sufficient for our purpose in the absence of a better method.

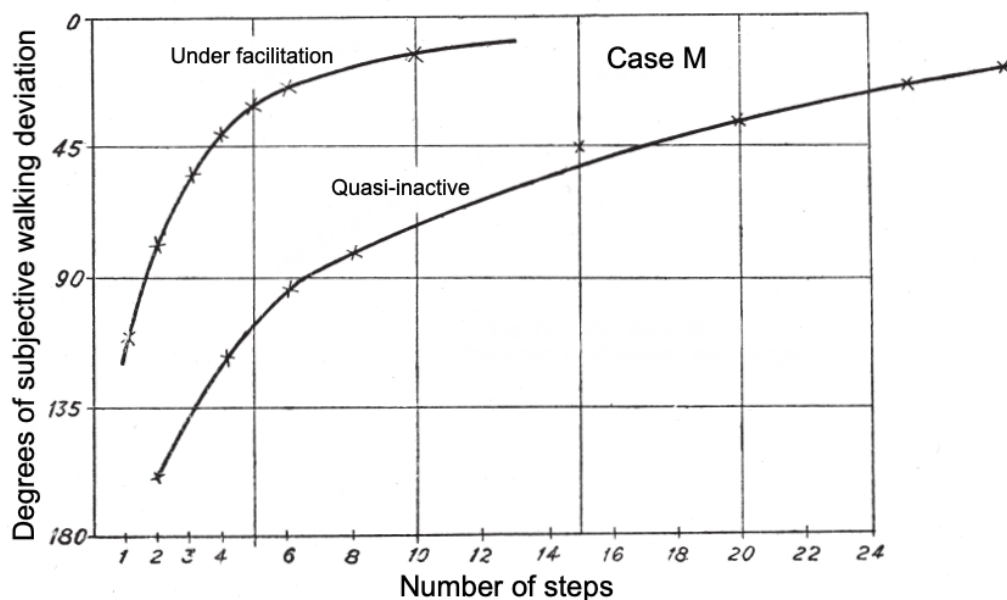


**Figure 141.** Diagram of the trajectory perceived by subject M as a function of the number of steps. Frequency of two steps per second, step length 40 to 50 cm. Note the spiral development (very open spiral branch), the perceived shortening of the trajectory, the suppression of the initial steps, etc.; for the inactive state and for the state under facilitation. Compare with the curves in Fig. 142.

Using the data from the tables and taking into account the corresponding spatial contraction (more pronounced at high degrees of inversion), the trajectories shown in Fig. 141 are determined in an approximate way. Direction, space and time of the steps vary continuously, and this is perceived by the subject directly. In Fig. 141, the perceived direction is indicated by the tilt of the line corresponding to each step, and the perceived space and time is indicated by the length of that line, very short at the beginning and longer at the end, tending to resemble the real size. The trajectory tends to take the form

of a spiral, more pronounced in the inactive state, to which corresponds a slower variation throughout the process. Comparing the results of the inactive state with that of facilitation, it is observed that in the latter state, the perceived walking after ten steps is almost normal (see Table 36 and Fig. 141). In contrast, in the inactive state and with the same number of steps, the patient feels that he has traveled half the path as with facilitation, and about one third of the real path. In the inactive state, the tenth step taken is felt with a deviation around 90°. The disorder is more evident in the inactive state, and the subject needs to take 20-25 steps to achieve an effect similar to that obtained with ten steps under facilitation. Therefore, the difference is two times greater or more.

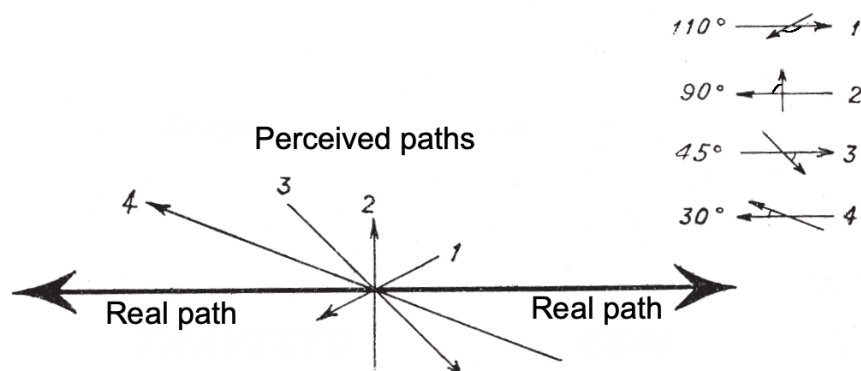
These important defects have been extracted from the subject by means of much patience. The subject never noticed them spontaneously since in ordinary life they are easily ignored, either by the corrective effect of facilitation by more or less unconscious muscular effort, or because they are only very noticeable disorders at the beginning of walking and of short duration. Furthermore, there is a natural tendency to exclude abnormal phenomena, and even more so if they are incongruent with voluntary activity, as in the case of walking.



**Figure 142.** Recruitment curves of the sensed direction of walking as a function of the number of steps, according to the data in Tables 35 and 36. Note the different slope and position of the curves for the quasi-inactive state and the state under facilitation. The subject walks objectively in a straight line, with equal steps of ordinary length (40-50 cm) and a frequency of two steps per second.

In the trajectories studied, both the variation of the direction and the variation of the length of the steps are involved, depending on the number of steps. From the data in Tables 35 and 36 we can obtain functional relationships between the direction of the steps and their number, which are the iterative recruitment curves shown in Fig. 142. This type of recruitment has already been analyzed in different experiments on touch, as shown in Fig. 114 (localization recruitment as a function of the number of stimuli at a given

location on the skin). All curves of sensory level recruitment are of the same type, regardless of the type of excitation, whether it is a single increasing stimulus (Fig. 113), a constant stimulus iteration or an increasing facilitation by increasing muscular effort. They correspond more or less to logarithmic curves of the type of Fechner's law of sensory variation. These curves in Fig. 142 can be compared with the data from the test on the perceived orientation of a rectilinear stimulus on the back of the hand as a function of pressure (Fig. 127). In the curves of Fig 142, the iterative stimulation conditions are the same for the quasi-inactive state and for the state under facilitation. As asynchrony is lower under facilitation, recruitment in that state is much higher, as can be seen from the position and slope of the curves.



**Figure 143.** Visually perceived directions by subject M of the oscillatory motion of a white disc of 1 cm in diameter at the end of the pendulum of a mechanical metronome. Note the increase of the perceived path when the perceived direction is straightened. To simplify the representation, this diagram corresponds to the state under facilitation according to Table 38. For the inactive state, a similar diagram can also be obtained but with other luminous stimulation values. See text for experimental conditions.

This iterative recruitment of spatial orientation in the sense of touch is not an isolated event in the set of central syndrome disorders. We also find it in vision, as it is to be expected, since touch and vision are equal systems in structure and excitability defect. To observe the phenomenon in vision, we place a white disc of 1 cm in diameter at the end of the pendulum of a mechanical metronome that oscillates at a constant number of beats per minute. There is beat for which the subject reaches the phase in which he begins to perceive motion. From that beat, each consecutive corresponds to a new perceived direction of motion, the direction evolving in the same way as that of the steps when walking. That is to say, not only the perceived direction changes but also the amplitude of the oscillation and the speed, apart from a chromatic change in this case due to chromatopsia of the white color of the small disc. The experimental technique is analogous to that of walking, although with some particularities that make the visual test more difficult. At the beginning of the perception process, the perceived motion is strongly inverted, i.e. almost horizontal but in the opposite sense to the real motion, and it can be indicated by the patient since the real oscillation is not very fast. Subsequently, the motion tends to be perceived as vertical (rotated 90°), to become again perceived as



horizontal in the almost correct sense as the recruitment increases, as shown in Fig. 143. The experiment is conveniently fragmented into several partial trials in order for the subject to indicate as accurately as possible, after a given beat, the perceived direction. The experimental conditions are: metronome at 25 cm distance, white disc of 1 cm in diameter at the end of the pendulum illuminated with medium natural light, displacement of the small disc about 10-12 cm between each beat, rate of 1 beat per second, and vision only with the right eye of patient M. The mean values of several tests are shown in Tables 37 and 38.

**Table 37.** Visually perceived direction by subject M, in the inactive state, of the motion of a white disc of 1 cm in diameter at the end of the pendulum of a mechanical metronome, depending on the number of beats elapsed. Experimental conditions in preceding text.

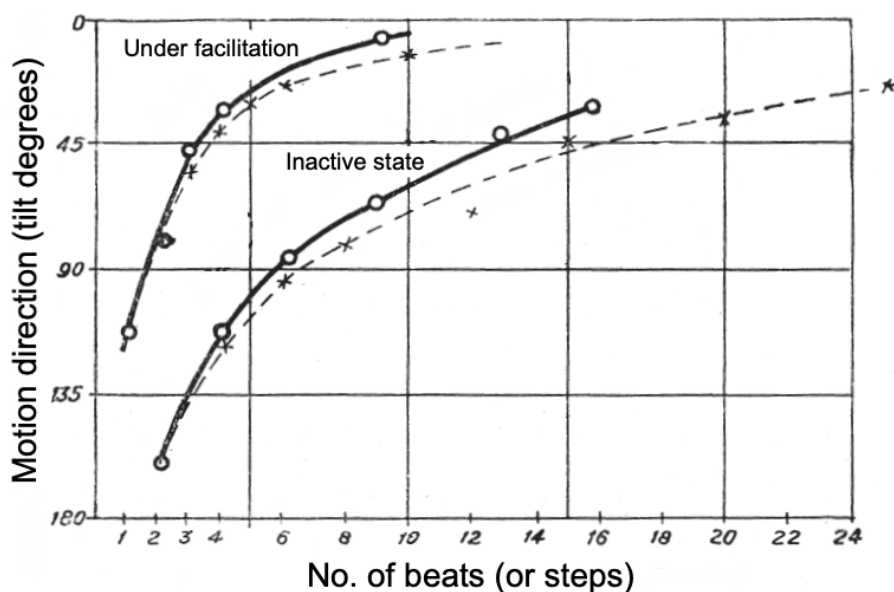
<i>No. of beats</i>	<i>Perceived direction, color. (Sensory level)</i>
1	Dim motionless luminosity, achromatic sensation
2	Very short inverted motion, dark green
3	Idem but more oblique
4	Idem and still more oblique 120°?
5	Close to vertical 90°? pale green
6	80°? scarce white
8	60°?
12	Rather horizontal, not inverted, more white than green
14	Idem but more horizontal

**Table 38.** Same as in Table 37 but subject M is under maximum facilitation by means of maximum muscular effort.

<i>No. of beats</i>	<i>Perceived direction, color. (Sensory level)</i>
1	Inverted oblique motion 100-120°? pale green with hints of white
2	Close to vertical 80-90°
3	Close to oblique 45°?
4	Approaching horizontal but not inverted
6	Idem but even more horizontal
7, 8	Idem, completely white

As for the color change indicated in Tables 37 and 38, dependent on the degree of inversion and contraction of the path perceived by subject M (especially in the inactive state), it has already been studied in the part concerning visual functions (Sec. 11.2 in Vol. 1). As for the simultaneous gradual variation of the different factors, it may be useful to compare the data in Tables 37 and 38 with those in Table 14 in Vol. 1 on visually perceived orientation of a vertical white arrow pointing upward. Much more relevant

now is to compare Tables 35 and 36 on the perceived direction of walking depending on the number of steps, with Tables 37 and 38 on the visually perceived direction of motion depending on the number of metronome beats. We then find that such iterative recruitment of the direction of motion is possible in both vision and touch, which allows us to realize that this process has a very general significance. Furthermore, it is shown that, by properly adjusting the experimental conditions in vision and touch, the development of sensory recruitment is, in quantitative terms, completely identical in both sensory systems. This is expressed by means of the curves in Fig. 144, where it can be seen that the tactile and visual curves are very similar. The visual curves were made several months after the tactile curves in 1946, since having first made the recruitment curve for step direction, it was thought much later that a similar effect could be determined in vision. In both the metronome beats method and the step method certain imperfections are unavoidable, and these methods cannot compete in accuracy with the compensation method used in the curves on the image orientation of a vertical arrow. However, we have already seen in other tactile tests how it is possible to achieve valuable results despite the shortcomings of the methods.



**Figure 144.** Iterative recruitment of the direction of motion perceived by subject M in the inactive state and in the state of maximum facilitation. Solid curves: visually perceived direction of motion of a white disc of 1 cm in diameter at the end of the pendulum of a mechanical metronome, as a function of the number of beats (see Tables 37 and 38). Metronome at 25 cm distance, white disc illuminated with medium natural light, displacement of the small disc about 10-12 cm between each beat, rate of 1 beat per second, and vision only with subject M's right eye. Dashed curves: perceived direction of walking as a function of the number of steps (Fig. 143); see data and experimental conditions in Tables 35 and 36. Note the high similarity between visual and tactile curves.

#### 25.1.4 Localization of movement in the body, size of body schema, displacement on the ground

During the initiation of walking, the localization of the movement of the lower extremities varies significantly due to the proximal deviation of stimulus localization, resulting in a change in the size of the body schema. The first perceived steps only elicit a sensation of movement around the most proximal part of the limbs, i.e. the hips. Thus, we can only refer to perceived steps in a broad sense. At the same time, the perception of the body is diffuse, rather soft and poorly delimited, difficult to describe accurately by the subject. After several steps, the subject tends to feel the movement in the knees and much later in the feet, the previous localizations being preserved.

Tables 34 and 35 provide details on this *evolution of localization* in tests of walking at a rate of two steps per second. In the inactive state, after two steps the subject feels movement in the hips, after twelve steps the sensation of movement in the knees is initiated, and after 24 steps, signs of movement in the feet are felt. Instead, under maximum facilitation, with a single step the movement is already felt in the hips, with two steps, it is felt a little in the knees, and after six or seven it is felt in the feet. When the localization of the movement begins to be felt in the feet, it is felt only as small indications, as a movement of much smaller magnitude than the actual movement of the foot, and deviated by about  $20^\circ$  (see Tables 35 and 36). With more steps, the movement felt in the feet becomes more intense up to a certain limit.

The decrease in the proximal deviation effect, when the localization of the movement is more distally, is equivalent to an *increase in the size of the body schema*, so that the body is felt to grow in the course of walking. The shortening of the body schema with the distal exclusion of the lower parts of the limbs does not necessarily mean that the subject feels supported on the leg stumps. Here the support is indifferent, it is simply that the body does not feel complete. The consequences of this incomplete and diffuse sensation are addressed in the following experience.

A complementary test, very significant for the new phenomena it reveals, consists of the patient making the movement of taking steps but without moving from the site. At a rate of two "steps" per second, the subject, in a quasi-inactive state and with eyes closed, takes 12 seconds (25-27 "steps") to realize that he is not moving from the site. Under facilitation by means of strong muscular effort, the same result is achieved with 10 "steps" in 4.5 seconds. This result is achieved when in both cases the localization of the movement is quite evident in the feet, and the body schema is well developed. At that point, the deviation of the perceived direction of the step is about  $20^\circ$  or less. During the previous steps, the subject is convinced that he is walking displacing himself over the ground, and not that he is in the same place (as is actually the case). This result shows how much is destroyed in the perception of walking. It is likely that for the subject to distinguish the type of walking, with or without translation movement, it is not so important the localization of the movement in the feet, but rather that the body schema develops by achieving sufficient internal coherence to establish references between the different movements of the limbs and trunk that occur when there is translation movement and when there is not. It is therefore a matter of reaching a certain level of organization

that allows sufficient discrimination. The spontaneous *sensory illusion* of believing that there is displacement, together with the corresponding tendency to automatically exclude the defect, is typical of diffuse and unstable sensory activity.

From all that has been said, it follows that ordinary walking in a more or less inactive state remains for a certain time at a very primitive level from the point of view of the subject's own perception. In a strict sense, walking steps are only perceived after some time, when the sensory level has increased considerably.

### **25.1.5 Subjective body orientation during walking**

The different subjective size of the body is accompanied by a different *subjective orientation of the body in space*, as occurs with the steps. The subjective position of the body undergoes certain turns that we have already studied when dealing with the orientation of the body schema statically (Sec. 24.4). The body seems to rotate in the three directions of space, as follows: it rotates on itself to the right and backward and also tilts to the right side and forward. These sensations are not easy to specify by the subject, especially in the first sensory phases. It should be noted that not all phenomena manifested at a given moment are equally evident. We must therefore stick to simple indications of the patient.

The aforementioned body deviations tend to become evident when the body schema begins to emerge. When subject M is in a quasi-inactive state and perceives the direction of walking strongly inverted, he cannot say whether it is the whole body that is moving in the opposite direction to the real one. Rather, it seems that the inversion is restricted to the lower limbs, which are as if disconnected, without reference to the body schema. Recall the test of moving the feet without translational movement, in which the subject was unaware that he was actually at the same place. However, although the sensations about his own body are extremely diffuse at the beginning of walking, as soon as the subject feels his body more defined, he clearly feels that the gait trajectory does not take place on a horizontal plane, but as if going down a slope. When the body is felt more clearly, the different deviations are better felt. The subject feels his body tilted forward and to the right, and somewhat rotated on itself to the right, such deviations corresponding to the felt deviations of the steps between 80° and 40°.

It is reasonable to interpret these tilts and rotations as residual phenomena of body schema inversion, an inversion that is not possible to obtain more pronounced due to the disintegration that the body schema undergoes when the sensory level is very low (see Sec. 24.4). It could be assumed that the body schema tends to make a spiral turn in space, following a helical trajectory. Given the tendency to inversion, it is conceivable that there is a moment in which, being the tilt of the body schema very strong, the slope referred to above would be very steep. However, it has been repeatedly found that the subject does not perceive such anomalous orientations, which seems logical since he would lack any reference to the body schema at such low sensory level. Thus, it is understandable that the subject always decisively rejects the question we ask him if at the beginning of walking it seems to him that he is walking almost upside down, or at least that the slope he perceives is so steep that it seems to be going down a vertical wall with his body tilted

90°. None of this can be perceived because the body schema does not subsist at such a low sensory level, and in order to study the issue it is necessary to stick to the data of the more attenuated disorder. It is also possible that difficulties may arise for the subject to localize his own body very inverted in space, or that mechanisms of exclusion or attenuation of the defect may intervene. We have already seen in the test of moving the feet as if walking but without moving from the site, the deficit that arises to appreciate the lack of translational movement. Therefore, there is a limit to how far we can go in analyzing the process, and at present it has not been possible to make any further progress on this issue.

However, we have found it possible to study to some extent the type of inclination felt by subject M. He is asked to walk in an almost inactive state on a board inclined about 45° to the horizontal. If he walks ascending he tends to perceive a horizontal path. But if he walks downward he feels that there is a large almost vertical drop. These sensations are greatly diminished under facilitation, and also vary with the length of the path. A similar situation occurs when going up or down a staircase. If he goes up the staircase in an inactive state, he senses a lesser slope than the actual slope and that the path tends to be horizontal. But if he goes down the stairs, he feels a much steeper slope than the actual one, and protects himself by holding on to the handrail. Even with his eyes open, he cannot descend a single step without helping himself with the wall or grasping the stair handrail. In contrast, under facilitation by maximum muscular effort he can descend more easily.

In general, in the conditions of ordinary life (medium facilitation), going up or down a staircase involves considerable difficulty for the subject, since the tactile proprioceptive information is deficient or unclear, the range of motion is felt to be reduced and there is imprecision about the displacement of the own body, apart from the deviation of the direction of walking, incongruities, etc. Under intense facilitation, these difficulties usually appear only at the beginning of walking. The difficulties are aggravated if the staircase is carpeted, because the steps are cushioned and the steps are less visible. On stairs with turns, without corners, with steps with uneven bottoms, the subject can easily fall backwards. For these reasons, it is very common to see him, outside the tests, descend a staircase with great caution and without ever letting go of the handrail. This could be interpreted as a visual difficulty, but in this case the major defect corresponds rather to touch, and here most likely to the subjective increase of the slope. Despite the many significant sensory impairments of patient M, his behavior in ordinary life at first glance is entirely normal, with the important exception of the case of stairs where he is at risk for many mishaps.

After dealing with numerous issues related to the disorder of perceived direction (orientation) during walking, all of them simple consequences of phenomena already studied, the most relevant characteristics are summarized below. We have first examined the perceived trajectory during walking and then the changes in the body schema during this process, aspects that evolve in parallel. Thus, a certain felt size of the steps is accompanied by a certain felt size of the body, and likewise, a felt direction of walking is accompanied by an orientation of the body schema in space. We have only referred here to two very relevant aspects: *size and direction (orientation)*. Synthesizing further, it can

be stated that the whole process follows a *spiral development in space* (helical) due to the different reduction of both the body schema and the steps taken, and to the corresponding inversion in the steps and in the body.

## 25.2 Induced objective postural deviation

All the orientation disorders described so far are strictly sensory in nature, and the phenomena on the body schema, both static and during walking, are subjective and can only be studied indirectly through the sensations of the patient. Something quite different occurs in the phenomena we are now going to describe. These are motor deviations of the body, i. e., alterations in the postural tone that result in changes in the attitude of the body, either in its entirety or partially in its segments, depending on the conditions. These phenomena are thus revealed in an objective and direct way. Such postural motor deviations, of a spontaneous nature, are well-known phenomena in clinical neurology. These are: head tilts, deviation of the whole body (Romberg's sign), deviation of the index finger in finger-pointing tests, and deviation of the whole body during walking.

In all these motor phenomena the deviation always consists of a rightward rotation, as in cases of subjective deviation. Facilitation also has a considerable effect in correcting postural motor deviation. Thus, these phenomena could be related to the sensory disorder of orientation in touch. Such a disorder would alter in a primary way the sensomotility (Exner 1894), and would induce secondarily a motor deviation.

First, the motor phenomena will be described, and then their interpretation will be addressed.

### 25.2.1 Postural deviation of the head

The head of patient M is usually spontaneously tilted to the right and rotated a little around its axis. In this test, patient M is in an inactive state (or semi-inactive, as corresponds to his usual state) and his head is passively moved in various directions, with the neck relaxed, leaving the head free in any position. If he is then asked to put his head in a natural position, he ends up putting it in the position indicated at the beginning of the test. If at that point he is asked to apply facilitation by means of intense generalized muscular effort, the deviation is corrected immediately by straightening the head. The objective deviation of the head in the inactive state is about 30°. If there is ever any objective head deviation with the mentioned facilitation, it will be barely perceptible. Sometimes a leftward deviation can be observed, also small, probably to counteract the small subjective sensory deviation felt even under facilitation. During the test, the subject is seated with eyes closed, although with eyes open the result hardly changes. It is very important to note that the subject is not asked to give indications about the position he perceives of his body, but is only asked to try to adopt the most natural or comfortable posture. Thus, an almost reflex function is allowed to act.

### 25.2.2 Body deviation. The Romberg test

If the subject is standing, in frontal position, with eyes closed, after a few seconds he tends to deviate with the whole trunk to his right and somewhat backwards, showing a positive Romberg's sign (Romberg 1846/1853), although moderately. The degree of deviation is measured by the displacement of the tip of the nose in the course of the test. After one minute, the deviation is 18 cm, the whole body gradually turning in the direction indicated above. Under facilitation by strong muscular effort, the deviation is corrected, and the tip of the nose is only two or three centimeters away from the normal position. If the facilitation persists, there is a real deviation to his left that counteracts the subjective deviation disorder. Performing the test applying facilitation from the beginning, the deviation obtained is 4-5 cm in 18 seconds, i.e., about one third of the value for the inactive state, in about one third of the time. Under facilitation he also deviates a little backwards. In other tests in an inactive state, it can happen that after he has deviated a few centimeters, he almost falls to the right, but this is not common.

As in the previous case of the head, the deviated position is more natural than any other, although when a certain degree of postural deviation is reached, an action can cause a static imbalance and a fall.

### 25.2.3 Deviation of index fingers

In this test, subject M is seated with eyes closed and arms extended forward pointing with his index fingers. If asked to maintain this posture, the index fingers tend to deviate slowly and continuously to the right, the right index finger somewhat more than the left. The values are similar to those of the Romberg test. In the inactive state the index deviates about 30 cm without any perceived displacement, but if facilitation by strong muscular effort intervenes at the end, he spontaneously corrects the deviation even without having received any command to do so, and brings the finger towards the center. Performing the test applying facilitation from the beginning, the same finger deviates only 5 to 12 cm in different tests, and in a time of 15 to 25 seconds, without increasing the deviation no matter how long the test is prolonged, the average being about 10 cm to the right.

In the test of moving the index finger with the entire upper limb rigid, in the direction from bottom to top in the corresponding sagittal plane, the same lateral deviation occurs, moving away from that plane. In all these tests, the position of the upper limbs deviated to the right is also the most comfortable or natural position, i.e. the one that corresponds to the neutral motor position, i.e. that of functional balance.

The usual clinical test of touching the tip of the nose with the index finger shows the same type of deviation. In the semi-inactive state, both index fingers are deviated, so that the finger touches the right cheek near the nose, at other times it touches under the right eye, etc. In such state, the subject feels neither the finger nor the place touched, since the pressure applied (semi-inactive state) only allows him to obtain a tactile sensation of the type of phase I or perhaps even phase II. In addition, the pointing movement is very

peculiar (see its mechanism in the section on body schema), and when the subject initiates the requested movement, it does so with an abrupt impulse and without defined direction. The aforementioned deviation would be even greater if it were not accompanied by some facilitation due to the effort required to give some coordination to the finger-pointing movement. Under facilitation (always by muscular effort), the deviation is almost completely suppressed and both index fingers can easily reach the tip of the nose, although sometimes there is still some deviation of the right index finger touching the right wing of the nose. Apart from this, when facilitation is applied, the sensation of finger and nose contact is much better than in the inactive state, although normalization is not reached since there is proximal deviation for the finger (it is felt shorter) and also for the nose (it is as if he touches towards the root of the nose). Under facilitation, the initial impulse referred to occurs to a much lesser degree and the movement is better coordinated.

#### **25.2.4 Deviation during walking**

In the previous tests on direction of walking, the reversal or deviation of direction is felt by the subject, walking the subject in a straight line and forward, either with eyes open to comply with the command not to deviate, or with eyes closed but being guided appropriately. However, if with eyes closed (and sometimes even open) he is allowed to walk freely, a great tendency to deviate objectively to the right is evidenced, more so in the inactive state than under facilitation. In a walk of about 5 meters taking 7 or 8 steps with eyes closed and the instruction to walk forward by himself, he deviates to the right by 1.70 meters at the end of the walk. In contrast, under facilitation, he deviates only 0.60 meters. As in the other examinations, in the inactive state he shows three times more deviation than under facilitation (see Table 39). In this type of test, it is easy to obtain, in the inactive state, a “star walking” by asking the subject to walk a few meters alternately forwards and backwards, as he will deviate respectively to the right and to the left.

It is noteworthy that, as in the previous motor deviation tests, spontaneous gait with objective deviation seems to the subject to be more natural and comfortable than correct gait when guided by the hand, regardless of whether there is a subjective deviation. What is important now is the most natural posture that the subject can feel, whether static or in movement; and the objectively deviated gait is felt by the subject to be naturally correct, and he is not bothered by the postural anomaly. Instead, when he is guided and walks correctly, there is something that disturbs him causing the effect of walking in a situation of imbalance.

The deviated posture to the right in patient M is so natural that if he is lying supine, in the inactive state, and is urged to adopt a natural position, he usually ends up bent at the waist to the right. But if he applies facilitation by muscular effort, he corrects the deviation spontaneously without receiving any special indication.

Considering all the phenomena, the general characteristics are the same in all cases, the following two aspects being noteworthy. One is the different deviation in the inactive state and under facilitation. The other is that the deviation appears as a neutral motor state,



i.e., as a posture of preference. As for the difference between the inactive and facilitated state, the respective quantitative values of deviation maintain a practically constant ratio for the different tests (see Table 39). The deviation in the inactive state is in all cases about three times that under facilitation. In degrees of deviation (index finger and gait tests), about 25-30° is obtained in the inactive state, and about 10° under facilitation. It can therefore be stated that there is a *deviation of the postural model* according to the respective states. Of course, the fact that the aforementioned motor anomaly appears segmentally, i.e., in the head, limbs, or in the whole body (Romberg test and walking), depends only on the degree of freedom of movements in the corresponding tests.

**Table 39.** Motor deviation values in patient M, in inactive state and under facilitation by strong muscular effort.

<i>Test</i>	<i>Inactive</i>	<i>Under facilitation</i>
Head	30°	10° ?
Romberg	18 cm in 60 s	4 -5 cm in 18 s
Index fingers	30 cm in 60 s	10 cm in 20 s
Gait (6.5 m)	1.70 m	0.60 m

Patient T has also presented deviations to the right side in the Romberg test, index fingers test, finger-nose test, etc. The Schneider patient (Goldstein and Gelb 1919) has also presented them to some extent and they have been attributed by these authors to the involvement of the cerebellum in the brain lesion, in our opinion with insufficient foundation.

Addressing at last the interpretation of the postural anomalies, it is clear that, if the other phenomena shown by our patients are not taken into account, those postural anomalies would have the appearance of a motor tone disorder, either cerebellar or cerebral, particularly of the frontal lobe in the latter case. Apart from this, other characteristic defects of a cerebellar lesion are entirely lacking.

The cerebellum and frontal lobe affect the red nucleus which is an essential organ in the distribution of postural tone. Lesions of this nucleus cause postural abnormalities of the same type (torsion of the head and trunk), according to several authors and ourselves (Gonzalo 1935, Kleist und Gonzalo 1938).

Such postural anomalies do not bother the patient at all, but on the contrary, they constitute new equilibrium positions of the static system, or “new comfortable postures” according to Goldstein (1926).

In our patients, any cerebellar or frontal lesion is excluded, since there is only a parieto-occipital lesion corresponding to the central syndrome here studied. However, postural deviations are also known in parieto-occipital lesions, as in the cases reported by Hoff and Schilder (1927) and others; although it could be hypothesized that cerebral repercussion somehow affects the frontal lobe, which is more related to postural tone than the parietal lobe. However, the previously studied sensory disorder of orientation in touch does not enter into this argumentation at all. If we have included the phenomena of

postural deviation in this Sec. 25, it is precisely because of their formal similarity with the mentioned sensory disorder, thus trying to obtain an interpretation as unitary and simple as possible for the whole of the various phenomena. In this regard, it should be admitted that postural function, being linked to sensomotility, depends both on the purely motor element and on sensitivity. It could then be thought that the orientation disorder exists primarily in tactile sensitivity and exerts a parallel influence on postural motor function, which is thus secondarily altered.

By examining certain alterations described in the functional complex of sensomotility, we find aspects of great interest for our purpose. Thus, in both cerebellar and frontal lesions, which present the corresponding defect of postural tone, [Goldstein \(1926\)](#) has also observed special perceptual disorders in several sensory systems, such as tilted perception of the visual image, deviations in the localization of tactile and auditory stimuli, etc. This symptomatic complex has also been confirmed in some cases of similar lesion by [Hoff and Schilder \(1927\)](#), [Fischer and Pötzl \(1924\)](#), [Marburg \(1931\)](#) and other authors. It happens that postural tone deviation due to cerebellar or frontal lesion induces some sensory spatial deviation, towards the same side in cerebellar lesion and towards the opposite side in frontal lesion.

In the cases reported by [Goldstein \(1926\)](#) with a cerebellar lesion, the visual deviation from the vertical is mainly perceived by the homolateral eye as a rotation to the same side of the lesion. There may also be certain deformations of the visual image of the object, in some of its axes, etc.; and in addition, different sensory stimuli (tactile or auditory) can influence the aforementioned visual deviation. Analogous manifestations are also found in touch, for example, drawings on the skin are felt deformed in the direction of the pathologically deviated motor tone. There is therefore a spatial sensory alteration due to the motor postural alteration, and such sensory alteration is diversely influenced by intersensory action. But there is also the reciprocal effect that the sensory factor exerts an influence on muscular tone, thus closing a circle of mutual effects. Thus, it is possible to obtain deviation in pointing, and deviation in body position, by means of acoustic, tactile, visual stimuli, etc., the deviation being towards the same side of the body on which the sensory excitation acts [phenomena studied by [Goldstein \(1926\)](#) and later confirmed by [Hoff and Schilder \(1927\)](#)].

In short, the postural deviation due to cerebellar lesion has an effect in the same direction on the sensory space (deviation of the localization of a tactile stimulus, tilted vision, etc.); and reciprocally, modifications in the sensory field by its excitation determine changes in the distribution of postural tone. That is, mutual sensorimotor inductions occur due to the special lability of the sensorimotor functional complex.

Two cases of [Weizsäcker \(1924\)](#), seemingly suffering from a unilateral labyrinth lesion, would belong to the first type of induction mentioned (sensory spatial deviation secondary to a primary deviation of postural tone). In these cases, the alteration of postural tone secondarily caused a tilted visual image. We have observed a similar induction phenomenon in a patient diagnosed with left cerebellar abscess and successfully operated on. This patient was affected by very marked cerebellar motor disorders on the left side of his body, and showed episodic disorders of visual image orientation during the acute period before surgery. He spontaneously said that sometimes, upon waking up in the morning, he had seen for a few moments the whole visual

scene tilted 90° to his left (side of the lesion). However, his vision was not blurred and did not show any color change, contrary to what occurred in subject M.

With respect to these spatial sensory alterations secondary to postural deviation, it should be noted that, for example, the visual image tilt is moderate, and above all, it is a disorder that has nothing to do with asynchrony and dynamic reduction of the sensory field. The induced disturbances are nothing more than orientation changes in the sensory space due to the effect of the sensory-motor correlation that acquires a new aspect due to an alteration of the motor element. Regardless that we may return to this topic in another part of this work, for the moment we bring it to an end.

Coming back to the starting point, and in the light of the various phenomena considered (mainly sensory deviation in postural abnormalities due to a cerebellar lesion), it seems appropriate to reciprocally interpret the motor disorder in subjects M, T and even Schneider, as an effect induced by the inverted or tilted sensory field. Although inversion in patient M occurs in all sensory systems (visual, tactile and auditory), when dealing with motor deviation it is sufficient to refer to the disorder of orientation in touch since it is most strongly linked to the sensorimotor complex. Thus, the interpretation is different from that of Fischer and Pötzl (1924) and other authors. These authors believe that the lesion of the angular gyrus or neighboring regions causes the same postural tone disorders as the cerebellar lesion, thus postulating the existence of a primary motor center in that brain region which, however, is in a fully sensory area. Instead, the explanation given here, much simpler and unitary, of an *induced postural deviation* (motor deviation secondary to the deviation of orientation in touch) would suffice. Thus, the body schema of subject M is rotated to the right both subjectively and by posture (objectively), by the sole action of the sensitivity deficit.

Finally, for the sake of clarity, we summarize in Table 40 the different sensorimotor pathological phenomena, especially in the case of cerebellar lesion (primary motor disorder) and in the case of central syndrome (primary sensory disorder).

**Table 40.** Sensory-motor dynamic phenomena.

<i>Type of lesion</i>	<i>Primary manifestations</i>	<i>Induced Manifestations</i>
Cerebellum	Homolateral deviation of the postural muscular tone	Homolateral deviation of spatial values in the sensory field (tactile, visual, etc.). Reciprocally, sensory stimuli may deviate postural tone.
Parieto-occipital (Central syndrome)	Contralateral deviation of the sensory field (tactile, visual, etc.)	Motor postural deviation in the same direction as in the sensory field

## 26 Theory of orientation in the sense of touch

### 26.1 Orientation and localization in the spiral development

We have seen how the orientation disorder is interconnected with that of stimulus localization. Both functions, inversion and proximal deviation, always go hand in hand, and in certain phases, such as contralateral localization, they are so closely linked that they are equivalent. In fact, a change of laterality is a change of orientation. Such characteristics deserve a careful analysis in order to address the theory of spatial orientation.

#### 26.1.1 Differences from allochiria

Contralateral localization of stimuli in subject M leads us to some brief considerations on allochiria (contralateral localization of tactile stimuli), in order to properly delimit these various contralateral manifestations. Allochiria was first described by [Obersteiner \(1892\)](#) in certain migraine auras, then studied by [Jones \(1908\)](#), by [Kramer \(1915, 1917\)](#) in brain and medullary lesions, by [Redlich and Bonvicini \(1911\)](#) in brain lesions, and finally by [Schilder \(1923/1935\)](#) in a brain case and two cases of tabes dorsalis. This phenomenon has also been studied experimentally, first by [Mott \(1893\)](#) in monkeys with medullary hemisection, and more accurately by [Dusser de Barenne \(1913\)](#) in dogs and cats by combining medullary hemisection and strychnization. The clinical observations reported are brief and do not exhaust the study of the phenomenon, apart from the fact that it is often a transitory phenomenon. Patients are uncertain to distinguish the side of the body that is touched, and sometimes tend to refer the sensation to the symmetrically opposite side (allochiria or alloesthesia). Although it is a rather rare symptom, other cases have been subsequently reported by various authors but without much more precision than in the above-mentioned observations.

We have observed a case of allochiria in a subject with biparietal brain lesion (Fig. 145), who described the phenomenon spontaneously during the examination since he had become aware of it under special conditions.

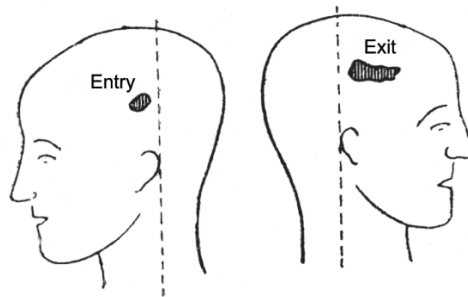
Patient U.G. suffered a gunshot wound (see Fig. 145) in 1938 during the Spanish Civil War. At first he suffered from left hemiparesis with sensitivity disorder on the same side, language difficulty (especially nominal amnesia), visual fatigue, etc.; and remained so for about two weeks. He also showed a set of symptoms of agnosia and apraxia such as left-right confusion, finger agnosia, constructive apraxia, agraphia (or dysgraphia), acalculia, amnesic aphasia especially for

colors, and dyspraxia, especially in the left hand. All this was possible to be considered as Gerstmann's syndrome (Gerstmann 1931) with predominance of apraxia.

The left hemiparesis improved rapidly, while hemihypoesthesia on the same side was still evident. He spontaneously reported during the examination that when walking in company and carried by the right arm, he felt the contact on the left side (the affected side). During the examination he spontaneously reported that while walking in company held by the right arm, he felt the contact on the left side (the affected side). On examining touch, it was found that stimuli on the right side were indeed localized symmetrically on the left side. The opposite never occurred. The contralateral localization, symmetrical to the excited site, took half a minute or less to occur, but at the same time it was possible to perceive the stimulus at the stimulated site. Thus, the sensation was double, on the normal side and on the affected side. This occurred mainly when the stimulus was strong, the sensation being weaker and diffusely localized (as deep currents) on the contralateral side. If the stimulus applied was very weak, it was only perceived contralaterally. By lightly rubbing the right cheek with a very thin paper (tickling), he felt after about 15 seconds the need to scratch the left cheek, without perceiving anything at all on the right cheek. When the change of side occurs, a dizzying sensation may appear due to confusion in distinguishing which side is actually stimulated.

Examination of tactile sensitivity on the left side revealed some impairment of all functions; for example, localization with proximal deviation, a deficit in perception of passive joint movement and postures, a deficit in Weber's test, a tendency to contract the musculature to enhance perception, some failures in manual recognition of objects, and indirect way of recognizing them, etc.

The visual field showed a very moderate concentric reduction and several annular scotomas due to fatigue. There was no inversion of chromatic isopters.



**Figure 145.** Patient U.G. Biparietal brain injury (transverse trajectory of the bullet, somewhat oblique forward and to the right). The patient showed moderate left hemiparesis and hemihypoesthesia, various disorders related to agnosia, apraxia and aphasia, some concentric reduction of the visual field, etc., and allochiria: tactile stimulation on the right side (healthy side) was localized either on the left (affected) side only, or on both sides at the same time. The reverse case (from affected to normal side) never occurred.

In summary, as far as alloesthesia is concerned, there is a tendency to localize stimuli applied on the healthy (or less affected) side on the contralateral hypoesthetic side, and in a symmetrical way. This type of examination, carried out rapidly during the daily care of a large number of war wounded with brain injuries, cannot be considered complete for an exhaustive study of allochiria, but it will suffice for the moment as a guideline. This case examined by us can be equated to the case of Schilder (1923/1935) suffering from hemiplegia and hemihypoesthesia, apparently due to a capsular and perhaps also a cortical lesion. This patient also suffered from allochiria only when the healthy side was stimulated. We discard the numerous hypotheses to explain the phenomenon, especially those related to the body schema, as in the case of Schilder, as being very poorly founded.

Instead, it seems clear that the simplest interpretation of these cases is to relate them to hemiplegic synkinesias. The affected side has an abnormal permeability to stimuli from the healthy side. This would occur either by muscular innervations that also provoke reflex or synkinetic contractures on the impaired side, or by tactile stimuli on the healthy side that tend to be localized on the affected side. Furthermore, both in synkinesias and in these cases of alloesthesia, the transfer is symmetrical. In the light of the above, it is not possible to identify this type of allochiria with the one obtained in the tactile inversion studied here, since both the mechanism and the phenomena are completely different.

However, it cannot be ignored that other described cases of allochiria do not fit the type of the two above. Thus, in the two cases of [Kramer \(1915\)](#), one with bulbar apoplexy and the other with cortical hemiplegia, as well as in the case of [Redlich and Bonvicini \(1911\)](#) with cortical hemiplegia, the change of stimulus localization was, contrary to what was indicated above, from the affected to the healthy side, also symmetrically. Here, as in some tests of monocular polyopia, it might be thought that the stimulus is deviated toward the most sensitive site. The same occurs in the mentioned experiments of [Dusser de Barenne \(1913\)](#), although strychnization then intervenes in a special way. Singular phenomena have also been described, such as the allopargia of [Fuchs \(1908\)](#) in peripheral nerve lesions, which consists of paresthesias and localized pain contralateral to the lesion. This is a disorder that could be similar to the contralateral “repercussions” of the functional disorder in peripheral nerve injuries, first described by [Bourguignon \(1923, 1932\)](#) (in radial nerve injury, excitability is altered in the radial nerve of the opposite side), and later confirmed by [Altenburger \(1933, 1936/37\)](#) in different ways. In conclusion, all these types of contralateral localizations do not seem to have anything to do with the contralateral localization in the tactile inversion described in the present book. In the latter tactile inversion, there is not only contralateral but also proximal deviation, whereas in classic allochiria the localization is symmetrical, a fact that all authors have always insisted on and that in our case of allochiria is very clear (slight excitation of the healthy cheek leads the patient to scratch the affected cheek).

Trying to find similarities, one could suppose that there is an inverted orientation in the cases in which the perception of the stimulus moves from the affected zone to the healthy one, as occurs in the cases of [Kramer \(1915\)](#) and [Redlich and Bonvicini \(1911\)](#). But the transposition should not be symmetrical but with a strong proximal deviation towards the body axis. Until more complete and conclusive analyses are available, all known cases of allochiria cannot correspond to the tactile orientation disorder described in the present work. This last disorder does not refer, of course, only to the lateral transposition of a simple touch, but also to a whole set of phenomena and tests described here on the inversion of a cutaneous rectilinear stimulus, motion inversion of a moving object on the skin, inversion of joint movement, etc., all these phenomena following well-defined rules.

With respect to vision, similar considerations must be made with respect to the visual alloesthesia of [Hermann and Pötzl \(1928\)](#). This disorder consists, as its name indicates, of an allochiria of the visual field. The visual scene or peripheral images from one side of the visual field are perceived on the opposite side. Even more than in the described cases of tactile alloesthesia, in these visual cases the phenomenon is sporadic and very ill-

defined, like the change of site in scotomas due to migraines and similar states. The cases reported by these authors correspond to old clinical observations that are very superficial and inappropriate for drawing conclusions, apart from the transience of the visual disturbances. More evident is the last of these cases, observed in 1925, with several areas of softening, mainly in the right parieto-occipital convexity, where visual alloesthesia appears more clearly: objects in the periphery of the left field tend to be perceived on the right side. This phenomenon also appears sporadically. If the change of side is from the amblyopic to the healthy side, a reversal of orientation could be admitted, although much data is lacking to be sure. Given the imprecision of the tests, it is not known whether there was proximal deviation (towards the center of the visual field) when changing from side to side, and nothing is indicated about inverted or strongly tilted vision with respect to the vertical. Regarding the interpretation of visual alloesthesia, the aforementioned authors make extensive comments, in contrast to the exiguous clinical observations, establishing a parallelism with tactile alloesthesia (allochiria), and especially with the characteristics of the experimental studies of [Dusser de Barenne \(1913\)](#). Probably, as the authors were impressed by the lateral transposition, they do not consider the general problem of inverted orientation in vision.

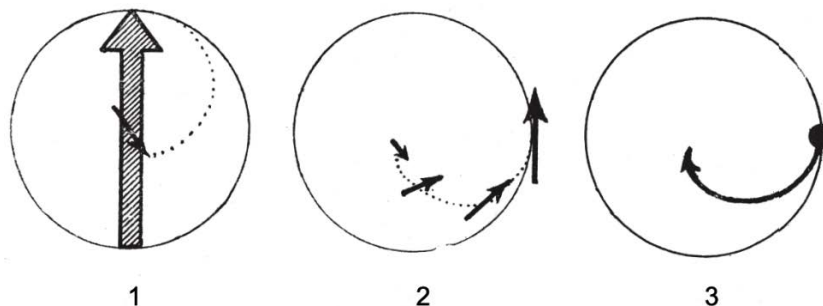
### 26.1.2 Spiral development in vision and touch

Having established the difference between true spatial orientation inversion and alloaesthetic disorders, we now return to the issue of orientation as an essential factor in spatial localization. In our patients with brain lesions, the spatial disorder is of such a nature that, by combining inversion with proximal deviation, a *spiral development* of localization (spiral deviation) is obtained along the process. We have already referred to this spiral trajectory above. Suffice it to recall Figs. 100 and 125 for a point stimulus on a hand and on the face respectively, Figs. 127 and 130 for a rectilinear stimulus under analogous conditions, Fig. 132 for a stimulus moving on the skin, and Figs. 139, 140 and 141 for articular movement leading to a subjective trajectory during walking shown in these figures. Thus, the spiral trajectory is a feature of general significance that we must use as a starting point in the theory of tactile orientation.

This type of spiral trajectory is as characteristic of the tactile system as it is of the visual system. The spatial similarity of both systems allows a more complete study of the different phenomena. Proximal deviation is more satisfactorily determined in touch, whereas vision is more appropriate for studying the regularity of the inversion rotation and its quantitative evaluation. Apart from this, there are other differences described next. For the visual field, the demonstration of the spiral trajectory is simpler than for the tactile field, since the structure of the latter is less homogeneous and lacks a central point (or fovea) on which to center the mentioned spiral development.

In cases 2 and 3 of Fig. 146, a diagram of the spiral development for the visual field is shown, in the case of a stimulus of varying intensity and situated at the visible end of a horizontal meridian. By supplying sufficient luminous intensity, the stimulus can be seen in that meridian more or less peripherally; but as the intensity decreases, the perceived stimulus tends to follow a spiral course, progressively approaching the center of the visual

field and moving to the opposite side, finally coming very close to the center. Thus, a contralateral centripetal translation is obtained, i.e., the aforementioned spiral deviation is obtained as a combined effect of the rotation of the visual field and the concentric reduction of the field (proximal deviation).



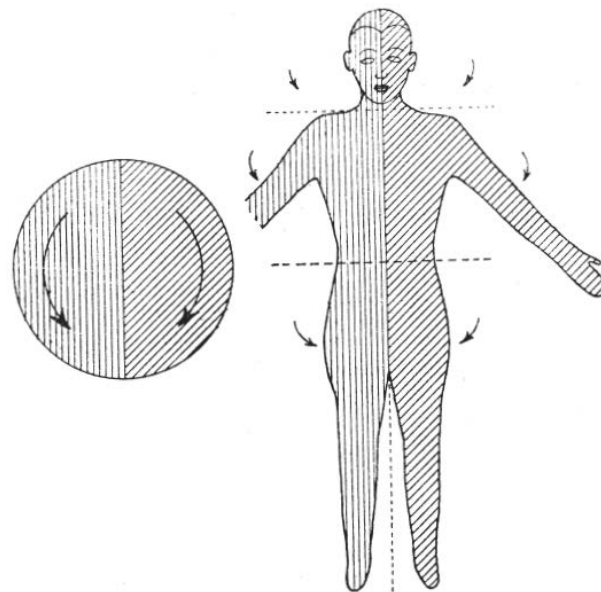
**Figure 146.** Diagram of the spiral trajectory in the visual field. 1: Arrow in the center of the visual field whose perceived image is becoming inverted and decreasing in size as the illumination is suitably reduced. In this process, the upper end of the arrow (as well as the lower end) describes a spiral curve due to the joint effect of the orientation reversal process and the reduction of the size of the arrow. 2: Arrow located at the visible end of a horizontal meridian of the visual field. When the illumination decreases, the image perceived during the inversion process follows a spiral with some displacement with respect to the previous case. 3: Spiral trajectory as in case 2 but here the test object is a small circle situated peripherally.

It should be noted that when studying the inversion of the visual image orientation, the test was made with the arrow in the center of the visual field (case 1 of Fig. 146), so the mentioned spiral translation was not obtained. However, if we take into account that the size of the image of the arrow is progressively reduced during the image inversion process, the result is that each end of the image of the arrow follows a spiral trajectory progressively approaching the center. Recall the reduction of the perceived trajectory in the inverted visual perception of a moving object, and also in the steps perceived as inverted. In the above-mentioned test with central position of the test arrow, if the position of the arrow is vertical, when the image of the arrow is inverted, its upper end will be down and close to the center; but if the position of the arrow is horizontal, the change of the image will be from one side to the other side, which corresponds to the typical case of inversion in the tactile field. In relation to visual inversion, it should be noted that in the corresponding sections (Secs. 12-14 in Vol. 1) only one experiment on peripheral inversion (Fig. 71) had been reported, pointing out only the tilt of the image with respect to the vertical test arrow, but not its absolute position in the visual field, i.e., its spiral translation had been omitted. But now this can be shown in case 2 of Fig. 146.

As for the tactile field, the spiral deviation does not seem to show such a simple and unitary character, since in addition to the irregularity of the field in question, both morphologically and functionally, certain fragmentations take place. Thus, a stimulus in the cephalic area, for example in the face, is not perceived in the lower part of the body (the feet or even in the pelvic region). In this case, the inverted perception, in its contralateral rotation around the vertical axis of the body, is localized also in the head and



toward the vertex (Figs. 125 and 130). A deviation more similar to that of the visual field is only obtained when the stimulus acts on the anterior plane of the trunk, over the chest, as shown in Fig. 126. Thus, the difference in the spiral rotation between the visual and tactile fields lies in the fact that for the former it always takes place around the central point of the field, whereas in the latter there are, as we know, *independent rotations* around the axis of the body, since there is no central point as in the former case. During the spiral reduction, the tactile field is fragmented into the three zones already studied, as shown in Fig. 147. Apart from this peculiarity, there is a similarity between vision and touch in the inversion rotation, and each lateral half of the sensory field (visual and tactile) can act independently by rotating in opposite directions, as is schematically depicted in Fig. 147.

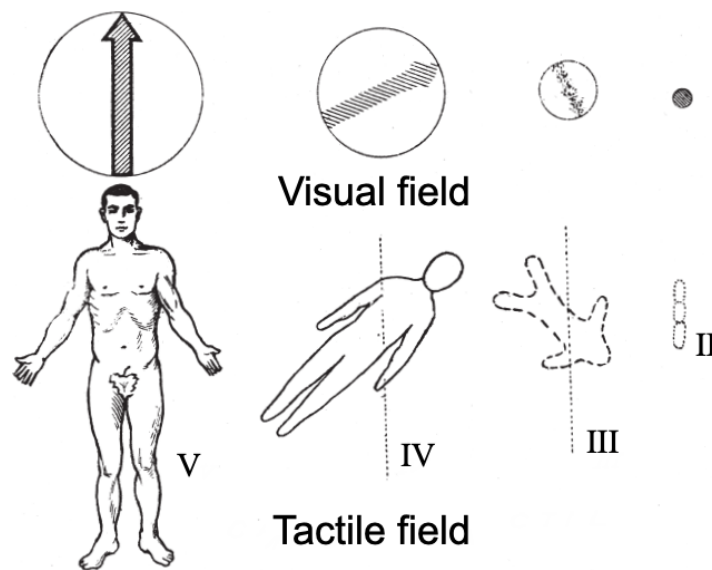


**Figure 147.** On the left: In the inversion of the visual field, each half of the field rotates around the center independently. On the right: In the inversion of the tactile field or body schema, each half of the body has 3 independent zones for inversion, namely, head, upper limbs and lower limbs. Note that the opposite rotations in both halves have the same sense in vision and touch.

### 26.1.3 Spiral development of the sensory field. Sensory organization law

The spiral process, considered more broadly, involves a spiral development of the sensory field (tactile or visual) that affects the whole sensory organization. Thus, the greater the degree of inversion, the more pronounced the proximal deviation, i.e., the greater the reduction of the sensory field. This reduction reaches all types of functions, thus there is a structural change of the field resulting in a *residual organization field*. Its characteristics refer mainly to inversion, reduction and irradiation, affecting also sensory qualities, although in the spiral process, the spatial organization acquires the greatest significance. It is not possible in such a situation to determine independent factors, such as orientation inversion, field reduction, or irradiation. On the contrary, these factors are aspects of a same process in which the spatial structure of the field tends to become a

residual structure. Inversion is a particular aspect of localization, changed in sign by special circumstances that we shall study. The reduction derives from the contraction of the sensory dimension affecting both space and time, thus adequately explaining disturbances in motion perception, spatial acuity, size, etc. Irradiation means a reduction of local specificity, i.e., dilution of the local sign or spatial value. This dilution manifests itself as a loss of cohesion between the parts of the field, this being more evident in touch than in vision. Naturally, the greater the residual character, the greater the loss of functional differentiation and the greater the destruction of functional entities (individualities). In this way, the normal complexity of what is heterogeneous or discontinuous is changed, by effect of the reduction, into homogeneous and continuous, where all traces of organization end up vanishing.



**Fig. 148.** Spiral reduction process of the sensory field through the different phases of residual organization. Note the similarity of phenomena for the visual field and for the tactile field. In both, the degree of inversion is parallel to spatial contraction (concentric reduction, proximal deviation) and to irradiation. At the end, all differentiation or organization disappears. See text for further details.

This overall, unified process of sensory field reduction is illustrated in Figure 148, where different phases of the process are depicted for both the visual and tactile systems in patient M. In both systems the rotation is to the right side (clockwise). In vision, this occurs for the right eye in foveal image. For touch it occurs when the whole tactile field (i.e. the body schema) is considered, either static or during walking. In the case of vision, when the visual image is gradually tilted until it ends in an almost complete inversion, the field becomes smaller and smaller, i.e. the perceived image reduces in size and at the same time becomes diffuse due to irradiation, until a moment arrives in which the entire configuration is lost and only a weak, amorphous and achromatic luminosity remains. As for the color, the almost white arrow in optimal vision, passes through gradually darker green colors due to chromatopsia, until even the color disappears when the whole

configuration vanishes. Thus, at the end, all traces of organization disappear. Similarly occurs in touch, although the whole process cannot be determined as directly and simply as in vision. Indeed, in moderate reduction, the tilt of the body schema and its reduction in size are obtained immediately with suitable experimental conditions. But we know that with a subjective tilt of more than 50°- 60°, the body schema vanishes due to loss of cohesion (independent rotations of its parts, etc.), being then impossible to follow directly the course of the reduction. For this reason, phase III as depicted in Fig. 148, is an ideal construct based on fragmented determinations, since by means of local excitations in that phase the size of the body schema can be inferred (by means of proximal deviation), as well as the subjective orientation. Apart from that, the corresponding irradiation and alterations derived from it lead at the end, as in vision, to erase all functional differentiation, as it occurs already in phase II, and mostly in phase I.

In relation to the reduction process, some peculiar aspects should be pointed out. First of all, it should not be forgotten that the distal sectors of the field lack inverted representation since the proximal deviation is then present to the maximum, so particularly in touch, the limbs cannot suffer any inversion. This constitutes an essential difference with the usually known alochiria where the side transposition is symmetrical. It is also important to take into account, for further theoretical interpretations, the noticeable reduction of the field size as inversion occurs, and especially in touch, the fragmentation into parts (loss of mutual references).

Finally, we must emphasize that a deeper understanding of the inversion phenomenon relates this anomaly to a disturbance of the whole spatial structure, especially associated with spatial reduction (field shrinkage, etc.). Inversion is thus one of the fundamental aspects of the *spiral development of the sensory field*. This process accounts for all types of sensory activities and the various possibilities of general organization of the field. This statement thus constitutes a law of sensory organization, the psychophysical basis of which will be discussed in the next section. We now point out that this statement constitutes one of the two fundamental laws of the brain dynamics described in this book. The other law refers to the repercussion effect of the lesion depending on its magnitude and position, as we already know. All this will have to be dealt with more extensively in another work, but its importance can already be appreciated now.

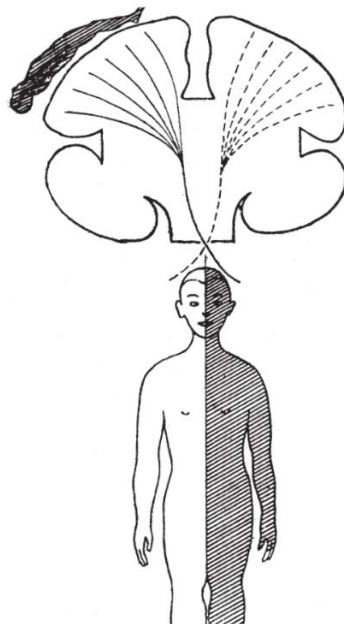
## 26.2 Mechanism and structure of spatial orientation

In the section on visual image orientation (in Vol. 1), the inversion was interpreted as an effect of retinal autonomy (with primary image inverted) when the functional visual-haptic complex (visual-tactile correlation) that would normally provide normal orientation was broken. Later it was shown that the orientation disorder also occurs in other sensory systems, as now in touch. This type of disorder is thus of a general nature, and needs to be studied more extensively to satisfy the various questions that arise in a general way. Also in touch, inverted orientation is interpreted by referring to the primary image of the inverted projection in the brain. Instead, it would be impossible in a sense as fundamental and primary as touch to derive normal orientation from the influence of

another sensory system, as was done in vision. Because of these and other similarities and differences between vision and touch, the orientation issue needs a more careful examination and a new, much broader approach. The following points will therefore be addressed below: origin of spatial inversion, mechanism by which it appears, theories on orientation, and general principles involved in our interpretation.

### 26.2.1 Origin of spatial inversion

The origin of spatial inversion must be sought in the layout of the tactile field in the brain, more specifically, in the projection cortex (marginal zone). Not only is there a transposition of sides due to decussation of the sensitive pathways, but there is also a vertically inverted arrangement in the cortical projection (see schematic in Fig. 149). The same is known to occur in vision, although the inversion of the visual field is considered to result from the optical system of the eyeball, and the inverted orientation that already exists in the ocular retina is projected anatomically point by point onto the calcarine cortex or “cerebral retina” of [Henschen \(1926\)](#). Thus, the chiasm intervenes only to achieve binocular correspondence, i.e., to produce the cyclopean eye. Therefore, cortical sensory projection areas show a contralateral and vertically inverted arrangement with respect to their sensory fields (visual, tactile, auditory). Hence, there is a spatial congruence on the brain surface between the different sensory systems; for example, tactile and visual stimuli from the lower right are projected into the upper and left brain.

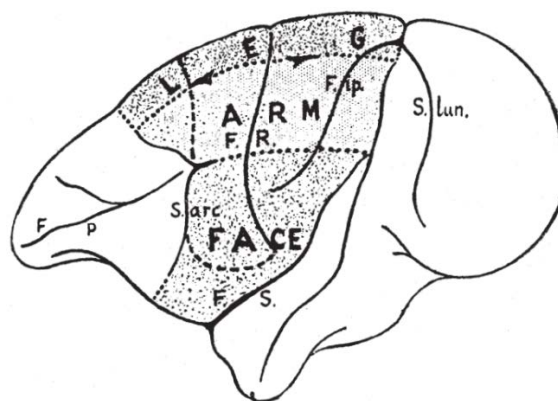


**Figure 149.** Diagram to recall the brain projection of the somatic sensitivity (tactile field), contralateral and vertically inverted for each half.

Thus, in the interpretation of inverted perception in vision we have first considered the local effect of retinal inversion, being equivalent to refer to ocular retina or to cerebral

retina for such spatial effects, as was said. But now, having discovered inverted perception in other senses, touch and hearing, the question of inversion is focused on the projection onto the cortex. Thus, it is curious that the old and much discussed issue of ocular image inversion (see Sec. 12.1 in Vol. 1) has led, through the study of our brain-injured patients, to a cerebral effect encompassing all sensory systems (inversion occurs in vision as well as in the other spatial senses). Inversion has therefore ceased to be merely ocular and exclusive to vision, becoming a strictly cerebral effect of a general nature. From the anatomical point of view, the starting point is the ocular crystalline lens that produces the optical inversion, transferred in the same way to its cortical projection. According to the theory of [Ramón y Cajal \(1898\)](#) on nerve crossings, the decussation and inversion of the other sensory pathways achieves a congruent spatial projection (of the same sign) in the brain.

As for the remarkable phenomenon of fragmentation of the body into three autonomous zones (head, trunk-upper limbs and pelvis-lower limbs) showing independent rotations in inverted perception, this phenomenon probably has a physiological basis in the special somatotopic projection of the sensitive cortical area, according to the cortical strychnization experiments of [Dusser de Barenne \(1924\)](#). As described by the author himself, “these experiments reveal a well-defined subdivision of the sensitive cortex into three sectors: area of the face, area of the trunk and arm, and area of the leg (Fig. 150). The boundaries between these areas are precise and respected even by strychnine, which generally causes a diffuse discharge when introduced into the central nervous system. Strychnine has never been observed to spread from the leg to the arm or to other subdivisions when applied locally to the postcentral circumvolution”.



**Figure 150.** Sensory cortex in the convexity of the hemisphere of the macaque monkey, as revealed by the method of local strychnization of the cortex ([Dusser de Barenne 1924](#)). Note the three independent areas (face, arm and leg), clearly delimited by dotted lines. The dashed lines indicate the frontal boundary of the electrically excitable cortex.

Leaving aside for the moment the analysis of these morphological issues, we simply emphasize that the acceptance in our theory of the effect of projection pathways, is an important concession to the anatomical aspect of the functions. This stance cannot surprise the attentive reader of this work, since already at the beginning of it (Sec. 1.2.3

in Vol. 1) the syndrome of cortical projection zones (which we have called marginal syndrome) was accepted as an extreme case fully characteristic of brain localization since it affects the conduction of the long pathways. However, such acceptance of brain anatomy does not imply that we are going to develop a situation of compromise with the brain localization theory whose statements we should reject. Rather, we merely state that, along with mass action, excitability and organization, the anatomical nervous texture must be taken into account, especially for the spatial structure. This anatomical factor has often seemed to play a minor role in our conception, but in certain issues such as the present one, it acquires a major significance. Thus, the pathophysiological question of spatial orientation (and of space in general) can have an anatomical-physiological basis.

In this sense, it is convenient to indicate the role that the anatomical-physiological complex plays in the orientation function, according to the possible cases (or syndromes). Let us consider the following cases in gradation: normal subject (localization sensation congruent with stimulus), central syndrome (sensation preserved but inverted localization), and projection or marginal syndrome (suppression of sensation contralateral to the lesion). In a normal subject, the anatomical factor of contralateral inversion is present in a latent form, being supplanted by the physiological action of the central mass, which, by reorganizing the field, excludes any spatial discordance between stimulus and sensation. Instead, in the central syndrome, which arises from a brain or hemisphere with reduced activity, the marginal (projection area) effect tends to emerge, and the greater the brain deficit, the more evident this effect becomes. Thus, with a great loss of central nervous mass (great asynchrony as we have seen), the primitive action of the anatomical factor of the marginal projection becomes evident. In this case, simple sensation or elementary excitability is possible to some extent for the corresponding "sensitive field", but its spatial localization undergoes a change, being guided by the contralateral and inverted orientation of the marginal arrangement (in the projection area). That is, the orientation of the "spatial field" tends to reflect the orientation of the projection area. There is thus a functional incongruence between the sensitive field and the spatial field. Finally, if the brain lesion reaches the projection (marginal) area, the ordinary projection syndrome (which we call marginal) appears, in which the absolute sensory defect remains contralateral to the lesion since the conduction pathways go to the projection area. As is well known, in a marginal (projection) lesion, defects are produced on the contralateral side and inverted vertically; thus, a lesion in the left superior calcarine causes blindness in the right and inferior quadrants.

In view of the above, it is striking how the anatomical texture of the marginal projection can give rise, depending on the circumstances, to two manifestations which, despite their external dissimilarity, have a certain internal unity. That is to say, in one case we have the classic projection (or marginal) syndrome with totally contralateral and inverted defect, and in another case we have the inverted perception of orientation in the central syndrome. Thus we see that two brain theories as antagonistic as the classical one of anatomical localizations in the brain and the one of brain dynamics presented here, find for once a common nexus in basing the issue of orientation on the anatomical-physiological complex.

Returning to the configuration of the projection area, it should be noted that in order to fully explain the tactile inversion in patient M, each body half should be located in the totally opposite cortical area, i.e., in addition to lateral and vertical inversions, there should also be inversion between front and back (e.g. the anterior part of the body represented backwards in the cortex).

### 26.2.2 Inversion mechanism

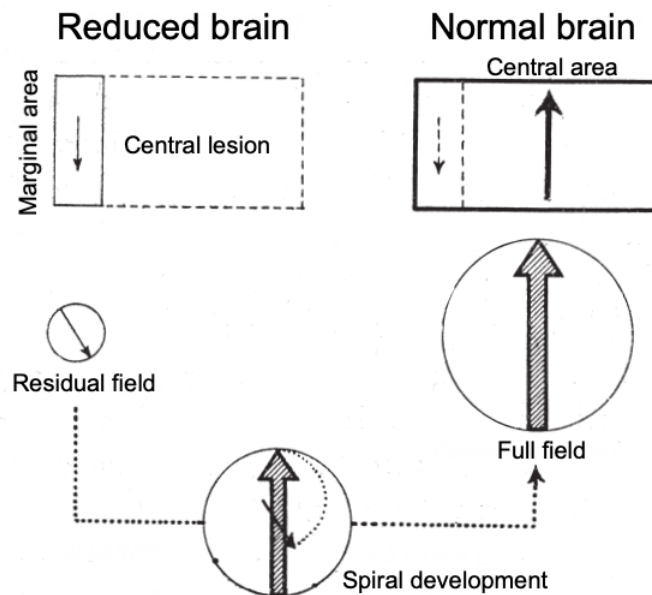
Here we analyze the conditions for each of the two extreme orientations, the normal one and the inverted one, and how the transit between them occurs. For this, both the brain mechanism and the symptomatic changes in the sensory field must be considered.

As already indicated above, inversion is produced by the action of the marginal brain disconnected from the influence that the central area must normally exert. Such an inverted orientation genetically corresponds to a primary (nativist) orientation that could also be called autocentric or idiocentric, i.e., an orientation that arises directly from the marginal structure, being independent and prior to any external experience. On the contrary, the joint action of the whole brain (central action), correcting the marginal action, gives rise to an allocentric orientation, normal and congruent with the stimulus, that is, adapted to the experience by virtue of the reorganization of the sensory field.

As for the “brain mechanism” of inversion, the experiments described above show that there is an asynchrony of excitability between the two orientation systems, marginal and central, resulting in an abnormal sensory interval between the two extreme orientations. According to the rule of dynamic reduction, inversion appears with weak stimulation because it is a more elementary function, whereas the correct orientation corresponds to strong stimulation. Starting from the marginal action, with inverted orientation, the activity of the central area can be achieved by increasing the stimulation. In this way there is a recruitment of brain mass that corrects the orientation following Fechner's law. This recruitment occurs through different balance states between marginal and central action.

Parallel to this recruitment, the phenomenology of the sensory field evolves, and not only in orientation but in the whole organization, as indicated by studying the process of spiral reduction in Fig.148. This parallelism or correspondence between the state of the brain and the state of the sensory field is illustrated by means of the diagram in Fig. 151. It shows that a residual field corresponds to a brain functionally reduced by a central lesion. This field is considerably reduced, has an inverted orientation analogous to the marginal projection of the field and other pathological features that we shall see. Reciprocally, an entire or full field corresponds to a normal brain (with active central zone). Such a field, with full integrity of the sensory functions, is a larger field because it responds to a greater activity of brain mass than in the previous case, and has a normal orientation congruent with the stimulus, all due to the sensory reorganization determined by the central mass. The transition from one extreme state to the other by recruitment of brain mass gives rise to the aforementioned spiral development of the sensory field by the joint effect of progressive re-inversion and enlargement. The *neurosensory correspondence* (between brain organization and sensory field organization) is therefore

evident with respect to both orientation and size. Such characteristics of the field and the brain are valid for both vision and touch since orientation phenomena and organization are similar in both.



**Figure 151.** Diagram to illustrate the brain mechanism of orientation and the corresponding sensory field (both visual and tactile). A very small *residual field* with inverted orientation, as in the marginal area, corresponds to a brain reduced to the marginal area by a “central” lesion. A full field of maximal size and functional reorganization such that orientation is congruent with the stimulus, corresponds to a normal brain, i.e., one with a larger brain mass. The transition from one brain state to another by recruitment of brain mass is reflected in the spiral development of the sensory field (field enlargement and re-inversion).

In trying to understand the phenomenon of re-inversion (normalization of orientation) new questions arise. As for vision, we admitted that tactile or haptic support is involved in the formation of normal orientation, thus establishing the visual-tactile complex for normal (correct) orientation. But now, in the case of touch, where does the support come from in order to establish an orientation congruent with the stimulus? It is clear that it is not possible to resort to intersensory supports when all systems suffer from the same inversion disorder. Besides, it would seem inadmissible in a sense as fundamental and primary as touch, so intimately linked to the corporeality and orientation of the own body. Therefore, the explanation in the case of touch must be sought in the tactile system itself, i.e., necessarily in its state of organization. Thus, the orientation problem is simplified and unified.

In relation to phenomenology, the raised question of how touch re-inverts its orientation by itself can be solved by taking into account the characteristics of the sensory field. Thus, we see that the characteristics of the field for the correct orientation and for the inverted one are very different. The inverted field has a residual organization, it is an extremely reduced field, presenting an inverted local sign, and on the verge of losing all structure. In contrast, the normal field, by virtue of its reorganization, has the opposite



properties, such as large size, cohesion, orientation congruent with a stimulus, etc. Thus, the sensory field corresponding to inversion does not resemble the normal field at all, but would rather resemble phenomena of instability and incoherence of a pseudo-hallucination. Within all these characteristics, the essential difference could be found in the respective continuity of the field. In the normal case, the field, of large size and cohesion, can form a total and unitary continuity, so that spatial references related to each other are established, and then there must necessarily be spatial congruence between a stimulus and the corresponding sensation. A different situation is that of the residual field which, restricted to a reduced sector (the periphery is excluded, i.e. the extremities, etc.), without internal cohesion (recall the independent rotations in inversion), lacks the adequate amplitude to establish sufficient continuity between spatial references and to form stable and congruent links with a stimulus. This field being thus disconnected from all support and abandoned to itself, it tends to show its primordial marginal orientation (inverted). That is to say, if the field is complete and continuous it must show spatial congruence; but if it is very reduced, there is no internal continuity and it has an idiocentric orientation, being incongruent with the outside. In short, orientation depends on the continuity of the organization.

### 26.2.3 Theories on orientation

As a complement to the above considerations, it is worthwhile to make a quick review of the theories on orientation and localization in touch, mentioning also the problem of the orientation of the visual image. According to the above, the primary factor in spatial determination (localization and orientation) lies in the cohesion and functional integrity of the sensory field, and not in diverse associative mechanisms, intersensory or not. This means a clear opposition to the empiricist associationist conceptions of space in general, whose critique was exposed in Sec. 22.4. Such conceptions had a great impact on the issue of visual image orientation, first theoretically and later experimentally (Helmholtz 1896), especially after the famous experiment of Stratton (1896, 1897) (see section 12.1 in Vol. 1). It is clear that the pathological inversion (spiral reduction) is of a primordial (nativistic) nature, totally independent of experience and without influence from other senses. In the experiments of the cited authors, especially in the one by Stratton, new values in visual orientation could be obtained due to the fact that the sensory field being intact, its continuity gave rise to a reorganization adapted to the configuration artificially promoted in the experiment. For this reason, the Stratton experiment (re-inversion by habituation to the artificially inverted visual scene with lenses) was not possible in the case of our patients, with residual field, since there was already difficulty in adapting to the conditions of ordinary vision.

According to the ideas indicated in the preceding subsection on orientation as a function of field organization, the interpretation that can then be given to the experiment of Stratton (1896, 1897) does not refer to the formation of new associations between vision and touch (visual-haptic complex), but only to the sensory reorganization provided by the central mass of a healthy brain. Such a mass would allow adaptation to the artificial inversion given by the lenses, as normally occurs for the natural inversion of the latent

marginal (inverted) action. Thus, the process would be entirely visual without any other sensory aid. Already Müller (1903, 1923) is in favor of this visual independence to change the organization in Stratton's experiment, discarding the primary necessity of visual association with touch. All this means that also for the problem of visual orientation, a mechanism similar to the one described for touch should be admitted, granting a large autonomy to the visual field in the formation by its own of spatial orientation. The visual-haptic complex would then be reduced to a totally accessory role, if its acceptance is in any way justified. A more detailed examination of similar problems, especially concerning visual orientation - which is somewhat out of place here - is reserved for another work.

#### **26.2.4 General principles in our interpretation**

Finally, given the theoretical importance of the topics addressed, it seems appropriate to add some indications on the general principles involved in our interpretation. The different phenomena of the brain dynamics presented here have been interpreted from a strictly physiological point of view based on the excitability factor, which in turn depends on the damaged central nervous mass. In this respect, the orientation disorder followed a behavior analogous to that of other functions. Even inverted vision seemed to be explained by the particular effect of ocular inversion, without having to resort to new fundamentals. But the discovery of inverted perception in other senses, and the attribution of such a phenomenon to marginal action (action of the projection area), has brought the anatomical factor (configuration of the nervous matter) into the picture. Thus, the orientation function, due to its peculiarity, is not like many other altered functions, but is of fundamental importance because it plays a decisive role in the second law (on spiral development) of the brain dynamics described here. The orientation function reveals by itself the nature of brain activity, composed of both the anatomical factor and the physiological factor (nervous excitability in relation to brain mass). Indeed, no other function could have evidenced the anatomical factor, since in the spatial functions all we find is the change of size: reduced or enlarged space, depending on the activity of the brain mass. In fact, the anatomical arrangement is manifested in a very characteristic way in the alteration of the orientation function, precisely because the anatomical arrangement is opposite to that of the physiological organization. In short, for the complete interpretation of the phenomena of sensory organization it is necessary to resort to the physiological principle of mass action together with the principle of anatomical configuration (anatomical connections), each of which corresponds respectively, in a broad sense, to the functional principle and the principle of localization.

The aforementioned principles, which are indispensable for understanding the different functions of the nervous system, intervene in different ways in the system. Thus, the anatomical principle of connections is particularly relevant in all phylogenetically older structures (spinal cord and subcortical formations in general), with a more fixed function, although without dispensing with the functional principle (remember that the isolated spinal cord becomes iterative). However, these structures have an extremely

restricted activity at the cortical level, especially centrally, and such activity only has an effect in the marginal (projection) area, as indicated when describing the various brain syndromes in relation to orientation, etc. In the central cortical mass, the anatomical connections do not play a role comparable to that of other nervous structures, so that it is possible to consider this central area as an undifferentiated (non-specific), rather plastic area, whose function is to enhance excitability and reorganize activity. This is seen both in the re-inversion that must normally occur to correct the anatomical marginal inversion and in the adaptation evidenced by the experiment of [Stratton \(1896, 1897\)](#) showing the maneuverability of the central mass, regardless of whether a visual-tactile association or our interpretation is accepted.

According to such considerations, it can be said that, whereas inverted orientation corresponds rather to a *static* spatial localization, re-inversion (correct localization), congruent with the stimulus, corresponds to a *dynamic* localization by means of the central effect of reorganization of the field, either by normal synchronism in a healthy individual or by recruitment in patients with brain lesions. We can then characterize the central action as a maneuvering mass in contrast to the primitive and anatomically fixed function of the marginal (projection) area.

Inverted orientation corresponds to a primordial orientation, which is in favor of nativism, as already said, whereas correct orientation would be in favor of empiricism, even more so in the case of re-inversion in the Stratton experiment. This antinomy is resolved by going beyond the nativism-empiricism dispute; and then, what is evident in all cases is the *correspondence* between brain organization and the structure of the sensory field.

Such a correspondence, already pointed out in dealing with the mechanism of orientation (see Fig. 151), brings our study on orientation and sensory organization to the plane of natural philosophy with the issue of psychophysical correspondence. We are facing the very remarkable result that sensory organization and brain organization are identical or superimposable: the inversion of the residual field copies the marginal organization (reduced and inverted), whereas the normal field with correct orientation is supported by a larger brain mass with possibility of physiological reorganization. Such a type of correspondence refers to a psycho-physical isomorphism. This isomorphism must be understood as equivalence of spatial structure between the sensory field and the brain action. This action constitutes a dynamic field influenced by the anatomical configuration and the maneuvering of the central mass. Then, the law of sensory organization referring to the spiral development of the sensory field (see Sec. 26.1.3) constitutes a law of psychophysical organization, which can be stated more causally and explicitly as “spiral development of the sensory field due to psychophysical isomorphism in the cerebral recruitment”.

Further discussion of all these theoretical issues will be found in a future work. For the moment, the issue of orientation has been sufficiently examined in its various consequences so as to see that it occupies a privileged place at the basis of the brain dynamics described here.

# Tactile schema

## 27 Body schema

### 27.1 Degrees of body schema

The theory of the body schema results rather confusing, regardless of the usefulness of this concept, since phenomena of very different types are mixed indistinctly. Indeed, this concept refers both to the phantom limb syndrome of amputees and to the inverse syndrome such as asomatognosia and autotopagnosia (disorientation of the own body in many different forms). The concept of body schema also refers to localization defects of the allochiria or alloesthesia type, and above all to the postural disorder (disorder in the recognition of body attitudes); finally, it is deeply related to the complex and varied apraxic and apractognosic syndromes.

The body schema concept was first formulated by [Head and Holmes \(1011/1912\)](#), supported by the rich clinical material of [Pick \(1908a, 1908b, 1915, 1922\)](#), developed by [Schilder \(1923/1935\)](#), who introduced the term body image, and by many other authors such as [Anton \(1893\)](#), [Hartmann and Schilder \(1927\)](#), [Pötzl \(1924\)](#), [Pineas \(1926\)](#), [Stockert \(1934\)](#), not always coinciding in the delimitation of the concept and much less in the nature and generation mechanism of such a function. According to [Head and Holmes \(1011/1912\)](#), the body schema is an essential function for determining the site of skin excitation on any part of the body, and especially for recognizing its position. But later, such function has been widely extended both to the spatial activity of the own body and to its connection with the external space. It has been assumed that this function is due to many different factors, such as cutaneous and also deep tactile sensitivities, visual influence, vestibular factors, motor factors (muscle tone), sensomotility, etc., all of them integrated in a superior complex of spatial structure. On the other hand, its nature has often been overloaded with psychological intellectualisms (representations, comparisons, judgments, etc.), as is usual in gnostic type functions.

For our part, taking into account the phenomena observed in our patients, providing new data (which systematize the body schema process), as well as the acceptance that there are no functional individualities per se but continuity of organization and gradual development, we distinguish several degrees in the body schema, the latter consisting essentially in the spatial tactile organization. This organization is based on the progressive differentiation of the tactile field without the need to resort to other elements of an

indeterminate nature, so there is perfect continuity between the diverse structures of the tactile sensory field. Without clamping a rigid delimitation of functional stages of the body schema, since the different activities are often intermingled, we will be guided by the following classification according to three degrees of increasing complexity in the spatial organization: corporeality or *somatic model*, *postural model*, and spatial schema or *praxis model*.

The most rudimentary degree is corporeality, i.e. *somatic model*, which only expresses how much it is related to the size and conformation of the body (soma), which implies a certain sensation of body orientation and also praxic or voluntary innervation. It has nothing to do with attitudes or kinetic form of the body, the definition being restricted to the mere static form (simple conformation of the body in its most stable aspect). The *postural model* refers to the perception of the various attitudes or postures of the body, and the localization of these postures in space. It is a more developed and subsequent degree to the simple corporeality. Finally, the spatial schema or *praxis model* mainly represents the functional structure (spatial action plan) where both the orientation about the own body and the orientation with respect to the exterior are articulated.

In a normal individual, these three main modalities (levels) are fully fused in the same activity, but in brain-injured patients, these levels become rather independent due to asynchrony, allowing a dissection of the structure of the body schema. In this situation, there is a pathological reduction of the body schema that tends to exclude the more developed stages, that is to say, it tends to convert them into preceding, simpler stages. The process of progressive reduction is then as follows: first, the structure of the spatial schema is simplified showing a reduction of the praxic behavior; next, the postural model shows more and more a character of simple corporeality by reduction to the most stable or habitual activity; subsequently, corporeality is reduced in size and conformation to finally vanish completely. A reverse process can be obtained to some extent by recruitment of the asynchronous levels, either by intensification of the stimulus (activation of the schema “from outside”) for corporeality and posture stages, or by the much more effective effect of facilitation by muscular effort (activation of the schema “from within”) for all stages of the body schema.

## 27.2 Somatic model

In the study of the simplest stage of the body schema we must pay attention to the following aspects that arise in the analysis of our cases. First, the somatic development (development of corporeality) according to the size and conformation resulting from the experiments already discussed on tactile space (Secs. 5-7). Second, the important aspect on the capability for elementary voluntary movements (praxic impulse). Third, the very special feature of facilitation, which activates the model. Finally, we shall give a brief theoretical summary.

### 27.2.1 Somatic development

In the previous chapters on tactile space and orientation we have often had to refer to the body schema in its most rudimentary manifestations of simple corporeality, this one being involved in any spatial tactile activity however simple it may be. The *somatic development* corresponding to the different phases of localization of a cutaneous stimulus is described in Sec. 21.2, also valid for deep sensitivity.

Looking at the stages of localization of a cutaneous stimulus, we see that, depending on the energy of the stimulus, a certain degree of body schema emerges, which means an activation of the soma “from the outside”. The degree to which it emerges, according to *size* and *conformation*, is shown in Fig. 110. Given that even in the most normal-like phase, there is a certain proximal deviation of tactile stimuli, especially for stimuli in the most distal areas of the extremities, there is a certain concentric reduction of the somatic model, i.e. a more or less reduced model according to the development of phase IV in spatial localization. A greater reduction in size and conformation presents stage III, with a shape corresponding to an “embryonic model”, losing also the posterior plane and other features. Finally, in phase II the simplification is so accentuated that at most a “filiform model” is produced (Fig. 99) in which all more or less defined corporeality (tactile spatial field) is close to disappearing. However, even in phase I, an extremely residual activity with no possible spatial localization persists in the aroused sensation; it is a certain influx of the body schema that determines the elongated shape of the irradiation (Fig. 106).

Likewise, several phases of corporeality are aroused with deep (joint) stimulation (activation “from within”), either in tests with joint segments (Sec. 23.4), or during initiation of walking (Sec. 25.1.4), where the size and coherence of the model grow as gait progresses. When starting to walk, as there is no corporeality or model to support the steps, there is only fusion of movements, that is, indistinct sensation of phase I or phase II, which, in addition, shows the lack of distinction between cutaneous and deep (articular) sensitivities, due to the low level of organization. Afterwards, it is already possible to distinguish movements, and finally steps and sensation of translation, due to the recruitment of the model and the emergence of spatial references. Such recruitment (somatic growth) as a function of stimulation can be measured and illustrated graphically, as shown, for example, in the curves of recruitment of localization (Fig. 113) and recruitment of orientation (Fig. 129) for a stimulus on a hand. In the case of walking, the subjective direction of the steps varies according to their perceived length and the size of the model (Sec. 25.1). Fig. 142 shows some recruitment curves in the case of walking. It can be noted in such examples that the recruitment of the somatic model, approximately of logarithmic type, is simply a recruitment of the tactile field since the proximal deviation (spatial shrinkage) is corrected. In all these cases of cutaneous or articular activation of the model, its emergence tends to be partial or fragmentary, as we already know, and the situation cannot be compared to the situation of a normal subject. Indeed, a major disturbance is the lack of continuity and cohesion of the model, whose parts are as if loose (disconnected). Other factors such as abnormal lightness of the body, flexibility, etc., contribute to the sensation of abnormality. All this has been indicated when dealing with corporeality in a general way (Sec. 23.5).

Another mode of somatic emergence to be considered corresponds to the state of natural passivity, without any particular movement nor cutaneous stimulus. When a normal subject is in this state, a diffuse sensation of body schema is produced in response to low-intensity cutaneous and articular tactile excitations. In the brain-injured patients, mainly in subject M because of his excitability deficit, any sensation of corporeality is then hardly awakened, arising only under a more intense general excitation, either directly or indirectly through facilitation by muscular effort. As for patient M, the degree of corporeality is thus related to the degree of general tactile activity. For example, when he is sitting comfortably, relaxed and completely inactive, he does not perceive any corporeal sensation, unlike the normal subject. Instead, when he is standing, he can feel a diffuse corporeality that, at most, would reach up to his knees. He does not feel the head and arms at all, the body being reduced to an indistinct sensation of the trunk and the proximal sector of the lower limbs. This is a very faint corporeality and of a size that would be at most half that of normal. This sensation does not improve no matter how long the patient stands. A fuller emergence of the somatic model can only be achieved by increased tactile activity, such as during walking, or by vigorously applying facilitation through muscular tension.

### **27.2.2 Praxic impulse**

It should be noted that the state of absolute inactivity in patient M, seated and relaxed, is difficult to achieve in most examinations, since when he is asked or given instructions, he automatically exerts, without being able to avoid it, a certain facilitation by muscular effort. It must therefore be borne in mind that there is almost always a minimum of facilitation. However, by conveniently instructing the patient about the conditions of the experiment, he can maintain the state of complete passivity and relaxation during certain tests, which entails total absence of any sensation of corporeality, as said.

An expression of the degree of corporeality is certainly the capacity to voluntarily make use of one's own body, of the *praxis innervation*, manifested in the aptitude to voluntarily move or activate any part of the body. When a state of absolute inactivity is achieved in patient M, a test of relevant importance can be performed which allows us to understand the significance of the somatic model in relation to the praxic impulse (initiation of elemental voluntary movement). We thus arrive at the remarkable and unsuspected finding that when subject M is absolutely inactive he is completely unable to perform the most insignificant voluntary movement, no matter how little energy is required, such as opening his eyes. This is verified with absolute regularity in repeated trials. This shows that if the model is absent, it is not possible to promote any voluntary action no matter how elementary it may be, resulting in akinesia or rather total apraxia, due to the absence of the most elementary orientation about his own body. Faced with such a result, it seems at first sight that the possibility of freely applying facilitation by means of muscular effort to activate the model is in contradiction with the above. However, the incoherence disappears when we consider that this type of facilitation is exercised in a diffuse way and almost as a reflex, very involuntarily, like any other automatic action of ordinary life, as will be seen.

In examining praxis in relation to the emergence of the somatic model, we first find an abolition of the praxic impulse when all corporeality is absent. This is in itself a datum of great importance. It is necessary to go a little further, to the initial limit of activation of the model under minimal facilitation, in order to “catch” the emergence of the praxic impulse. The following tests allow us to assess this process.

Patient M being in the inactive state with eyes closed is asked to open them upon hearing an acoustic signal (loud enough to be noticed even in the inactive state). The patient is allowed to use some facilitation by means of minimal muscular effort. In this situation it takes him four or five seconds to open his eyes, even though he thinks he does it instantly! But if, when receiving the signal, he is under the sustained action of the facilitation by maximum muscular effort (very intense and permanent contraction of the whole musculature), he executes the order quickly, in tenths of a second, like a normal subject. However, it is remarkable that he claims that he was equally fast in both cases (quasi-inactive and with intense facilitation), and he states this emphatically in several trials.

This latency time to perceive and activate the body varies according to the muscular effort made. However, the different objective times thus achieved, corresponding to different levels of nervous activity, are considered to be the same by the subject. Thus, when he is allowed to make a weak effort, the latency time can drop to about three seconds, although in tests performed on a different day he has taken up to nine seconds, maximum value reached in such conditions. This should be attributed to the different degree of muscular effort applied during the test rather than to a different degree of attention. Nevertheless, in this test, the change in the subject's attention is equivalent to the change in excitability due to muscular effort. When he takes nine seconds to respond, he still believes that he has executed the order quickly, as in three seconds, or in tenths of a second under maximum facilitation by strong muscular effort. He is very surprised and complains when he is told that he has needed quite a long time. Finally, when he is very inactive, he may spend a minute without opening his eyes even though he does not stop trying to comply the order. In this case, it does not seem to him that a long time has elapsed either, only about two seconds in comparison with the test under maximum facilitation.

Regarding the process of following the order, it is observed that a little before opening the eyes, slight movements in the upper eyelids may occur, as frustrated opening movements. For this, it is necessary that, upon receiving the acoustic signal, the subject applies a certain facilitation by muscular effort in the form of a reflex that involves an activation of the trunk and, weakly, of the head. At this point it is then possible to initiate movement of the eyelids. The limbs are not felt at all, i.e. the model is activated somewhat similarly to when the subject is standing, but with more tendency to include the head and lose the limbs.

Other examples of this type of voluntary movements in patient M are the following. Opening the mouth using the facilitation effect as little as possible. The patient takes three or four seconds to do this although he thinks he does it instantly. Another example is moving a finger under the same conditions. The patient takes 3.5 seconds, showing shortly before slight jerks (the referred innervations or frustrated movements) in some



other finger of the hand or in the same one that subsequently moves. The same behavior is observed for the head movement; he also takes 3.5 seconds. Larger movements such as moving a whole limb obviously lengthen the latency time, reaching seven to nine seconds. For example, lifting an arm takes six seconds, and lifting a leg and foot takes seven seconds. Also in these cases he feels that he executes the order very quickly, although he admits that perhaps he executes it less quickly than in the case of opening the eyes or other simple movements with half the latency time. When in these tests he is asked to move a very distal part, a finger, a foot, etc., the model must be activated at least up to the middle of the limb (up to the elbow or knee), and the somatic emergence may be regionally diverse. Thus, the distal area of a limb may be activated considerably while the head or other limbs are activated much less. This means that facilitation by muscular effort is directed, albeit diffusely, towards the region to be moved, as we will see later on.

It should be noted that such elementary voluntary movements may be subjectively ignored, given the weakness of both the facilitation applied (by muscular effort) and the movement performed, as well as suffering various subjective alterations of inversion, localization, etc.

Summarizing the development of the praxic impulse, i.e. of voluntary movements of elementary character, it can be said that such movements are closely related to the degree of corporeality. Particularly in the initial stage of the somatic model, the praxic impulse shows the typical features of a low functional level. These are: noticeable slowness of the process (to search for the model, to transmit the movement, etc.), movements initially frustrated by disintegration of the process, and even reduction of the subjective time of the process as it corresponds to a brain state of reduced excitability. In short, depending on the degree of corporeality, the praxic impulse can be either abolished (absence of the somatic model in a state of total inactivity), or emerge slowly and inarticulately (initiation of the somatic model under weak facilitation), or be practically the same as in a normal subject (somatic model fully activated by maximal facilitation).

### **27.2.3 How facilitation by muscular effort is exerted and its effects**

The phenomenon of facilitation by muscular effort shows important aspects in tactile functions. In relation to our study on corporeality, we must examine how such facilitation is performed and how the body schema is activated.

In view of the abnormal manifestations in the different sensory systems, it seems evident that the phenomenon of facilitation by muscular effort has spontaneously originated in tactile activity. It is clear that daily movements, both manual and of the body, already normally require diffuse muscular tension. This leads not only to improve proprioceptive function due to increased stimulation of the receptors involved, but also to improve any tactile function by central nervous summation effect. In this sense, since patient M in the inactive state has extremely impaired spatial tactile functions, these must always have been accompanied by muscular tensions of considerable intensity (tensions, contractions, movements, etc.). These tensions, although involuntary and unnoticed by the subject, are absolutely indispensable for his daily life. Even in patient T and in other brain-injured patients with less brain deficit, some degree of facilitation by muscular

effort is necessary to activate tactile space (localization, deep sensitivity, etc.). This facilitation usually consists of diffuse muscular tension or muscular jerks. (see Sec. 21.1), being also spontaneous and of unknown significance to the patient. This explains the great functional difference shown, for example, in joint sensitivity: very disturbed when examined in an inactive state by passive tests, and instead quite functional in spontaneous voluntary activity. It is then understandable that in the long run, a certain degree of facilitation by muscular effort is automatically associated with all types of tactile activity, thus considerably improving such a fundamental sense for the organism as touch.

Facilitation by muscular effort has originated mainly in the tactile system, and has been extended by applying it to the other sensory systems since they normally work together. However, facilitation by muscular effort is much more necessary for touch than for other senses such as vision or hearing. The latter are already partially compensated in their brain deficit in a simple and natural way by the summation effect provided by the pair of organs, and are not as impaired in their activity as might otherwise be expected. It can then be said that patient M and patient T automatically exert some degree of muscular facilitation to any type of sensory activity, having developed an unconscious habit over time after suffering the brain injury. This would also apply to the Schneider case of [Goldstein and Gelb \(1918, 1919\)](#). However, the subjects being unaware of the application of this type of facilitation and even more of its nervous summation effect, they were unable to take advantage of its voluntary application and its significant maximum effect, until this phenomenon was discovered. As already mentioned (Sec. 1.3.2 in Vol. 1), the phenomenon of facilitation by muscular effort was discovered in 1939 after persevering research. Since then, especially patient M benefited extraordinarily from the voluntary application of facilitation by means of maximal contraction of the entire musculature. Thus, his sensory horizons were instantly broadened in all kinds of activities, achieving a very considerable correction of the defect.

The application of the facilitating muscular effort occurs, as already indicated, almost as a reflex as soon as some sensory activity arises, therefore the state of inactivity in the tests is difficult to achieve, and a proper instruction of the subject is required to achieve complete relaxation and passivity. It is at this point when the useful time of subject M in the different tests of sensory excitability with adequate stimulus achieves up to about seven seconds. The loss of the somatic model, absence of corporeality, does not seem to be an impediment to exert muscular tension since, as observed and as the patient states, he performs a diffuse muscular tension which, since it is not localized, does not need to be guided by the somatic model. This is a very different situation from that of elementary movements associated with praxis, where the movement is specifically chosen. In conclusion, the application of facilitation by muscular effort, whether involuntary or conscious, arises as an undifferentiated action impulse that provokes a diffuse muscular tension of the body without the need to be guided by the somatic model.

It should be noted that although the execution of facilitation by muscular effort is rather diffuse, the site of origin is in the trunk musculature, as evidenced by observation of the patient. There are several reasons for this preference. First, the trunk is the central area of the body, and as such, acts as a support for all haptic activity. Second, it is a very undifferentiated region in terms of touch and therefore lends itself to diffuse activities.

Finally, it is more resistant to dynamic reduction in the fading of the body schema, since, being a central region, its survival is possible on many occasions. It is also plausible that the area of spontaneous thoracic breathing movements is an attraction to more or less automatically exert facilitation by muscular effort. Indeed, it is very common that when subject M voluntarily applies facilitation, he starts mainly by making an effort with the breathing musculature (thoracic muscles and abdominal pressure). It is possible to perceive his suffocation produced in such a situation, especially when asked to apply a strong muscular effort of maximum effect. These conditions occur especially when the subject is seated and totally inactive. When he is standing and needs muscular effort to increase any sensory activity, he tenses strongly the musculature of the legs and thighs, since when standing, this region emerges sufficiently in the body schema, and thus hides the muscular tension from the sight of other people. The patient acts in this way from the time he is aware of the effect of this type of facilitation.

Another issue to be studied is the type of “corporal activation” according to the amount of facilitating muscular effort. Within the trunk there may be muscular contractions and tensions of varying intensity which, at best, only cause a partial activation of the somatic model. Thus, in maximum muscular contraction of the trunk due to strong tension of the thoracic and abdominal musculature, the model emerges only up to approximately the knees and elbows, although the head is also usually activated by the tension of the neck. This type of facilitation is the one usually used by patient M when he is asked to apply it in tests, and the one he also usually applies in his daily life if he has to overcome difficulties in perception, comprehension, etc. But it is possible to achieve a greater effect by adding a strong tension of the limb musculature, thus obtaining the maximum limit of muscular effort.

It is very remarkable the fact that in order to clench the fist strongly, he must begin by contracting the trunk, and next the whole corresponding upper limb. It seems that it is absolutely necessary to maintain the same muscular tension in the trunk and limb as in the fist. Thus, if he is asked to continue clenching his fist tightly but relaxing the trunk, he is completely unable to do so, and by relaxing the trunk the hand loses all its strength. It seems natural that the trunk can be innervated without the limb, but the reciprocal seems impossible.

As has been said, no matter how considerable the muscular tension of the trunk (central area of the body) is, the activation of the somatic model does not reach the more distal areas of the limbs, and it is necessary to distribute the muscular tension more peripherally to make them emerge in the model. Various tests on how to activate (to perceive) the hand show the need to contract the entire musculature of the limb, with the help of the trunk, and also to clench the fist strongly. Even so, it seems that the distal half of the last phalanx may be excluded, according to tests with tactile stimuli at the fingertip that are perceived with some proximal deviation. If instead of closing the fist, the open hand is tensed, the activation is less complete and only reaches up to half of the fingers. In the feet, by making an intense contraction of the toes, the activation of the model is likely to reach the beginning of the toes. This emergence of the somatic model is the same as the one reached iteratively when walking, after many steps (see Sec. 25.1.4).

Other consequences of facilitation by muscular effort will be discussed when dealing with praxia, manipulations, etc.

#### 27.2.4 Theoretical summary

Making a theoretical summary on the somatic model (corporeality), we can say that through asynchrony and therefore dynamic reduction, the process of emergence of the model directly shows the level of organization of the somatic field (tactile field).

Somatic emergence is determined by its size and, in part, by its configuration which is linked to the postural model, as will be seen further on. Such emergence is restricted to the central zones of the body in the most rudimentary phases, and subsequently expands following a process that is entirely identical to the recruitment of the spatial tactile field. Thus, reduction of the somatic model is nothing more than a concentric reduction of the tactile field. The localization of stimuli, either cutaneous or articular, is therefore the most direct expression of the development of the model, as pointed out by [Head and Holmes \(1911/1912\)](#) when dealing with the body schema. As for the shape of the model, a certain general shape tends to be preserved at all stages of model development, even in the irradiation band without any possible localization.

Both cutaneous and deep (joint) sensitivities contribute to the formation of somatic corporeality, however these sensitivities are only distinguished as such if there is a certain degree of development and organization of the model, otherwise they are perceived in an indistinct and diffuse way.

A very low or moderately developed level of organization also serves to address the nature of other properties of the somatic model, such as orientation and belongingness of one's own body, linked to the elementary praxis activity. This type of activity is completely disabled in absence of corporeality; and in stages of very moderate activation of the model, such activity becomes defective, slow and disjointed. The remarkable subjective perception of the reduction in the duration time of the processes is another sign of the low functional level.

The muscular effort that activates corporeality, that is, that brings out the somatic model, does not resemble the praxis activity, since the former corresponds to an instinctive impulse carried out in a diffuse way, mainly in the trunk, without choice nor local specificity.

In short, the disorder affecting the emergence and consolidation of the somatic model is due to an increase in the sensory threshold and to disorganization, and would correspond more or less to asomatognosia. This represents the highest degree of disturbance in the body schema and is one of the various stages that can be delimited in our cases depending on the conditions of the test.

It should be noted that the term asomatognosia was used by [Lhermitte and Trelles \(1933\)](#) and [Lhermitte \(1939\)](#) precisely to denote the total fading of the body image or body sensation in the cases of [Foerster \(1903\)](#) and [Deny and Camus \(1905\)](#). These cases were considered rather of a psychiatric type by the respective authors, although these cases undoubtedly presented alteration of postural sensitivity, for example, and had shown a very typical symptomatology of absence of any somatic corporeality. It should

be noted that when we use the term *asomatognosia* for our cases, especially *M*, we simply refer to the loss of corporeality by abolition of the tactile (somatic) field, discarding here hypotheses and more complex conceptions of other authors in this regard.

## **27.3 Postural model**

The next degree to the somatic model is the postural model, which refers to the perception of the different attitudes of the body. Postures (position sense) play a major role in body schema theory. Thus, for [Head and Holmes \(1911/1912\)](#), the concept of body schema arises as support for the attitudes of the body. Also in our study of the body schema, postures play a prominent role and provide new empirical data. Postures do not involve a different situation from what has been explained so far about the schema, but rather they are only a further stage in the tactile spatial organization.

Although the interest is focused on the study of segmental postures, for a more complete and systematic study of the postural model, we shall address other aspects of postures, dealing successively with: postures in relation to spatial localization of cutaneous stimuli, general body posture, segmental postures, and finally, theory of the postural model.

### **27.3.1 Localization of tactile stimulus and postural model**

It is clear that for a correct localization of a tactile stimulus on a limb displaced from its natural position, it is necessary to have a sense of attitude, i.e. localization must occur on the postural model. If due to the circumstances of the pathological case, or to the test conditions, the perception of posture is abolished (as may occur in subject *M*), the localization of the stimulus is possible to some extent in a diffuse corporeality (somatic model) but not in relation to the surrounding space. Two levels of localization can be distinguished. The simplest level corresponds merely to the site on the body, and statically abides to the somatic model, neutral in postural terms. The other more developed level of localization corresponds to an absolute spatial placement by intervention of the postural sensory model (not to be confused with the postural motor function mentioned in Sec. 25.2.4). However, this classification into two levels of localization does not mean a complete independence between them, as they are degrees of the same organizational process that varies as a whole. That is, if there is a deficit in the schema, the somatic model and the postural model suffer alterations both at the same time, but the latter to a greater extent. This is analogous to what happens in other functions, as mentioned in different sections. Hence, if postural localization is abolished, the stimulus will not be perceived somatically at the exact location of stimulus application but will be perceived with some proximal deviation.

These conclusions about modes of localization and about schema are deduced from the examination of our brain-injured patients. It is clear that localization under residual sensory field conditions (localization with strong proximal deviation, inversion, etc.)

corresponds only to a more or less reduced somatic model. However, the levels of organization, although considerably out of synchronization, can be recruited by intensifying the stimulation. This recruitment is “from outside”, i.e., by the action of stimuli. Thus, a painful pinprick that can markedly reduce proximal deviation in patient M inactive, also produces postural activation of the corresponding limb, albeit in an incomplete and diffuse manner. Indeed, an intense prick on a finger can produce a very little deviated cutaneous localization and at the same time awaken more or less the perception of the attitude of that finger, but it is not able to provide a general activation of the whole limb. In fact, the activation reaches at most only up to the wrist in this case. Instead, if the prick is slight and the corresponding sensation stays in very elementary spatial phases, no postural activation is possible and only a certain diffuse corporeality is awakened. In general, it can be stated that when somatic localization is rather good, attitude (posture) emerges, but only for the stimulated segment, either an arm or a finger (see segmental postures in Sec. 27.3.3 below).

In short, segmental posture can be activated by recruitment under intense cutaneous stimulation. In this way, the somatic model evolves to a certain extent to the postural model, thus almost completely overcoming the asynchrony between both models, and therefore achieving the localization of the stimulus in space.

### **27.3.2 General posture of the body**

The *general posture* of the body deserves a separate study. Such a posture is somewhat different from segmental postures which will be discussed in the next section. The issue of the general posture of the model arises in our study because of the phenomenon of orientation inversion in tactile space. As we know, such inversion also appears in the body schema (see Sec. 24.4).

According to what has been said about the alteration of orientation in the body schema (Sec. 24.4), the subjective rotation of the body felt by the patient constitutes a *deviation of the somatic model*, i.e. an alteration of the verticality and the normal position of the body as a whole. This alteration connects the stage of the somatic model with that of the postural model, perhaps closer to the former than to the latter. Indeed, the subjective rightward tilt of the somatic model in the form of a spiral turn of the body, when patient M is standing or sitting under weak facilitation by muscular effort, is more pronounced the more reduced the somatic model is (Figs. 137 and 138). This occurs up to a certain point where it is not possible to feel any position of the body as a whole due to the vanishing of the model. This subjective deviation of the somatic model thus corresponds to a functional level at which segmental postures can hardly be felt. That is to say, it is almost impossible for the postural model, in its common meaning, to arise in such a situation. Trying to link the different phenomena (since everything refers to sensory posture), the *general posture* of the body can be considered as the first step in the development of a postural model, model that finally reaches its greatest differentiation with the segmental postures.

Accepting the above, it can be stated that the postural model comes from the somatic model, since the general posture refers to the position and orientation of the somatic model in space. This orientation depends on the state of the local spatial sign which, due to the inversion process in our patient, tends to produce a rotation (tilt) of the model. Specifically, the postural model derives from spatial localization, and thus, an uninterrupted line of phenomena can be established between stimuli localization and postures; *posture is localization*. Once again we find continuity in the organization. Thus, posture, generally considered as an intellectual or associative phenomenon, is merely an extension of localization or of the local sign. It means that posture is established in a simpler and more direct way.

Certain alterations of the *general* posture of the body have been described in some special cases involving “lack of perception by left hemiplegia”. Thus, in one case of [Kramer \(1915\)](#), two cases of [Pötzl \(1924\)](#), others of [Stockert \(1934\)](#), etc., there was a subjective sensation of certain rotation of the affected half of the body towards the healthy side. The case of Kramer also had allochiria or tactile alloesthesia (mentioned in section 10.1.1), but not those of Pötzl. The cases of Stockert presented different complications of the body schema, and in addition to the rotation of the model towards the healthy side, they had alterations of localization and orientation in vision, proximal deviation in touch, and auditory alloesthesia when stimulating the ear of the hemiplegic side. From our point of view, the clinical descriptions of such cases are insufficient to establish that the rotation of the somatic model is the result of an inversion process, and for the moment, without going further into these cases, we merely cite them. Other more or less clear cases of deviation of the model correspond to induced manifestations on the body schema, as in the cases with cerebellar and frontal lesions described by [Goldstein \(1926\)](#) and others, as already mentioned in Sec. 25.2.4.

### 27.3.3 Segmental postures

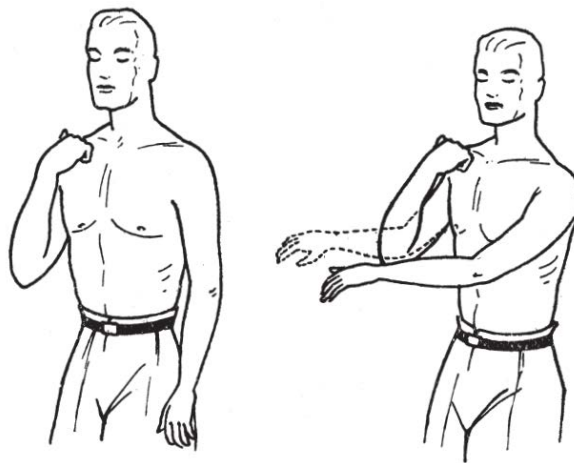
With the study of segmental postures we enter fully into the specific activity of the postural model and into one of the most characteristic manifestations of the body schema. Attitude recognition is severely disturbed in our brain-injured patients in the usual state of activity. In general, this ability is always one of the most disturbed functions in cases of cortical involvement of tactile sensitivity, as was first pointed out by [Head and Holmes \(1911/1912\)](#) and [Head and Riddoch \(1920\)](#). Many authors have confirmed this later, and we have been able to verify it in a large number of brain-injured subjects.

As mentioned above, the postural model comes from the somatic model, and the general posture of the body becomes the initial postural activity from which local or segmental attitudes are derived by differentiation. The relationship of the postural model with localization has also been indicated, as well as the role that a segmental posture plays in the correct localization of tactile stimuli, with the possibility of activation of such posture by intensification of such tactile stimuli (Sec. 27.3.1). However, in the usual recognition of postures, recognition is not activated “from outside” but “from within” by

the proper organization of tactile sensitivities, particularly the deep one, up to the “figure” level.

The pathological degradation of segmental postures clearly reveals the nature of the process. Thus, within the parallelism between localization and posture, it is found that in both localization and posture the spatial perturbation is of the same nature, i.e., a proximal deviation. That is, in tactile localization there is deviation towards the midline, and in posture, deviation towards the habitual position of the body (somatic configuration), i.e. towards the *neutral posture*. In short, in the first case, somatic shrinkage is produced, and in the second, postural shrinkage, in such a way that the change in attitudes tends to be neutralized or underestimated.

As early as 1938, long before the discovery of the phenomena of dynamic action excitability (facilitation by muscular effort, etc.), patient M was examined in his habitual state, corresponding to a weak or moderate facilitation by muscular effort. It was then evident that neutral positions predominated in many tests. For example, in the following test, first the forearm is passively flexed over the arm until his fist reaches his shoulder (Fig. 152 left), and he is asked to imitate that movement with the other arm. In trying to do so, he tends to raise it stretched forward (Fig. 152 on the right), placing it horizontally; that is, he places it in an intermediate position between the habitual one (hanging inert next to his body) and the fully flexed one that he must imitate, thus showing a deviation towards the neutral posture.

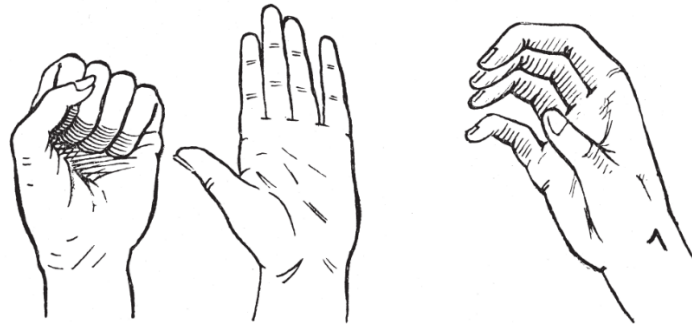


**Figure 152.** Typical phenomenon of postural deviation towards the neutral posture. The passively induced attitude of the right upper limb (left drawing) is perceived, by means of some facilitation, as very deviated towards its most habitual posture; that is, the complete flexion is perceived as a weak flexion, the fist being forward (dotted line in the drawing on the right). Thus, in the active imitation with the other limb, this deviation is displayed (drawing on the right), objectifying the subjective postural deviation.

Likewise, if keeping one hand completely open, even by his own voluntary movement, he is asked to imitate that attitude with the other hand with eyes closed, the second hand adopts a semi-flexed neutral posture corresponding to the inert attitude of the hand (Fig. 153). On opening his eyes, he is surprised by the difference. If what he has to imitate is a clenched fist, the resulting attitude is likewise a semi-flexed hand (Fig.



153). Already before the discovery of the facilitation by muscular effort, the patient needed to tense the hand, open or closed, in order not to make mistakes. Otherwise only a diffuse perception was possible, and the usual semi-flexed attitude was predominant.



**Figure 153.** Phenomena of postural deviation to the neutral position. On the left, closed fist and completely open hand. On the right, imitation of both positions with the hand semi-flexed (neutral position of the inert hand).

At present, patient M exhibits similar behavior in ordinary circumstances. In the inactive state, he is completely unaware of posture since, as we know, the somatic model does not provide any sensation in that state. However, he maintains the tendency to respond with a neutral position rather than manifesting his inability to perceive a change of attitude in his limbs. For example, with the hands hanging naturally on both sides of the body, if one of them is passively raised, he still believes that both are in the same position, even if he applies some diffuse facilitation. However, when asked insistently, he ends up answering that he does not know for sure but that he thinks they are fine. This postural illusion disappears when facilitation by strong muscular effort is applied. But if the effort is not very intense, several errors may occur, both by deviation to the neutral position and by defects corresponding to the low spatial level (centripetal deviation, inversion, etc.). In order to perceive postural changes in very distal regions, such as the fingers, facilitation must be applied in a generalized manner with the muscular tension being more intense than in the case of asking the patient to voluntarily move a finger.

The most direct tests are those in which the patient describes the posture felt, a posture that has been produced passively. Depending on the intensity of the facilitation applied and the type of posture, the following results are obtained in patient M.

A test that may be useful for examining general posture is head posture as well as trunk postures, as these are midline regions. The head is passively placed backwards (the subject ignores the maneuver because he is in inactive state). The patient is asked to moderately contract the musculature of the body (weak facilitation); then, after five or six seconds he gives notice of the perceived posture, explaining that the head is slightly forward. Due to the low functional level reached, the position (localization) is inverted and with a small deviation from the vertical, since this phase corresponds to a shrunken somatic model.

A test on complex segmental posture is as follows. The forearm is passively placed in flexion over the chest. After five seconds of diffuse muscular effort (weak facilitation), the patient feels the posture and describes it as the forearm somewhat flexed, but not on the chest but somewhat separated from it. He tends to give as a response an intermediate posture between the neutral posture and the passively established posture, showing the phenomenon of deviation towards the neutral position. There is thus a sensory reduction in posture.

A test of differentiation in simple segmental posture is as follows. The thumb is passively maintained in extension, the other fingers in semi-flexion in an inert position. After five seconds with some facilitation by muscular effort he states that all fingers of the hand are more or less extended. Thus, there is a postural differentiation deficit, since in diffuse perception the position of the thumb is generalized to the other fingers.

Under intense facilitation by strong muscular effort, the errors of appreciation disappear or are considerably reduced in all these cases, although some errors may persist in very complex tests.

Another mode of examination corresponds to the usual clinical test in which one limb actively imitates the passively produced posture of the symmetrical limb. Similar results to the previous ones are obtained, as indicated below.

Imitation of the left hand placed on the left parietal: after seven seconds he places his right hand in front of his head, almost at the same height as the other hand. With a very intense facilitation, he performs the appropriate correction.

Imitation of two extended fingers (index and little finger) of his left hand: he extends all the fingers of his right hand. With intense facilitation he corrects with some difficulty, but he is able to distinguish the two fingers from the others.

In these tests it is easily observed that postural imitation produces some deviation towards the neutral position, albeit slight, since the imitation does not complete the full path despite the use of a fairly intense, although not maximal, facilitation. Given the tactile alteration for the whole body in the central syndrome, postural imitation in the ordinary state of the patients is easily altered, as seen in Figs. 152 and 153. Both the attitude of the passive model limb and that of the active limb that has to move the patient suffer a sensory reduction. However, it can be admitted that the main defect comes from the passive limb, since the other is more activated by the facilitation. In short, a very intense facilitation is needed in patient M to overcome all defects. But this is not always achieved in very complex attitudes, being in these cases necessary for him to open his eyes to perceive a postural discrepancy in the tested limbs.

#### **27.3.4 Theory of the postural model**

The theory of posture was initiated by [Head and Holmes \(1911/1912\)](#) who introduced the notion of body schema as something plastic and hypothetical to which to refer changes in body attitude, which in reality are produced by the postural model. This theory also emphasizes that the perception of position and passive movements depends on the ability to relate perceptions to each other, which is accomplished through the plastic function of schema. It also points out that for the localization of cutaneous stimuli, another type of

schema such as a model of the body surface is required. Such a classification corresponds to what we have explained here about the somatic model (for localization) and the postural model (for attitudes), but these being conceived in a much simpler sense than that expressed by [Head and Holmes \(1911/1912\)](#), since we are dealing purely and simply with tactile spatial organization without sensory influences other than touch. Thus, in contrast to the emphasis on the ability to compare or relate perceptions to each other, in our conception, phenomena are rather a direct expression of the state of the sensory field (degree of organization, spatial dimension, etc.).

As for the various sensory influences, there is an old opinion about the collaboration of visual images to the sense of position. This opinion is also shared by [Head and Holmes \(1911/1912\)](#), but becomes more prominent after the interpretations of the Schneider case by [Goldstein and Gelb \(1919\)](#). These authors think that the whole disorder of tactile space is due to the loss of visual influence on touch. This point of view was later adopted by [Schilder \(1923/1935\)](#) to support his study on the body schema. This issue has already been discussed in Sec. 22.4, devoted to the theory of tactile localization and spatial organization. The explanation of the visual influence in the Schneider case has been discarded in the first part of this book, when exposing the central syndrome (Sec. 1.4 in Vol. 1). As for localization, it has been shown that it depends on the state of the tactile sensory field as a direct expression of neural organization (localization phases according to neural mass, decussations, etc.). There is thus a simple interpretation for tactile localization of stimuli, and something analogous is valid for passive movements and postures since posture can be understood as localization. About this we have said in Sec. 27.3.1 how posture can be recruited by intensification of a skin stimulus in the same way that proximal deviation is overcome in the somatic model. We have also indicated (in 11.3.2) that the subjective lateral deviation (rotation) experienced by the general posture of the body is due to the effect of inversion of the local sign. Finally, it was indicated in Sec. 27.3.3 that segmental postures are pathologically reduced in the sense that they are perceived to be closer to the habitual (neutral) positions. This reduction runs parallel to the proximal deviation in the somatic model. In conclusion, considering posture as localization is fully justified by the observations.

It should be noted that in the detailed studies of [Goldstein and Gelb \(1919\)](#) on the Schneider case nothing is mentioned about the phenomenon described here on the subjective postural deviation towards the neutral position. Nor is it mentioned in the meticulous observations of [Head and Holmes \(1911/1912\)](#). In all these authors the concept (and facts) about degrees of disturbance of a given function seems to be completely absent. This concept plays instead a fundamental role (asynchrony, new phases, abnormal steps, etc.) in the brain dynamics presented here, as we have repeatedly shown.

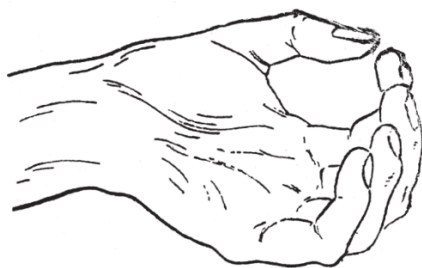
According to [Goldstein and Gelb \(1919\)](#) the Schneider patient was not aware of the position of a given limb as long as he did not move it. When he was holding the arm in a horizontal position, he tried to get a clear idea of its position by oscillatory movements of the shoulder joints, then of the elbow joints, and then of the whole body. He recognized postures by their critical points (*ausgezeichneten Stellungen* according to these authors); for example, in the fact that the elbow can no longer move in a certain direction. When he was in a horizontal position, he

recognized it by the pressure he felt on his back. When he was in a vertical position, he recognized it by the pressure on the soles of his feet and a certain sensation in his knees. He did not perceive deviations from the horizontal up to 45°, and he considered this deviation as horizontal. He was able to imitate the posture of a limb with the symmetrical limb when the positions were critical and by moving both limbs.

Postures were thus “recognized” by means of contrivances and in a very indirect way. Although the patient could seem to behave normally by being able to make movements or muscle jerks, the authors claim that he was not able to perceive the posture as such, but only to have some idea by means of indirect processes. As already indicated, in our opinion, muscle jerks constitute a facilitation of nervous summation in the nervous centers that improves the general level of excitability. In addition, they intensify joint proprioception, as has been observed in many brain-injured patients and even in a normal subject after a long rest. Therefore, the Schneider patient, to the extent that he achieves facilitation according to the intensity of movements and muscular tension, is able to perceive the posture directly. The same occurs in subject M with a greater brain injury.

Considering more specifically the phenomenon described here of postural deviation towards the neutral position, we have a very remarkable precedent in the normal individual under certain conditions. It does not invalidate this pathological phenomenon, but rather gives it greater support. In a normal subject, neutral body attitudes exert a great influence on spatial sensation and even cause sensory confusions. It is a topic that comes from [Aristoteles \(384-322 BC\)](#), and is developed especially by a series of authors such as [Czermak \(1852\)](#), [Henri \(1900\)](#), [Kramer \(1915\)](#), [Tastevin \(1937\)](#), [Ponzo \(1910\)](#), [Rupp \(1912\)](#), and mainly [Spearman \(1907\)](#) and [Skramlik \(1937\)](#) among others. The influence of the natural positions of the fingers explains the well-known ‘Aristotle’s illusion’ and the so-called ‘Japanese illusion’ (error when moving a finger having the hands crossed and the sides changed), as well as other illusions. Thus, a stimulus on deviated lips tends to be felt in normal position although not completely. The observations of greatest significance for our purpose are the following. According to [Henri \(1900\)](#), there is a tendency for the position of the excited arm to be perceived as too low, with preference for a position close to the body. [Kramer \(1915\)](#) also indicates that by placing one arm in a symmetrical position with respect to the other, there is an inclination towards a more comfortable or habitual position. Mainly [Spearman \(1907\)](#), studying normal illusions, finds that there is some proximal deviation with shortening of the arm as well as an illusion about the angle which is perceived as smaller. This illusion increases notably when the joint is at rest. This is also pointed out by [Rupp \(1912\)](#) when referring to the tendency to prefer the closest positions to the body. As [Skramlik \(1937\)](#) states, every habitual (neutral) posture acts sensorially so intensely that deviated postures are perceived as being close to the neutral posture, and are thus underestimated. For example, different tests with the hand prove that the perceived postural deviation is toward the semi-flexed position of the fingers, i.e., to the neutral posture resulting from inert muscle tone (Fig. 154). It should be noted that in the normal subject such postural illusions occur mostly under certain conditions, such as attention focused in a particular way, a long period of rest, etc. In a long period of rest, joint positions do not produce any appreciable sensation and then the image of the neutral position predominates. Sensory illusions disappear when moving the joints, as for example in the finger-crossing test. Thus, postural illusions in the normal subject appear easily due to a certain diffuse perception, thus giving rise to the

neutral posture, which corresponds to the most frequent configuration of the body by virtue of its architecture and muscle tone.



**Figure 154.** Normal hand position according to Skramlik (1937). When the perception of the hand attitude is very diffuse, this habitual (neutral) position tends to be perceived. See Fig. 153.

Thus, it is observed that the difference between the pathological cases and a normal individual is only quantitative. Whereas in the normal there is a certain deviation under special conditions, in the pathological cases the predominance of the neutral position in perception is persistent and very noticeable. The more diffuse and unstable the tactile function is, the more the neutral posture is manifested in perception. Thus, when the sensation of touch is about to disappear, any type of induced posture is perceived as a neutral position. As we have seen in patient M, when there is no perception of the passively acquired posture, he responds according to the neutral posture, which, in addition, implies a certain unawareness of his defect. When the perception of the posture is incomplete, the response is deviated towards the neutral posture. This process of deviation towards neutral posture is part of the reduction mechanism in the different activities of form perception. We have already seen that figures drawn on the skin tend to be perceived as deformed along the longitudinal axis of the limb (Fig. 122), or simplified to more stable figures (Fig. 123). The latter is more clearly demonstrated in the visual system with respect to visual forms (metamorphopsic pseudoagnosia, see Sec. 11.3 in Vol. 1), and in different schematic drawings (Figs. 73-78) that give rise to a greater sensory illusion the greater the perceptual instability.

In conclusion, posture is considered a type of localization. It develops from the general posture of the body to the segmental postures. Pathologically the perception of posture is not suppressed at once but presents, like all functions, degrees of reduction that are manifested in the degree of postural deviation towards the neutral position, giving rise to the corresponding underestimation. This conception is very simple and consistent with the postural mechanism in a normal individual. Such reduction tends to convert the postural model into the somatic model, since the latter constitutes a static configuration and the postural model involves an increase in the spatial organization of the somatic model. There is a reduction of the schema that first affects the postural model and then continues with the somatic model according to the degree of organization of the sensory field.

## 27.4 Praxis model

Voluntary movements are linked to the body schema both in motor initiation and in the subsequent development of the action. Thus, praxis obeys an organizational plan derived from the body schema, justifying the term 'praxis model' used here.

Such a conception, which is now very essential, already existed more or less in many authors, such as Schilder (1923/1935), Grünbaum (1930), Conrad (1933), Lhermitte and Trelles (1933), Lhermitte (1939), etc. Earlier classical authors on apraxia such as Liepmann (1900, 1920) or Pick (1905) could also adhere to such a concept if the notion of body schema had existed at that time. Schilder considers that apraxia depends on a deficit in the evaluation of the body schema, and Lhermitte thinks similarly. In Grünbaum and Conrad we find complementary concepts. According to Grünbaum, handling tools or utensils is equivalent to an extension of the body schema, whereas for Conrad, the concept of body schema involves both the conceived space and the space of action. Similarly, Bogaert (1934) interprets the postural model as active, extending it to praxis activity.

In our study, the concept of praxis model becomes necessary because the action plan runs parallel to the body schema in its various degrees, until the normal praxis behavior is reached by virtue of the corresponding reorganizations. Given the nature of the praxis model, in its pathological reduction, praxis behavior is simplified becoming diffuse and discontinuous, the coupling with the environment becoming disrupted. In severe alteration, phenomena of previous stages are manifested, appearing a great disorganization of movements as well as a series of incongruities due to the effect of somatic inversion.

The purpose here is not to study the classical apraxia phenomena or their variants, but to study peculiar disorders in relation to the alteration of the body schema, according to different functional levels that show us the internal structure of action. To this end, the following issues will be addressed: dyspraxia in rudimentary model, dyspraxia in the coupling of the model with external space, and dyspraxia due to instability of the model. Finally, a theoretical summary on general aspects will be given.

### 27.4.1 Dyspraxia in rudimentary model

It can be stated in general that actions are derived from the spatial orientation provided by the body schema, and thus, praxis model means to a large extent motor orientation. Especially in the most elementary phases, such as the first motor attempts, orientation with respect to the body acquires a major significance. Therefore, we must take up again certain aspects of the already studied phases of the body schema.

#### 27.4.1. a) *Asomatognosia and motor action*

As we know, in the inactive state of subject M, there is an effective abolition of corporeality (asomatognosia), and of course also of all orientation and capacity for voluntary selective movements (see Sec. 27.2.2). Such a deficit is hardly perceived by the subject, so we can consider that there is anosognosia, i. e. lack of perception of his deficit, as is the general rule for the various disorders in our brain-injured patients. In such

conditions, it seems that this deficit is replaced with a diffuse sensation of the neutral model, as mentioned above. That is, the illusion of the neutral postural model dominates, giving rise to a pseudognosis regarding the orientation of the own body. Similar to the appearance of a phantom limb in amputees, a whole phantom body, or at least an illusory model, now appears.

In these circumstances, patient M makes all kinds of orientation errors with respect to himself. For example, he is asked to find with one hand the other hand that had been passively placed at the level of the head. He then directs the active hand towards the habitual (neutral) position of the other hand, and not finding it, he tries it in the vicinity, and in view of the failure, he looks for the elbow which he does not find either, and so he goes on until he reaches the shoulder, bumping into the observer's hand which he mistakes for his own and he tries to grasp it being fully convinced that it is his own. Also, with his own hand on his head he is passively scratched until he is able to perceive localization (phase II); then he says that someone is touching his head. If he applies facilitation by maximal muscular effort, he immediately becomes aware of the confusions.

In the test of one hand seeking the other, the action is rather *automatic* and not a selective movement in response to a specific request. For this reason, the action occurs in the absence of an effective model, without the need of activation through facilitation by muscular effort. Automatic action and anosognosia occur at the same functional level of undifferentiation of the body schema, neither the anomalous position of the limb nor any other type of corporeality being perceived. If the observer's head comes between the head of the subject under examination and his actively seeking hand, this head is taken as his own, as is the case with the alien hand as mentioned above. In short, automatic action is guided by the illusion of the neutral postural model according to the state of the rudimentary model.

In connection with the above, it is worth mentioning the very frequent tendency in various tests to maintain the postures that have been given to the limbs no matter how painful they may be, and to believe that all is well. There is a certain postural perseveration, which is unnoticed by the subject due to the lack of sensory control. However, pain arises with fatigue, and this can lead to the perception of various postures.

We have been able to observe this type of disorientation with respect to the own body in countless war brain injuries, and also in civilian cases due to diverse lesions (vascular, tumoral, etc.). In these cases, the subjects were cognitively unaware of their own defect. Most of our cases examined during the Spanish Civil War (1936-1939) had parietal lesions causing contralateral hemiparesis and abolition of spatial and deep sensitivity on the same side. Many of them had a spontaneous sensation of "missing" limb, more rarely of "foreign" limb or of total absence of the involved side. Most of them confused the observer's limbs with their own in the tests, and even failed to visually recognize their own hand (the affected one). One case presented the illusion of believing that the totally paralyzed limb was moving. In tests to find the affected limb, passively hidden, some of these subjects always searched for the limb (or hand) by going down from the shoulder on the same side. In the most severe cases, the subjects did not search systematically, or had little conviction when encountering the member. Other subjects were not even able to look for their own hand, etc. Ranking the cases according to their behavior in these tests, from least to most impairment, we obtain the following: 1, the patient moves the limb to activate the internal perception; 2, he searches systematically; 3, he does not search in an organized way, or has little

conviction when he encounters the limb; 4, he does not know how to search for the hand, or does not even try to, he moves the hand without being aware of it.

In connection with the above, a subject injured in the parietal area, examined in 1938, with right hemiparesis and hemihypoesthesia, presented phenomena of special interest, as follows. Hemihypoesthesia affected preferentially, as expected, spatial functions, mainly in the distal part of the upper limb. In the hand, the simultaneous Weber was abolished for any separation, whereas the successive Weber was preserved, although very enlarged (5 to 8 cm). Joint movement was rarely felt, and only in the shoulder. When the arm was passively moved, he perceived only a static contact in the shoulder. In the localization of skin stimuli, he made many errors in *naming* the site, and usually with proximal deviation. In *pointing* with the healthy hand he made the same type of errors, perhaps with greater proximal deviation, and often pointed diffusely from the air (see Sec. 21.1). Particularly noteworthy for the matter at hand is that if the affected limb was stimulated and then moved from its habitual position, he tended to point to the habitual position since the subject had no perception of the change. In other words, he localized according to the more or less preserved somatic model but not in relation to the postural model. Thus, having his hand next to its corresponding shoulder, when stimulating each of these parts separately, shoulder or hand, the *verbal* response was correct, but if he *pointed* to the place touched, he did well for the shoulder but very incorrectly for the hand. For the hand, he pointed from the air toward the natural (neutral) place of the hand. That is, as in the previous test, he localized according to the neutral posture. Such a condition of the patient, of course disturbed his orientation with respect to his own body. If the affected hand was displaced, he was looking for it irregularly all over the body and in the air, although he did not confuse it with a foreign hand. After many attempts, he used to go to the shoulder, and going down the arm he was able to reach the corresponding hand. He couldn't feel the hand at all, lacking any spontaneous sensation from the shoulder down. When he was asked about the hand, he did not know where it was. If it was in front of his eyes he was able to recognize it correctly although not very quickly. In bed he had the feeling that his arm was missing.

These cases and similar ones described by [Stockert \(1934\)](#), [Hoff and Pötzl \(1935\)](#), [Lhermitte \(1939\)](#), etc., generally deal with diverse disorders of the body scheme, in which the subject is either unaware of his own deficit (hemiplegia, hemianesthesia, etc.), or suffers from anosognosia of [Babinski \(1914, 1918\)](#) [syndrome of [Anton \(1898, 1899\)](#)], or disorientation with respect to his own body or autotopagnosia of [Pick \(1908 b, 1915, 1922\)](#) in a more or less pure form. Although further study of these phenomena is still necessary, the different cases can be classified with respect to the perception of the defect into subjects aware of it and those unaware of it, as [Stockert \(1934\)](#) does, making an analogy with subjects with hemianopsia having “vision noire” (visual blackness) and “nulle vision” (null vision) respectively. In those who are aware of their defect, the sensation that a part of the body does not exist (opposite effect to the phantom limb of amputees) would be due to deep brain lesions (subcortical), which according to [Hoff and Pötzl \(1935\)](#) and [Stockert \(1934\)](#), among other authors, would be parietal and thalamic lesions. In those who are unaware of their defect, the lesions would be more superficial, although [Stockert \(1934\)](#) refers them to the corpus callosum. In these last cases, the body defect is replaced with diverse totalizations, corresponding to true anosognosia, being the most common manifestation in these brain-injured patients, although with different variants as shown by our cases mentioned just before.

#### **27.4.1. b) Inversion**

At the sensory level considered in the precedent subsection *a*) (subject in an inactive state), movements occur almost automatically, without the need for any great initiative or plan of action on the part of the subject. Many daily actions are, of course, of this type, so the body schema deficit is not a major obstacle.

However, when relaxation is not so extreme and the body scheme is not so deficient due to the effect of certain facilitation by muscular effort, elementary voluntary movements can be initiated according to the characteristics already studied. When these



movements are either not large, or the sensory level is very reduced, the sensation of movement is not felt, or if it is felt, this sensation is very altered: with proximal deviation and inversion of the direction of the movement.

For example, if subject M is asked to raise a hand or arm very quickly while being as inactive as possible, he spends about two seconds to raise it 90° to horizontal, but he only senses a phase of simple contact and therefore does not sense the movement. Under facilitation by muscular effort, he takes less than a second to perform the same action, but when he is asked to drop the arm immediately, the sensation of movement, although reached, does not develop sufficiently due to the brevity of the test, and he always answers that the movement is of the opposite arm. Since the movement has been improvised at random, he is then guided by the movement felt and not by the one carried out. This implies a remarkable disorientation about himself. Only when the facilitation by muscular effort and the action last longer, there is no disparity between his action and his perception, the behavior being then practically normal.

In conditions of low functional level in which there is inversion of the direction of movement, the subject presents a great disorientation about his own body, resulting in an unprecedented type of *autotopagnosia by inversion*. This is what we have already studied as *sensory-motor incongruence of the movement direction* (Sec. 24.3.3). This is a notable situation of conflict in the praxis model, a situation that tends to disappear spontaneously by means of an automatic change of references in the sensory-motor complex which becomes entirely governed by the sensory factor now inverted. It can then be said that the voluntary movement of the subject becomes a pseudo-active movement since he is unaware of its true character. Thereby, elemental voluntary movements show aspects unknown until now, revealing further aspects of sensory-motor coordination in the genesis of voluntary movements. Another very singular type of inversion in praxis, as a result of the transposition of sides in the body schema, is observed in tests in which the subject has to choose one of his fingers in response to a verbal request under certain experimental conditions. The results are shown in Table 41.

**Table 41.** Response action of subject M to the verbal request to show a particular finger, in a semi-inactive state and under facilitation by maximum muscular effort. Response time in seconds.

<i>Finger to be shown</i>	<i>Response in semi-inactive state. Response time</i>	<i>Response under max. facilitation</i>
Left index	Right index. 7 s	Corrects quickly
Right thumb	Left index, then left thumb. 7 s	idem
Right little finger	Left little finger. 7 s	idem
Right middle	Left index, then left middle. 7 s	idem
Left thumb	Right thumb. 7 s	idem

In Table 41 it can be seen that in the semi-inactive state (with weak facilitation), after some delay, the remarkable phenomenon of inversion (lateral transposition) *persistently*. There is also some confusion about the kind of finger, but on a second attempt he gets it right although not about the side which remains changed. Keeping the eyes open or closed

does not noticeably influence the response. By contrast, if he applies facilitation by maximal muscular effort in each test, the order is executed immediately and correctly. Interestingly, the subject believes that no variation has occurred, which means that he is unaware of the errors in the semi-inactive state.

The persistent contralateral inversion of the response is somewhat shocking and unexpected, apart from the confusion between the fingers, since when studying the execution of an active movement (Sec. 24.3.3) we have seen that the requested action was correctly performed, although the sensation of inversion was felt immediately afterwards. The explanation for this difference may lie in the character of the movement in question, quite automatic and simple there (Sec.8.3.3), and much more selective now. If the requested movement is not particularly selective, such as lifting an arm or moving the head (midline organ), the movement has a basically automatic motor character. By contrast, if the request is very selective, such as moving a particular finger of a particular hand, a much greater bodily discernment is inevitably required - to choose first the side and then the finger - which forces the subject to be guided by his own body schema. Thus, the motor engram predominates in the first case whereas proprioceptive or sensory control predominates in the second one.

Everything seems to indicate that due to the rudimentary state of the body schema and the great importance that the sensory factor acquires in the sensory-motor complex of the praxic impulse (as explained above), the impulse for the corresponding movement suffers a *contralateral canalization* due to the sensory inversion inherent to a low functional level. The sensory-motor incongruity of other tests involving simpler or more automatic movements, which subsequently tends to disappear, does not occur in the situation we are now considering. In this latter situation, the change of reference arises from the beginning, and the motor factor is entirely subordinated to the orientation determined by the sensory factor. Due to the high specificity of the requested movement, as well as the subject's condition, the subject has no choice but to try to orient himself by his bodily sensation in order to activate the praxic impulse. This change of side is only the initial process of the motor impulse, which in order to reach a certain finger needs a new activation more distal of the scheme, which if sufficient, can achieve the mobilization of the requested finger. The process is therefore discontinuous. Deepening in this phenomenology, we find that, in order to avoid confusions between the fingers, the activation of the schema must reach the root of the fingers, otherwise he confuses them because he cannot discriminate between them. It seems that when the subject is asked to move any finger, without specifying the side of the body, it is not necessary that the body schema reaches beyond the shoulder, since no specific movement is requested. If the schema reaches up to the elbow, the request to move the right little finger results in a movement of the left middle finger. As for the easiness to confuse fingers, it seems to be much lower in the marginal fingers (thumb and little finger), a little more in the index finger, and the maximum confusion occurs with the ring and middle fingers. This is somewhat related to what usually occurs in the normal subject.

Another issue is that when the subject applies maximum facilitation immediately correcting the previous errors, he has the sensation that there is no change with respect to the inactive state condition, the change of side being totally ignored. This can be

explained by two reasons. One reason is that the subject has been guided, both in the inactive state and under facilitation, by the sensation of laterality that he has been able to have in each case, sufficiently understanding the existence of such laterality. The other reason is that, although he understands that there are different body sides, he does not distinguish between them sufficiently when he has *to compare them with each other*, at least in his usual (semi-inactive) state. In fact, the “left-right spatial arrangement” (rather than the lateral orientation of the body) is usually of little relevance to him, and therefore he does not perceive much difference with the result of the test under facilitation.

As seen, the degree of failure in the tests depends on the degree of somatic activation. It is noteworthy that subject M, when first examined in 1938, already showed some evident disorder of the finger agnosia type, although of a different nature from that now described. In that disorder, lateral confusion was not very frequent (20% of the tests), and confusion between the fingers was quite persistent (70% of the tests) although he was sometimes able to correct spontaneously. The result was not much different if the eyes were open or closed. This type of disturbance, corresponding to another functional level, consists of a left-right deficit (not primary spatial inversion) and confusion between fingers, being part of the usual syndrome of Gerstmann (1924, 1927, 1930, 1931, 1940). However, in the case of persistent contralateral canalization, the disorder is deeper, beyond that syndrome. In the light of our research, these old results should be interpreted as the effect of a habitual facilitation (by muscular effort) that produces an incomplete but sufficient activation to overcome the somatic inversion. By contrast, in the later tests, lateral inversion has arisen without being sought, probably because the subject has been trained over many tests to avoid facilitation or to graduate it variably, spontaneously giving rise to favorable conditions for this phenomenon.

#### **27.4.2 Dyspraxia in the coupling of the praxis model with external space**

At a functional level higher than the one studied above, praxis activity is related to the degree of coupling of the body with external space. A certain organization of the area of activity is necessary in order to reach a more complex level. As before, the alterations we found are both agnosia and apraxia, without being able to establish priorities, but rather, the whole disorder of the praxis model can be understood as an apractognosia which we study here at different levels of alteration.

##### **27.4.2. a) Spatial orientation and left-right actions**

The ability of spatial orientation in praxis is determined by pointing tests, either towards oneself or towards external directions. As for the first type, patient M in his habitual state (with moderate spontaneous facilitation) makes mistakes when asked to point at certain parts of his body, with his eyes closed and sometimes also open. For this reason, in the tests on stimulus localization in Sec. 21, it was preferable to resort to the verbal statement of the sensed location rather than to point at it. The mistakes made by the patient are usually a simple effect of the reduction of the body schema, with a tendency to point towards the center of the body, as can be appreciated in the following results:

If asked to point at the chin, he points 2-3 cm below and about 4 cm above the midline. If asked to point at the knee, he points at the thigh. If asked to point at the shoulder, he points at the middle of the arm. If asked to point at the wrist, he points at the hand. If asked to point at the forehead, he points at the nose. The subject corrects these defects spontaneously and easily by applying facilitation through maximal muscular effort.

This same diffuse or imprecise character in the action of pointing is also present when subject M must indicate with the finger directions in space. When he is sitting or standing, in his habitual state of activity (little facilitation), he indicates spatial directions very incompletely, both with his eyes closed and open, although especially in the first case, as shown in the following tests:

If asked to point upwards, he points with the right index finger obliquely upwards and to the right, with an inclination of about  $45^\circ$ . If asked to point forward, he points index finger forward and slightly to the right. If asked to point downward, he points obliquely to the right and  $30^\circ$  below the horizontal. As in the previous tests, a complete and spontaneous correction is obtained by applying facilitation through maximal muscular effort.

As can be seen, the requested directions are indicated partially and with deviation to the right. The latter is interpreted by the deviation of the body schema to that side (see Sec. 24.4 and Sec. 25.2.3). In both types of tests, on oneself and on external space, there is an effect of reduction (shrinkage) of the praxis model since the deviation of the direction occurs in parallel to the reduction (shrinkage) of the somatic model, as we know.

As for the *left-right orientation* and the corresponding action, there are numerous different phases of alteration. We have already seen that elementary actions, such as briefly lifting a limb, remain disconnected from the whole and provide no sensation of movement and laterality. At other times, when the subject pays attention to what he feels and not to what he has moved, the action is perceived contralaterally due to the effect of inversion. In other cases, when the fingers are moved in response to a given order, the action is produced from the opposite side, due to an inversion effect by insufficient activation of the model. However, the subject is not aware of the change, as demonstrated when correcting by means of facilitation by muscular effort. In better conditions, even if the subject has a certain idea about laterality, he is not able to sufficiently distinguish both sides, especially when he has to relate or compare them with each other. As long as simple situations are involved, such as indicating a particular side of his body or of the environment, a fairly acceptable action can often be achieved if his functional level is not too low (moderate facilitation); but as soon as the test becomes more complicated or different relationships have to be established, the defect arises. A somewhat complicated test is to ask the subject to indicate the left or right side of the examiner in front of him. At the beginning, the subject usually has difficulty and confuses the sides, but soon he manages to orient himself better, either because he applies a greater facilitation by muscular effort or because he understands the situation better. However, if after a while the tests are repeated, the subject makes the same mistakes again at the beginning, being able to correct them by applying more facilitation.

The distinction between left and right, and in general the discernment of spatial orientation is very unstable, which is demonstrated in many ways. Let us begin by recalling that letters and numbers drawn on the skin, either in normal form or as mirror writing, are perceived in the same way as long as subject M does not apply facilitation by maximal muscular effort (see Sec. 23.3), showing a behavior similar to that shown with respect to the orientation of visual figures. Even more remarkable is the lack of spatial orientation of subject M when he draws any number in the air. Thus, if he is asked to draw a 2 in the air, in a semi-inactive state, there is a latency of 6 seconds preparing for the action; then, with the index finger of the right hand he usually draws the 2 correctly, but with the left hand he draws the mirror image. Sometimes he simplifies very much the 2 and draws a kind of 9. In the execution of the drawing probably contributes both a certain automatism with a diffuse orientation (see below Sec.11.4.3) and a moderate facilitation that corrects the latent praxis deficit. It is noteworthy that sometimes he can draw the 2 in the correct position with the left hand, but without having previously drawn it with the right hand; otherwise, he writes it as a mirror image. However, with sufficient facilitation (by muscular effort), there is no inversion with the left hand even if he had previously drawn it correctly with the right hand under facilitation. It has also happened that writing correctly with the left hand under facilitation, subsequently in a semi-inactive state he does it as a mirror image with the same hand. In conclusion, both in the case of numbers drawn on the skin (to test orientation on oneself) and when drawing them in the air with any hand (to test orientation on external space), there is an important orientation defect, the errors are not noticed, everything looks the same and laterality is blurred.

This disorder is not only for tactile space, it is a spatial reduction that also affects visual space, as studied in the corresponding place. Thus, for example, subject M knows whether an isolated object is located to the right or to the left of his body, but if several objects modify their relative positions, he does not notice the change (see Sec. 16.2 in Vol. 1). It is also worth remembering the 'orthogonal disorder' in drawings, prints, etc., in which very different and anomalous orientations of drawings, letters, numbers, etc., are perceived as normal and always the same, without noticing any difference (Sec. 16.1.2 in Vol.1). It is remarkable that even the right hand can invert or change noticeably the writing of numbers (e.g., 6 and 9). Even more noticeable are the special changes in the praxis of writing (which would be a singular parapraxia) when the subject is copying a certain symbol in an anomalous position which he perceives as in normal position (see Fig. 81 in Vol. 1).

In order to complete the present complex syndrome, we include various data from brain-injured patients examined in 1938 according to ordinary clinical procedures (prior to this research on brain dynamics).

A case with *mild* disorders of the type now studied is that of patient U. G., whose abbreviated clinical history is mentioned in Sec. 26.1.1 and Fig. 145. This case was considered as one of syndrome of Gerstmann (1924, 1927, 1930, 1931, 1940), with predominance of apraxia, in addition to allochiria and other disorders of unilateral sensitivity. Six months after he was injured, he presented various symptoms of Gerstmann's syndrome. As for finger agnosia (showing fingers as requested), he made only minor mistakes, especially at the beginning of the test, the mistake being only between fingers (changing the fingers of the same hand) and not changing the side of the body, which occurs only rarely. In addition, there was some deficit in naming the fingers

shown to him, as well as amnesia for colors, etc. In relation to the left-right orientation, there was only some confusion at the beginning of the examination. Thus, movements that should be performed with the left hand were executed with the right hand (left hand apraxia). When executing orders to make a "cross" movement (e.g., touching with one hand to the opposite ear), he tried to activate in an elementary way the parts to which the movements referred, in order to improve his orientation. Also, according to his account, he sometimes inadvertently wrote as mirror image from right to left with his right hand. In order to make visible in some way the left-right disorder, it was necessary to resort to more complicated tests. Thus, he made several mistakes when he was asked to point out the right and left sides on himself and on the examiner placed in front of him, since due to his functional lability, it was difficult for him to get adapted. He also presented some constructional apraxia (copying drawings with wrong orientation or changing the perspective, etc.), some dyspraxia with eyes closed at the beginning, agnosia, and discrete acalculia. There was a great improvement after several months, especially in agraphia, which practically disappeared.

Another case in which the praxis model was *severely* affected is that of the brain-injured patient J.C., who suffered from a deep brain abscess in the right upper parieto-occipital area as a consequence of shrapnel. It was drained for quite some time and the patient was examined at the end of 1938. No paralysis or anesthesia was evident with ordinary tests, presenting a patent severe autotopagnosia which was the cause of innumerable mistakes in orientation on his own body. He also presented an elementary difficulty in praxis, predominantly in the left hand, which made it difficult for him to initiate the movements he was instructed to do. There were also several apraxia disorders closely related to schema alteration, left-right disorientation, etc.; some selected examples are described in the following. As for orientation on his own body, he pointed at his elbow when asked to point at his knee. When asked to point at his hip, he pointed at his chest and afterwards at his flank. When asked to raise his left hand, he always moved his right hand and only much later did he get it right. In cross movements such as touching his right ear with his left hand, he was unable to do so, and he touched his right ear with his right hand. When he had to take his hands with his eyes closed, it was easy for him to confuse one of his hands with those of the examiner who put his own hands in between. Moreover, he remained firm in his confusion even with his eyes open. Disorientation and even the feeling of not belonging to his body seemed to exist especially for the left side. Thus, with both hands hidden behind his head, when asked for his left hand, he only said, "here", and then said that it was in front of his head. In contrast, he was more easily able to indicate his right hand. To know where his left hand was, he moved it but did not know how to say more than "it is here". When asked to look for his left hand, he did not know what to do and tapped his chest with his right hand. When asked again, he looked to the left and pressed his left hand. With his right hand he grasped the first hand he found, even if it was not his own. If he was asked to lower his left hand when both hands were raised, he lowered his right hand. He made serious mistakes in the position of both hands, needing to move them to activate the body schema. He confused the sides of the objects and those of the examiner in front of him. When he was asked to point at the parts of the examiner's body, he always did it with the right hand and with great difficulties and mistakes, continually getting upset by his failure. With his left hand he had great difficulty in all kinds of actions, even on himself, since he had great inability to initiate movements and even more inability to direct those movements. He looked for his own shoulder in the air, and then all around his body, etc. He imitated the military salute in any manner. He was extraordinarily apathetic and inattentive. When copying a square, he showed a great lack of initiative, then he drew a total of three lines, one separate and two at right angles, etc.

After a little over a month he had less difficulty orienting himself on his own body, finding his hands, etc., although he still needed to move his left hand to find it. The feeling of belonging to his body was practically normal (no confusion with other people's limbs). Instead, some left-right confusion persisted among their own limbs, and also when orienting in external space. There was almost no apraxia in the onset of movements but there was still a moderate general ideational apraxia (in the test of sending a kiss to someone with his hand, he only kissed the fingers together without any other action). He correctly performed automatic actions such as threatening, combing

his hair, military salute, etc., but failed when trying to perform a more complicated action such as crossing his hands to imitate a butterfly, crossing his hands to grasp the opposite ears, etc. With his eyes open, he imitated positions of the limbs, slowly and with difficulty. He did not know the names of the fingers and had no interest in it. In general, left hand apraxia was dominant. He was unable to decide to copy simple geometrical drawings, or finally did it very badly. He was rather euphoric, somewhat indifferent and lacking general motivation.

As a whole, it is a case of severe disorganization of the praxis model, with pure autotopagnosia generalized to the whole body, and with apraxia of basic character. The disorders can be sorted according to their severity as follows, starting with the most severe ones: disorders of belonging to one's own body, disorders of orientation on one's own body, disorders of orientation on external space, apraxia-type disorders and constructional apraxia. In a partial improvement, the most severe alterations disappeared, but there remained a certain disorder of orientation in external space and in left-right orientation, as well as dyspraxia, especially of the left hand, and constructional apraxia, the latter being one of the most difficult alterations to disappear because it corresponds to the most elaborated function of the praxis model.

Finally, we describe a third case (patient A.C.) of *moderate* praxis disorder, somewhat different from the previous ones since it is a Gerstmann's syndrome due to a typical lesion of the left angular gyrus in a brain-injured man, examined in 1938. Immediately after being wounded, he presented some sensory aphasia (he only understood isolated words and had jargon aphasia, among other disorders), evolving later to amnesic aphasia, in addition to agraphia (at the beginning he was unable to write even his name) which over time became paraphagia. On closer examination, he presented the various phenomena of Gerstmann's syndrome, with a predominance sometimes of amnesia or agnosia (left-right confusion, finger agnosia, agraphia, constructional apraxia, some acalculia and amnesic aphasia, especially for colors).

Regarding orientation and left-right handling, the execution of commands to move the limbs were correct, but confusions appeared in more complex actions such as crossed movements, and even more in actions performed on the examiner in front of him. Thus, the number of mistakes grew as the tests got a little more complicated. For example, when asked to touch the examiner's right eye (in front of him) with his left hand, the subject touched the examiner's right eye but with his right hand. However, when asked to raise his left hand, he did it correctly despite mistaking the hand a little earlier. As for the test of showing the fingers on request to check finger agnosia, he never mistook the side, but almost always mistook the finger of the hand. The constructional apraxia was of the same type as in the first case described above, as well as agraphia, paraphagia and acalculia; in the latter, he had difficulty in reading quantities and performing elementary arithmetic operations, writing 11 instead of 101, etc. The left-right disorientation was again evident when reading quantities with many digits; he did not know whether he should start ordering them from the left or from the right. He also had some general dyspraxia which was very clear when he tried to put on his gloves.

During the recovery from this disorder, the most resistant alterations have been, as always, finger agnosia and constructional apraxia, in addition to amnesic symptoms and difficulty in calculation, all corresponding to higher levels of the schema or of the 'categorical function'.

In relation to recovery, it is worth mentioning what was established by Zutt (1932), perfectly in line with our observations. Thus, in the recovery from the orientation disorder in the present case, the left-right orientation is recovered first for the body but not for external space, therefore the orientation relations between the body and external space, and the spatial articulation actions that derive from it, remain altered. In another type of phenomena, we should distinguish the disorder in writing (agraphia) from the disorder in drawing. Writing is a more automated activity, and the second is more free and constructive. It is in the constructive action where the spatial defect of praxis is more noticeable and persistent, being the constructional apraxia the most persistent defect.

In summary, the most genuine form of left-right disorder corresponds to a general disorder of insufficient differentiation in spatial orientation. This can be observed in

praxis as well as in the visual scene, in writing, drawing, etc. The disorder is analogous in nature to the orthogonal disorder studied in vision, consisting of various forms of apractognosia, or simply of a deficit in the formation of the spatial schema. The more complex the test or experimental situation is, the more this deficit tends to appear, being practically absent in elementary or immediate situations. The comparison between situations, complex relations, constructions, writing, transitive actions projected to external space, etc., serve to demonstrate the deficit in the spatial schema. In these actions there is spatial confusion or insecurity of choice because the difference between orientations is blurred and therefore the mistakes are ignored. All these disorders are highly objective as they are manifestations of praxis that are visible to the observer. The fact of not distinguishing between orientations constitutes what we may call “neutral praxis”, which, due to its character of functional reduction, has perhaps a certain analogy with the illusion of the neutral model. In both cases, there is, as always, a reduction in sensory organization. In neutral praxis, the praxis model is modified losing its specific orientations, becoming subordinated to the circumstances of the action proposed to the patient.

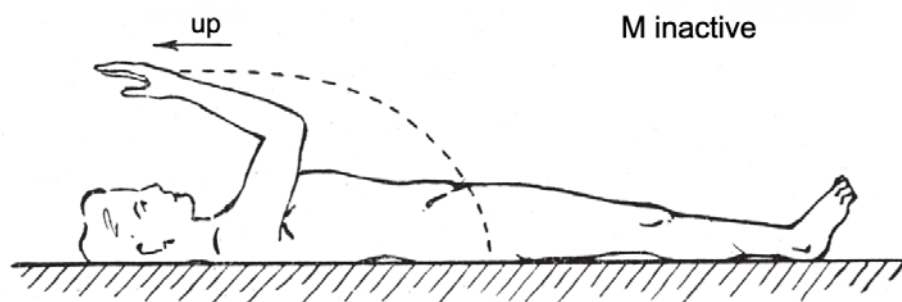
#### **27.4.2. b) Spatial orientation and egocentric-allocentric action**

Concerning spatial orientation, the above tests do not show all of subject M's disorder, i.e., all of the reduction of his action space. To show this clearly, it is enough to change the position of the subject in space, either to ordinary positions (e.g. lying down) or to very unusual ones (the body upside down). In this way it can be observed that when modifying the position of his body, spatial orientation is linked to his own body, that is, it is exclusively egocentric, thus, 'up' is always towards his head and 'down' is towards his feet. Hence, there is *egocentric prevalence* in spatial orientation.

If subject M is in the inactive state and seated in an articulated armchair, it is very easy to place him in a completely horizontal position without him hardly noticing the change. This is done with the subject M's eyes closed, but sometimes also with his eyes open, and in this case he still believes (or has the illusion) that he is in the initial position or rather in the habitual position, since at his functional level, he does not perceive bodily sensations clearly enough. When asked to point with one hand to a certain direction (up or down), he does so with reference to himself, i.e. egocentrically. If the instruction is to point toward the ceiling, he directs one hand toward the head as in Fig. 155. Up and down are always in relation to his head and feet respectively, whatever the position of the body in space. Instead, with facilitation by strong muscular effort, he immediately corrects the defect, acquiring the normal allocentric orientation where the directions in space are no longer linked to his own body and become fixed and independent. It has already been mentioned, in Sec. 16.2 in Vol. 1, that the patient of [Siekmann \(1932\)](#) behaved similarly with his eyes closed.

The mentioned egocentric behavior was already observed in patient M at the beginning of being examined in 1938, shortly after the finding of his disorders of visual inversion and of the orthogonal function, all of which led to a general examination of his spatial visual orientation, as described in Sec. 16.2 in Vol. 1. Years later (in 1946) he presented similar behavior under the same experimental conditions.





**Figure 155.** Prevalence of egocentric orientation. When subject M is in a horizontal position, with eyes closed, and is asked to point upward, he directs his hand toward his head. Likewise, downward is towards his feet. With facilitation by strong muscular effort, he corrects immediately.

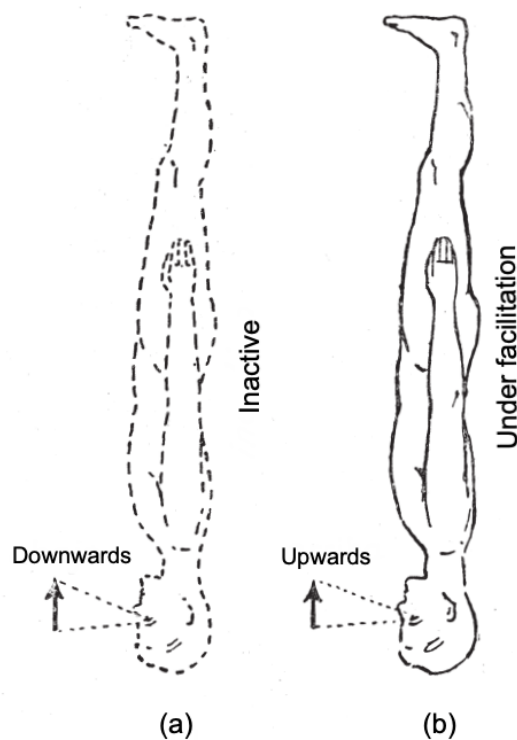
Orientation changes may vary according to the circumstances of each test. For example, in an armchair with a movable backrest, the subject can be reclined backwards without hardly noticing it, even with his eyes open. In this situation, if he sees the ceiling well and is asked to point upward, he does well. However, until he gets up and leaves the chair, he does not realize that instead of sitting, as he thought, he was lying down.

The lability of spatial orientation and the predominance of egocentric orientation can appear in special situations although close to ordinary life. For example, when patient M is at the top of a staircase, close to the ceiling, he may take his proximity to the ceiling (where he can lean) as a wall, and the far wall in front of him as the ceiling (Sec. 16.2 in Vol. 1). Here there is first a situation of chaotic orientation and finally a predominance of egocentric disposition (e.g., what is next to the body cannot be the ceiling or 'up').

If the subject is placed, by means of a tilting table, with the body vertically inverted (head down), trying to prevent him from perceiving the situation (eyes closed), he still admits that he is in a normal upright position as long as he does not apply facilitation by enough muscular effort. Moreover, if in this situation of body inversion he is asked to walk, he may at first believe that he is walking on the ground, seeming to him that the ground is soft (see Sec. 23.5.1). But with the movements (which iteratively activate the body schema), he realizes the situation and believes that after walking a distance he has been turned upside down without him realizing it. He does not know if he was really walking, he thinks he was, although considering that he was not moving, he also admits that he had taken a few steps without moving from the place. Such a confusing perception at the beginning of walking should not come as a surprise after what has already been indicated in Secs. 9.1.4 and 9.1.5. As for the actual inversion of the body, this is fuzzily replaced by the normal position, as in other tests in which the position was less unusual. Concerning the vestibular factor in this patient, see below.

Another experience of egocentric orientation, also with the body objectively inverted, is that described in Sec. 16.2 in Vol. 1 and which can be illustrated by Fig. 156. When the patient, being in the inactive state, is placed vertically upside down, a cardboard arrow 10 cm long pointing upwards is shown to him. The arrow is perceived correctly

because it is in near vision, but he says that it is pointing downward (toward his feet) because of the egocentric reference. Instead, when implementing facilitation by intense muscular effort, he immediately describes the situation correctly: arrow upwards and the own body downwards, the normal allocentric orientation being established. Thus, as soon as the perception of the body schema in space has been possible, the reference system has changed from being himself to being in external space.



**Figure 156.** Egocentric orientation and allocentric orientation, according to changes of reference. (a) Patient M inactive: the body schema is not perceived (dotted line), so it cannot be localized in space; the clearly visible arrow is perceived as pointing downwards *egocentrically* (towards the feet). (b) Patient M under facilitation: the body schema is perceived correctly, inverted in space; the arrow is also referred to external space and is perceived pointing upwards, i.e., *allocentrically*.

The lack of connection with external space and the consequent predominance of the egocentric reference appears in many other situations already mentioned (Sec. 16.2 in Vol. 1). Thus, if subject M looks at the ground while walking, what seems to him to be moving is the ground, at least during the first part of the gait. Sitting on a swivel chair rotating moderately, it is the visual scene that appears to him to be moving while his own body appears to him to be at rest. These phenomena disappear if the subject applies facilitation by means of suitable muscular effort. Of the same category is the [Aubert \(1865\)](#) phenomenon [Sec.13.5 d) and 16.2 in Vol. 1] which in our cases is pathologically present with light (in normal subjects only in the dark). It consists in the fact that when the head is tilted, objects are perceived as deviating from the vertical. In subject M, the deviation is in the opposite direction to the movement of the head, and the greater the tilt of the head, the more pronounced the tilt of the object.

In all these tests, egocentric orientation is the effect of the blurring of the body schema, due to the more or less inactive state (Fig. 156), thus suppressing reference to the

surrounding space. The possible activities in this state must then necessarily refer to the residual model, which gives no reference other than to itself, and therefore the directions of space are linked to himself. Any change of the body in space is ignored because it is not perceived, and instead there is the sensation of a habitual situation (an “illusion of neutral position”), i.e., the *egocentric prevalence is a consequence of referring to the neutral model*. Under such conditions, if the subject is asked to point upwards, he will always point towards his head even if he is lying down or upside down, and he will not mind doing the walking action, believing at first that he is doing it normally. When walking, the ground appears to move, and when sitting on the swivel chair, the visual scene appears to move. In fact, the images of the objects move on the retina in these cases, but the inconsistency of the body schema does not inform about the displacement on the ground or about the rotation of the body and therefore the body is considered motionless (kinetically neutral). Thus, the visual scene that moves with respect to the body is perceived as moving (induced movement). The normal references have changed, and as the subject is isolated, the egocentric orientation is established. Finally, the same happens in the pathological Aubert phenomenon, which differs from the normal Aubert phenomenon only quantitatively, according to the interpretations of Müller (1903).

According to Müller (1903), if a luminous line is observed in the camera obscura with the head tilted 90° to the right, the line appears generally tilted at the top to the left, which is the phenomenon of Aubert (1865) of type A. There is a type EA, where with less tilt of the head, the deviation of the line is towards the same side as the head. The position of the perceived image is influenced by both the visual condition and the head position. It should be noted that the subjective localization of the head may be different from the objective position (neutral deviation), and that the subject forgets that his head is tilted, etc. All these factors give rise to type A, as a result of the egocentric orientation.

This type A is the one that occurs in subject M when tilting the head; the visual scene is deviated to the opposite side (here the phenomenon of inversion by asynchrony does not intervene because the vision is clear and there is good luminosity). As we said, in the normal subject this phenomenon only occurs under special experimental conditions (camera obscura, special attention, etc.), and in a very attenuated form compared to our injured patients.

Among other types of allocentric alteration, of a somewhat different type from the previous phenomena, it is worth remembering experimental autotopagnosia, already mentioned in the study of vision (see Sec. 16.2 in Vol. 1). In subject M, one of his hands is taken and passively moved from side to side in front of his eyes under appropriate conditions to obtain inversion of the direction of movement (brief movement, at some distance and with weak illumination). If he is then asked to grasp with the other hand the one that is moving, he tries to do it in the opposite direction to the real one, and uselessly tries to grasp in empty space. This experiment, besides objectively demonstrating the phenomenon of inverted vision, causes disorientation about one's own body (autotopagnosia of a very special type) and about external space, with the consequent difficulty of grasping to various abnormal phenomena in the visual-tactile multisensory complex. There is the subject's own hand moving passively in front of his sight, which is visually perceived in the opposite direction, and also, the slight articular movement of a

single oscillation, which is not perceived at all. In such circumstances there is only visual information, and now it is inverted.

In comparison with the phenomenon of egocentric orientation, one could consider a type of “super-egocentric” orientation due to the effect of inverted vision (a much more profound orientation disorder). This type occurs in the body schema during *subjective* deviation in walking and other anomalies, such as the sensation of the body going down a slope (see Sec. 25.1.5). It also occurs, albeit *objectively*, in the phenomenon of inversion in praxis, in which proprioceptive inversion of the body schema induces an error in the praxic impulse.

To complete this topic on spatial orientation, we must comment on an aspect related to vestibular excitation. In the tests performed with patient M, a swivel and articulated chair or simply a rocking chair has been used, the patient being comfortably seated, leaning on the backrest, and in the inactive state. The body is made to rotate on itself or to oscillate in the forward-backward direction. The following is then observed. With a half-turn or full-turn clockwise rotation, the subject feels a brief counterclockwise thrust, and vice versa. With a single, wide and forceful oscillation from front to back, the subject feels a brief push forward, and vice versa. The thrust in the vertical direction has not been tested for lack of a suitable device, but similar inversion phenomena should also be present. Under facilitation by maximum muscular effort, inversion has not been obtained even with the slightest perceived oscillation. All these tests, which admit other varieties, show that the body impulse to be picked up by the vestibular system is perceived in different directions (and therefore also in strength, amplitude, etc.) according to the state of central activity (inactive or with facilitation), i.e., the stimulus picked up is inserted differently on the body scheme, producing inversion of the impulse in the inactive state and impulse of practically normal direction under facilitation.

Trying now to pass judgment on the vestibular action on spatial orientation, both in these tests of body impulse and in the previous ones on changes of body position in space, it should be noted first of all that in the Schneider patient of [Goldstein and Gelb \(1919\)](#) these authors came to suppose the existence of a disorder of the labyrinth function in view of the patient's inability to perceive very large changes of body position in space. They also think that there is undoubtedly a cerebellar disorder. As for the latter, it has already been said (Sec. 25.2.4) that it is unfounded, and as for the vestibular disorder, it should also be excluded, as explained below. In preliminary tests of vestibular chronaxie, subject M has shown no difference from the normal subject (neither increased chronaxie nor facilitation effect), and the vestibular orientation mechanisms must be considered completely normal. However, only in the case of a sufficiently developed body schema, a correct vestibular effect is possible since this effect depends on the vestibular connection with the body schema. Thus, if there is no body schema in the tests, the vestibular effect plays no role. However, depending on the intensity of vestibular excitation, the altered schema can be recruited to varying degrees, as observed in the impulse tests. In the inactive state, if the impulse is weak there is no effect. If the impulse is strong, there is sensation of impulse in the reversed direction because the vestibular action is connected with a body schema in a spatial inversion stage. With facilitation, the body schema is

sufficiently developed and the connection with the vestibular system is normal, the vestibular system remaining unchanged within its normality.

According to the above we can conclude that the normal body schema and external space are connected to each other, and that allocentric orientation and the corresponding action require a fully developed schema. The egocentric orientation is the result of a reduction of the body schema and the references are built on the neutral model. It is thus a “spatial neutralization”, as is the rule in many other functions of a similar nature (segmental postures, orthogonal figures, etc.).

### 27.4.3 Dyspraxia due to instability of the praxis model

Finally, we shall study the praxis model with some functional development in order to consider common dyspraxia symptoms. The instability of the body schema and the lability of the spatial schema result in a reduction of “praxis behavior” in the sense that voluntary actions are performed according to a diffuse plan. Two pathological aspects can be considered, the defect in the *initiation of praxis* and in the *constructional praxis* (fragmentation of the action, simplification, etc.). Thus, the lack of amplitude and unity of action, typical of ideational apraxia and ideomotor apraxia, becomes evident.

#### 27.4.3. a) *Defect in the initiation of praxis*

The initiation of voluntary action constitutes a very significant aspect, since it reveals by itself the state of the body schema, as previously studied, especially in Sec. 27.2.2. As we know, for the state of total inactivity there is no possibility of selected voluntary action, resulting in akinesia secondary to the loss of the body schema. With some activation of the schema, the action arises slowly, after a long latency period.

The Schneider patient of [Goldstein and Gelb \(1919\)](#) presented above all great difficulty in initiating any movement that was asked to him. If he was asked to move a certain limb or to perform any other precise movement, he was at first completely stumped. As described by the authors, the patient used to repeat verbally what he was asked to do, as if he wanted to have it clearer; then, it was observed that in order to prepare himself to perform the action, he performed movements of the whole body (head, neck, legs, etc.); then, the movements were gradually restricted to the limb to be moved until finally only the requested movement remained. What the patient was doing was not really complying with the indicated request, i.e., he was not performing a voluntary movement with a given limb, but rather this movement appeared as a final effect.

Since the patient lacked spatial appreciation of his own body, he had to start by finding the limb that had been indicated to him. Likewise, if he had been instructed to draw a square or a circle in the air (a motor action known to him), the beginning was particularly difficult until he was able to orient himself on the limb to be moved and on an external plane, and then the rest of the action proceeded with relative ease, in a very automatic way. Only the initiation caused difficulties.

All these results refer to tests with the eyes closed. However, the process was essentially the same with eyes open since the patient had to search for the limb in more or less the same way as with eyes closed, only the action proceeded more quickly and accurately. The aforementioned authors naturally distinguish between movements of ordinary life, however complicated they may

be, and voluntarily selected movements that respond to a given order. In the former, the action is executed quickly and easily even if the movements are complex and the eyes are closed or not looking (e.g., taking out a handkerchief and blowing the nose), since these movements are automated by habit. As for the difficulty described in the second type of movements, especially in their initiation, the mentioned authors explain it by the loss of visual influence, stating that the patient's behavior undoubtedly shows that visual data in the form of visual representations or perceptions are necessary for the realization of such movements. The authors leave open the issue about the nature of such data, insisting on the fact that visual data are particularly necessary for the initiation of movement whereas they are of little or no significance for the ulterior activity.

For our part we must point out that certain praxis disorders of the Schneider case (e.g., in the initiation of praxis) coincide with our observations in our cases, however we differ completely in the interpretation, which we make in a simpler form. For us, the visual influence does not count at all, i.e., it is not essential (congenitally blind people do not suffer from dyspraxia or difficulty in initiating conscious and selective movements). This point of view has been expressed several times throughout this book. In the Schneider case we would only have the loss of the body schema due to a lack of tactile organization, which hinders the initiation of voluntary movement, being necessary for the subject to activate the schema by means of muscular jerks for example, which is nothing more than applying a non-specific nervous reinforcement (facilitation). The difficulty is greater when trying to initiate the movement since it starts from a "dead point", although there may also be difficulty in the subsequent development of the activity (praxis discontinuity), as we shall see in our cases.

In subject M, in addition to the aforementioned initial difficulty (considerable latency in initiating the ordered movement), it should be noted that the first movement is usually particularly strong; the action is initiated with abrupt and accelerated impulse in most of the tests. This should be attributed to the deficit of proprioceptive control or kinesthetic sensitivity, the subject having the need to make a vigorous movement to perceive it in some way in the initial stage, which is the most affected.

In general, in the requested actions the movements are somewhat clumsy and rigid. The latency time for the semi-inactive state is five to seven seconds to initiate the movement. The action is influenced by insufficient proprioceptive control (resulting in a certain dystaxia in the initial movement) as well as by dyspraxia (inability to combine, sequence and unify the various movements), due to the weakness of the body schema.

#### ***27.4.3. b) Defect in constructional praxis***

In the study of actions, it is necessary to consider their diversity due to the degree of automation. It is known that very common and almost involuntary actions are correctly executed by apraxic subjects, since they are performed automatically and instinctively. Dyspraxia, instead, occurs in voluntary actions that respond to a specific command. Within these actions some are more habitual and automated than others and also have different degrees of complexity. The action of greeting is rather automated and rather simple; drawing or writing in the air is less automated and more complex; transitive actions, as well as the construction of movements, learning, etc., are devoid of any automatism and are also complex sequences of movements. It is easy to understand that defects in praxis are better manifested when the automatism is lower and the need for a construction is greater, as illustrated in the following tests in patient M, from lesser to greater difficulty:

i) A first type of voluntary actions shows the most elementary tests, of highly automated action and very simple movements, in which subject M shows no defect other than an initial latency and a certain discontinuity of action only in the most complex test.

A highly automated action is the military salute. In the semi-inactive state or with weak facilitation to activate the body schema, subject M takes five seconds to “get ready” but his salute is correct although the initiation of the movement is performed with a sharp and accelerated impulse which is typical in many tests. At the end of the action, he only has felt an arm movement of about 30°, as if he were raising his arm less than horizontal. This same test under facilitation by strong muscular effort, in addition to practically suppressing the initial latency, produces a much smaller reduction in the sensation of movement, since the movement felt reaches about 150°, compared to the 180° of the real movement of the salute. It is remarkable that the considerable reduction in the movement felt in the inactive state hardly disturbs the execution of the movement, since it is a rather habitual (automatic) action.

Similar results are obtained with the test of sending a kiss with the hand. In semi-inactive state the latency time is 5 seconds and correct execution. Under facilitation the latency time is 1 second.

A slightly less automated test is to wave goodbye in a semi-inactive state. After a few seconds, the subject raises the arm and a little later moves one or more fingers of the hand in a clumsy and rigid manner. Sometimes he moves all his fingers abruptly as if to catch something in the air. He only feels that he raises his arm a little, as in the previous test, and does not feel the movements of the fingers. Under facilitation, the latency is reduced to a quarter of that of the semi-inactive state, and although the movement is more correct, there is still some rigidity, and finger movements are perceived very slightly. In this test, in addition to other defects, a certain praxis discontinuity already appears (certain time between raising the hand and waving it), i.e., deficit in the unity of action, which becomes important in other tests.

ii) Other tests on less automated actions, such as drawing or writing in the air, reveal more clearly the various aspects of dyspraxia (spatial dyskinesia, mirror writing, etc.):

For example, subject M is asked to draw a cross in the air, being in a semi-inactive state (usual state of weak facilitation). He takes about six seconds to start and does so with some ataxia and drawing only one angle (spatial dyskinesia). In addition, he does not feel the movement while drawing. With facilitation by strong muscular effort, he takes only two seconds to start and draws the cross correctly with the index finger, but the felt movement is about one half or one third of the actual size. About writing numbers in the air, it has already been dealt with in Sec. 27.4.2. Without facilitation by muscular effort there is a constant tendency to produce mirror writing with the left hand, in addition to other errors (simplifications such as in the drawing of the cross). The automatism plays an important role in writing, failing rather the orientation of the writing.

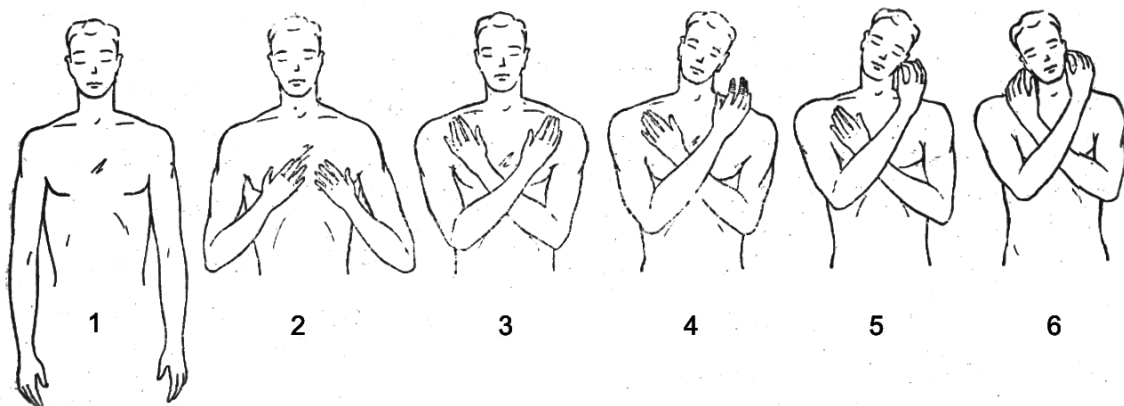
iii) Difficulties increase in actions devoid of habitual automatism and requiring above all a good spatial scheme such as combined transitive movements. In this situation, *praxis discontinuity* due to a deficit in the unity of action is fully displayed, as described below:

A transitive action is to bring the left hand up to his right ear. If he is almost inactive he has considerable difficulty in doing this. After a certain time, he raises his hand as instructed, but only to the middle of his chest, pauses there for a moment and then undertakes a more lateral movement, then tilts his head to favor reaching the indicated ear and goes groping along his face until he reaches the ear. In total, preparation (latency)

and execution, he takes about twelve seconds, whereas with facilitation by muscular effort he takes only one second, performing direct movement without pauses.

Another test of the same type, in an almost inactive state, is to place the left hand on the right shoulder. The subject first raises the left hand toward the chest and stops there, then moves the head a little or slightly the shoulder as if to orient himself. Next, the stopped hand is abruptly directed toward the shoulder, and finally he corrects by trial and error the wrong positions that might have resulted.

A very illustrative test is that of grasping the ears by means of crossed hand movements, in a semi-inactive state, as shown in Fig. 157. From the moment the subject is asked to perform this action, he takes about 15 seconds to perform it, and does not initiate it until after 7-8 seconds. The action is broken down into a series of successive phases as follows. Starting from the natural position, with both hands lowered along the sides of his body and after the aforementioned latency, in a first attempt he raises both hands to his chest, pauses, and in a new impulse crosses his hands and raises them almost to his face; there is another pause to try to grasp an ear with the opposite hand, for which he first moves his head bringing an ear close to the hand. Next, he tilts his head to the opposite side to bring the other ear closer to the other hand. This results in a total of five or six independent phases to perform the whole action. It seems to be impossible for him to reach both ears simultaneously no matter how hard he tries. At other times, in a more clumsy way, he slides his hands crossed over the opposite arms, advancing little by little up to the face.



**Figure 157.** Typical praxis discontinuity in transitive actions carried out by patient M. In his habitual state (almost inactive), the action of grasping the ears with the opposite hands presents an initial latency of seven seconds, and also a breakdown into five or six independent phases over another seven seconds. Under facilitation by strong muscular effort, the action is direct and brief.

All these tests can be performed with the eyes closed or open, and in the latter case the results do not vary essentially, although they may be somewhat faster. Praxis discontinuity in patient M, and also partly in patient T, was observed already in 1938, before the discovery of muscular effort facilitation and other dynamic phenomena. At present it is also evident if the patient suppresses a large part of muscular effort facilitation. Such a fragmentation is very characteristic of the disturbance of schema function of any kind (visual, tactile, etc.). However, as far as praxis is



concerned, it is not highlighted in Goldstein and Gelb's Schneider patient (Goldstein and Gelb 1919), and the authors focus the disorder on movement initiation, as stated above.

Sometimes a certain perseveration is observed in some tests, but it is not frequent and could be regarded as a misunderstood order. Thus, if subject M has to perform a transitive action and at the same time stick out his tongue, in a following test of a different type and which he executes well, he also sticks out his tongue even though he has not been ordered to do so. This is an example of a fuzzy state (difficulty of functional selection), a classic feature of praxis disorder.

iv) The defect becomes very flagrant in those actions that require a previous spatial scheme, a construction or “pensée spatiale” (Lhermitte and Trelles 1933). In the absence of this, partial actions and indirect attempts are produced, as shown in the following test:

In the common two-handed “thumbing one’s nose” test, subject M in 1938 (before discovering facilitation by muscular effort) first gradually joins the hands raised in the air and contacts both thumbs; then, groping with the palm of the hand, he manages to join the thumb of one hand with the little finger of the other; finally, having his hands in this way, he brings them close to his face until he touches his nose with one of them. At present, the result depends on the facilitation applied by muscular effort. When he is in an almost inactive state, the behavior is as described, although it may vary somewhat, taking about 12 seconds for the whole action. After 7 seconds of receiving the order, the subject first places one hand extended in good position, but over the mouth, then the other hand, also extended, tries to contact the little finger, but does so with the ring finger of the hand already positioned; finally, he succeeds in finding the little finger, and then shakes the fingers of both hands, but one of them is still over the mouth, and they are not in the same sagittal plane, the first one is medially and the second one is to one side. With facilitation by maximum muscular effort, the subject places the first hand on the tip of the nose correctly from the first moment, but he also places the second hand on the nose in the same way (simplification). However, he corrects immediately by moving the second hand and placing it in line with the first hand.

v) Other tests are very difficult because they consist of actions that are totally new to the patient, thus entailing a true learning process. They offer great difficulty even with maximum facilitation, at least at the beginning, and also in the much less affected patient T. Thus, neither of the two brain-injured patients (M and T) can, even with intense facilitation, imitate the “flying butterfly” (crossed hands hooked by the thumbs, showing upward the back of the hands and waving the fingers). Only after being taught many times and after multiple attempts, they finally succeed, always under facilitation, in performing the action. If once the action is learned and executed, they are asked to perform it without great facilitation by muscular effort, the action results very imperfect, but applying maximum facilitation it becomes correct.

As for the state of maximum facilitation by muscular effort, although it notably improves the praxis model, it does not achieve full normality. As we know, full normality is also not achieved for the simpler activity of elementary excitability, and if the functions are complex, the correction by facilitation is naturally less efficient. Despite the fact that in patient M there is an important difference in praxis between the ordinary state (weak facilitation) and the state under maximum facilitation by intense muscular effort, it has

been observed that in the latter state there are certain errors in the action, as described above, although they tend to be corrected immediately and spontaneously. The lack of agility is much more pronounced when the tests are totally new or very complicated where learning is necessary, as in the “flying butterfly” test. In short, the spatial schema or “pensée spatiale” is not entirely achieved by maximal facilitation. The same happens in other types of schema, and was already mentioned in the visual schema (see Sec. 15.1 and Fig. 74 in Vol.1). Thus, only by trial and error and in an indirect way can the activity be performed correctly or, in any case, in an approximate way.

Summarizing this third subsection on the praxis model, it can be said that the praxis reduction in actions of a certain complexity reveals a disorder rooted in the elementary function of the local space (somatic model, postural model) and thus compromises the development of the spatial schema, that is to say, of the of the action plan or constructive action. Therefore, this reduction in praxis is above all an inability in constructing, and the characteristic phenomenon is the fragmentation of the action.

#### **27.4.4 Theoretical summary**

We highlight below the most significant aspects of the praxis model. The multiple and varied phenomena of disorganization of the praxis model participate in both apraxia and agnosia, and although one or the other predominates depending on the type of phenomenon, they are always closely related. Thus, the disintegration (reduction) of the action space is part of the general syndrome of apractognosia of classical brain pathology.

Different levels in the degree of spatial organization of the praxis model have been observed, corresponding respectively to severe impairment (severe asomatognosia and somatic inversion), moderate impairment (disruption with the external space), and somewhat milder impairment (praxis discontinuity of voluntary actions that require certain development). The phenomena corresponding to these stages result from internal factors (inversion, neutral image) and external or adaptation factors (reorganization), and depending on their contribution determine the character of the `spatial reduction`.

It is worth highlighting some general aspects of this spatial reduction. The reduction is mainly manifested by functional *undifferentiation*, more or less pronounced depending on the levels of organization considered. At the lowest level of asomatognosia, the model is replaced by the neutral model. At a higher stage, the types of lateral confusion and undifferentiation appear. Apart from changes in the movements, we find first of all, as a profound alteration of the praxis model, a constant primary lateral inversion in simple tests. It is a primary phenomenon resulting from spatial inversion. In more complex tests, lateral confusion is observed due to lack of lateral differentiation. It is a more frequent phenomenon resulting from the lability of the left-right organization, similar to the so-called orthogonal disorder in vision. Finally, with less deficit, the confusion and undifferentiation is no longer lateral but only on the fingers of the same hand. Egocentric orientation that replaces normal (allocentric) orientation, as well as praxis simplification of voluntary actions and praxis discontinuity, also constitute a spatial reduction, that is, a functional undifferentiation.

Another general aspect of importance lies in the phenomenon of the *neutral illusion* (spatial neutralization of the praxis model), which plays a more fundamental role the greater the reduction of the model. Neutral illusion therefore plays a significant role in asomatognosia, and very especially in egocentric orientation; it even constitutes a feasible hypothesis for mirror writing in left-right undifferentiation. As already mentioned when dealing with the postural model, the neutral illusion lies in the predominance of the most habitual attitude of the body. When the organization of the body is labile, the tendency to the most stable or preferred situation appears spontaneously, which can be none other than the habitual (neutral) model. This is the general phenomenon of reduction of meaning in sensory organization, similarly to what we have observed in vision in orthogonal disorder (any orientation of drawings is good or natural) or in the understanding of schematic drawings (change to a simpler interpretation, etc.).

A third general aspect is the *unawareness of the defect* in spatial perception, which coexists with any kind of functional reduction, leading naturally to a limitation of the organization. In praxis inversion as well as in egocentric orientation, lateral undifferentiation, praxis discontinuity, etc., the examined subject does not perceive any defect. On the contrary, the corresponding activity seems to him the most appropriate and normal. This very general aspect has been mentioned repeatedly throughout this book.

Finally, it is possible synthetically to understand the disorders of the praxis model as a *constructive deficit* of the action space, which is the most representative defect of the alteration of the schema function in general. Naturally, this defect is particularly evident in functions of certain complexity, and so it is both in left-right disorganization and in the failure of allocentric orientation and organization of the external space, but above all, it is evident in the development of somewhat complex voluntary actions. Thus, the typical phenomenon of praxis discontinuity (fragmentation of the action) is a consequence of the reduction of the action plan. Since there is no construction of the whole, the action is developed by juxtaposition of fragmentary effects. It is the same situation as in simultaneous visual agnosia, in which the comprehension of the scene as a whole is lost and replaced by partial aspects. The degree of organization is lower, and it could be said, considering the “*pensée spatiale*”, that there is no speech but adjacent sentences or loose words. The most constructive activities are always the most affected and the last to recover. To conclude, the schema (here, spatial schema) can once again be defined as a construction by reorganization of elements in the sensory field (here, action space), which essentially means a functional growth as we shall see below.

## **27.5 Gradual series in apractognosia and functional growth**

After having studied the multiple manifestations of the body schema in our brain-injured patients, we shall address the nosological and physiological meaning of such manifestations. In presenting above the somatic model, the postural model and the praxis model, we have described phenomena that constitute a gradual series of disorders in apractognosia. This series indicates a functional unity, despite the autonomous entities of classical brain pathology.

Among the disorders with predominance of apraxia are mainly: innervatory apraxia (Kleist 1907, 1911), the different varieties of apraxia of Liepmann (1900,1920), constructional apraxia (Kleist 1907, 1911; Strauss 1924), with its close type, visual ataxia (Poppelreuter 1917), and agraphia. Of a rather agnostic character we can mention: autotopagnosia (Pick 1908b, 1915, 1922) and its varieties [anosognosia (Babinski 1914, 1918; Anton 1898, 1899, Redlich and Bonvicini 1909, 1911), the different types of Stockert (1934), etc.], asymbolia for pain (Schilder and Stengel 1931/1932), left-right disorientation (Pick 1908b, 1915, 1922; Bonhoeffer 1923, Pötzl 1924), and the complex syndrome of finger agnosia (Gerstmann 1924, 1927, 1930, 1931, 1940).

There are also some more recent cases included in the denomination of ‘apractognosia of spatial structure’, according to the observations of Ranschburg and Schill (1932), Schlesinger (1928), Zutt (1932), etc., comprising disorders of the visual system and tactile system, which in certain aspects are very similar to those of our cases.

It should be noted that in all these disorders the lesions are “central”, involving the supramarginal and angular gyri or their surroundings, i.e., the classic territories of apraxia and agnosia, either cortical or subcortical.

Many of the manifestations described as individual nosological entities can be found throughout the series of phenomena presented by our brain-injured patients, which argues very little in favor of the existence of such individual entities, and rather suggests different degradations of a same and single process. Within the wide range of phenomena in our cases, we find first of all that the so-called ‘innervatory apraxia’ is closely related to the disorder in our cases described in Sec. 27.2.2 and the praxis initiation defect [Sec. 27.4.3. a)], although the interpretation is very different from that of Kleist (1907, 1911), since in our cases it constitutes an elementary movement difficulty due to a severe deficit of the body schema. We also think that asymbolia for pain corresponds to a very elementary stage of the body schema. In the only case described to our knowledge, it is already interpreted as a disorder of the body schema. In our opinion, such a symptom, which seems to be very poorly defined, would involve both alterations of localization on the skin (disorder of the somatic model) and alteration of the action plan (difficulty in reacting to provoked pain), perhaps in relation to defects in praxis innervation.

Schilder and Stengel (1931/1932) report a case of dissociation between body schema and pain perception. Their case was that of a 73-year-old woman with cerebral arteriosclerosis who after a stroke presented sensory aphasia, mild apraxia disorders, some motor disorder, and asymbolia to painful stimuli consisting of a lack of reaction to painful stimuli, although pain perception did not seem to be altered.

Within the insufficient precision of such a phenomenon, we could provide an observation of this nature in 1935 in a woman with sensory aphasia, apraxia and, seemingly, asymbolia for pain. She did not understand most verbal and mimic commands (e.g., the invitation to be silent), presented intense logorrhea and paraphasia, was unable to imitate any action (in the two-handed “thumbing one’s nose” test she waved her hands separately in the air), wrote indistinctly with the fountain pen open or closed, as well as with a pin which at the end seemed to recognize it, did not know how to handle scissors, nor cut paper, etc. With a strong and repeated pinprick she said: “It pains me ... it hurts me a lot”. This *contrasted with the lack of defensive movements, absolute passivity and no opposition*. Her mood was euphoric.

As for the manifestations of the type of autotopagnosia, asomatognosia, left-right disorientation, etc., they are in fact different degrees of elaboration of the body schema in

the process that goes from the simple somatic model to the coupling of the body with external space, passing through stages such as egocentric orientation, hardly known until now. Thus, it can be seen that these are not nosological entities but modalities of a same function that we have been able to study in our brain-injured patients according to the conditions of central excitability.

Analogous considerations can be made with respect to apraxia phenomena, ranging from ordinary ideational apraxia to the subtlest types of constructional apraxia and visual ataxia. These are different stages of schema construction between the most automatic level and the least automatic level, as is already indicated by [Sittig \(1931\)](#), as well as by [Zutt \(1932\)](#) with respect to the sequential order of praxis destruction [see Zutt in Sec. 27.4.2.a)].

Special attention should be paid to finger agnosia in the syndrome of [Gerstmann \(1924, 1927, 1930, 1931, 1940\)](#), due to a lesion of the left angular gyrus, and which must be examined in relation to our cases.

In Gerstmann's syndrome, in addition to finger agnosia, there is usually left-right disorientation, agraphia, acalculia, and often there is also constructional apraxia and amnesic aphasia. In some cases there are also more widespread disorders, such as visual field defects, balance disturbances, etc. For this author, this syndrome constitutes a pure individuality, although he also recognizes that it appears as a recovery stage (residual syndrome) of more complex brain disorders. He tries to explain it as a selective disorder of the body schema, ascribed to the left-right orientation, and also as a disorder of the finger schema from which agraphia and acalculia would derive.

However, it seems to us more correct and simple to consider such manifestations as a broad apractognosia defect, as a deficit of the general schema function not only limited to the body schema. In particular, acalculia goes far beyond the body schema, even if one tries to associate aptitude for calculus and the handling of numerical signs with counting fingers. Even in the case of interpreting the syndrome more broadly than Gerstmann, as a defect of conceptual structure of space [“begrifflichen und schöpferischen Gliederung des Raumes” ([Lange 1936](#))], which allows to understand agraphia and even constructional apraxia, there is always something else in acalculia that cannot be included in this conception, apart from the very common existence of amnesic aphasia that also escapes from such an interpretation.

As for the individuality of the syndrome and its functional independence, it is already questioned when considering that it can appear as a residual defect of an even more varied disorder, as mentioned above. Its unity is also compromised when taking into account that symptoms of the complex syndrome, even the most typical ones (finger agnosia, left-right disorientation, agraphia, acalculia) are not inseparable, and some of them may be missing depending on the case.

About such an incomplete syndrome and the varieties of the syndrome, some cases from the literature deserve to be mentioned briefly. In the subject studied by [Scheller and Seidemann \(1932\)](#), there was only some left-right disorientation, diffuse apraxia and pronounced constructional apraxia plus dyslexia, hemianopsia, etc. A similar case is that of [Zutt \(1932\)](#), also lacking finger agnosia, and which undoubtedly must have presented orthogonal disorder of figures and letters, left-right disorder and very evident constructional apraxia, in addition to pure agraphia. In contrast, the case of [Schlesinger \(1928\)](#), in addition to some concentric reduction of the visual field, visual agnosia and various tactile symptoms, had moderate finger agnosia, constructional apraxia of the own body, but no agraphia nor acalculia. In the case of [Conrad \(1932\)](#) with finger agnosia and aphasia, agraphia was very moderate. A case of [Schilder \(1931\)](#) with finger agnosia did not show left-right disorientation. It should be noted that the syndrome in question, more or less complete, can be qualified according to more fundamental defects (aphasia, agnosia, apraxia, etc.). Thus, Conrad's case showed an aphasic trait with finger-naming disorder;

in Schilder's and Lange's observations there was a certain agnostic-visual predominance; in Schlesinger's case, an apraxic predominance; and in Wagner's case, the underlying disorder was a severe autotopagnosia. Schilder (1931) distinguishes up to five varieties of Gerstmann's syndrome according to the predominant feature, admitting all of them as perfectly localizable in the brain: the typical syndrome in the angular gyrus; the apraxic type, forward in the supramarginal gyrus; the aphasic type, towards the language area; the visual-agnostic type, towards the occipital area, etc.

Apart from the cases related to the brain dynamics described in this book, there are other patients that we have had the opportunity to examine, some of which are described in Sec. 27.4.2.a), in which different modalities of the syndrome in question are observed according to the predominant feature (rather apraxia in the first case, severe autotopagnosia in the second, and a very typical syndrome perhaps with some agnosia and amnesia in the third case). Also noteworthy in our cases was the late onset of the syndrome, which was rather a residual stage of a deeper disorder (of greater disorganization) of the schema. On the contrary, in another brain-injured patient (not mentioned above), who along with tactile disorders manifested from the beginning a moderate Gerstmann's syndrome, this disappeared in a few days. Thus, there are all kinds of degrees depending on the different localization of the lesion and the stage of the recovery process. This leads to admit a certain degree of apractognostic disorganization rather than a pure individuality of the syndrome.

One might expect, based on classical empirical data, that the two cases (M and T) studied throughout this work would correspond in some way to Gerstmann's syndrome, since they have a lesion of the angular gyrus or close to it (see Sec. 2.1 in Vol.1). Indeed, when subject M was first examined in mid-1938, he had the symptom of finger agnosia [see the end of Sec. 27.4.1.b)], constructional apraxia, certain diffuse disorders of agnosia, severe visual field constriction, etc. Likewise, subject T presented during the first months of 1938, several visual field disorders, tactile agnosia, constructional apraxia, very pronounced optic ataxia, agnosia for geometric shapes, etc. Thus, our brain-injured patients fulfilled to a certain extent what is usually established about a lesion in the angular gyrus or in the left parieto-occipital area. However, as established throughout this book, a lesion in the "central" brain area gives rise to the 'central syndrome' (bilateral and symmetrical repercussion). In this central syndrome all sensory systems are equally involved and in all their activities, i.e. the entire sensory field is affected as a whole (for each of the systems, visual, tactile, auditory, etc.). The "classic" parieto-occipital syndrome is only the part corresponding to the disorder of the schema function in the central syndrome that we have characterized. That part can stand out more than other disorders either when the magnitude of the central lesion is small, or in larger lesions compensated with facilitation by muscular effort. And if more extensive and more elementary alterations are then hidden, it is due, as we have repeatedly indicated, to the inaccuracy and quantitative insufficiency of ordinary examinations, which is what happened to us during the first examination of our two patients (M and T) in whom we were only able to detect agnosia disorders.

It should be noted that if a diversity of manifestations of Gerstmann's syndrome is admitted when the location of the lesion is slightly different, it does not contradict what has been established in the present research, since the type of dynamic repercussion in the central syndrome depends on the position of the lesion. Thus, the fact that apraxia predominates if the lesion is anterior to the angular gyrus, or that visual agnosia predominates if the lesion is posterior towards the occipital region, or that aphasia

predominates if the lesion is in the inferior region of that gyrus, is perfectly compatible with what is established in the brain dynamics exposed here, since such cases would be paracentral (asymmetrical) syndromes.

Regarding the reduced schema organization in subject M, we now see better that it includes phenomena similar to Gerstmann's syndrome or related to it, such as some left-right disorientation, finger agnosia, occasional mirror writing as well as inverted writing, and considerable changes in writing under special test conditions (Fig. 81 in Vol. 1). In addition, subject M presents a more profound disorder of body orientation due to the primary phenomenon of praxis inversion, which goes beyond the described Gerstmann's syndrome.

To understand the physiological significance of the phenomena we are concerned with, it is necessary to consider the functional unity of the disorders, which leads to the establishment of the important concept of functional growth.

The gradual series of apractognosic symptoms is interpreted here from a dynamic point of view as stages of a unitary functional development. Therefore, the ordinary nosological individualities appear as fictions, resulting from a superficial clinical empiricism. Moreover, the schema function does not emerge as something different from the other functions (of a lower level); on the contrary, there is a perfect *continuity* between all of them. Thus it is no longer possible to accept the ordinary separation between "purely sensory" functions and "intellectual" functions (gnosis). It is still noteworthy that in the usual classical brain pathology, all interest is focused mainly on the higher functions, and there seems to be hardly any other topics of interest apart from aphasia, apraxia and agnosia, i.e., schema functions, whereas the most elementary sensory and spatial phenomena (localization, shape, etc.) receive only brief attention and are interpreted superficially in a very simple mechanical way. Here in our study the opposite is true. We have first investigated the nervous excitability state (the sensory state) and then the spatial functions (localization, orientation, space, time, forms, etc.), that is, the structure of the sensory field, considered to be of the utmost importance. It is in this structure of the sensory field where the most fundamental and characteristic questions of the brain dynamics presented here are raised, and to which we have devoted the most attention.

All this means that, without ignoring the complexity of the higher functions, the importance of spatial activities (commonly called perceptions) must be recognized and put in relation with the schema function. Thus, if the schema is a construction based on reorganization in the sensory field, this can also be applied to the most elementary function, for example, of the local sign, especially in the re-inversion process. Functional unity results from a single pattern of organization of the sensory field, organization that varies only quantitatively according to the complexity of the activities.

Such unit and quantitative variation are best expressed by means of *functional growth*. In this respect, we find in the body schema a natural continuity between the three types of model (somatic model, postural model and praxis model), which appear as *phases of growth of the model*, and strictly, as stages of growth of the tactile field. Moreover, we can say that such growth of the body schema is linked to the law of psychophysical organization of the spiral development of the sensory field. This law can easily account for both the most severe alterations corresponding to a residual field (somatic shrinkage,

distortion of the model, praxis inversion, etc.) and the deficit in the organization of the higher functions, always linked to a certain reduction of the sensory field due to the overall alteration. In short, gnosis or normal schema is the last stage of the growth of the sensory field, as a result of integration or recruitment of the `central` brain mass. Such growth entails both an *increase in the sensory dimensions* of the field (intensity, space, time) and an *increase in functional complexity* (organization), i.e., a progressive individualization of functions (individualization is the result of organization, as indicated at the end of Sec. 22.4). It is clear that, within such a simple and general biological concept, the schema function is subjected to the general organizational characteristics of any other type of less complex activities, from which it does not essentially differ. All activity depends on the state of growth of the field. Thus, the characteristic constructional deficit of any agnosia implies both a reduction in dimension (size) and organization.

## 27.6 Difference between cortical and subcortical lesions

The fundamental problems will be dealt with elsewhere, but now we must advance some brief clarifications in relation to the above. It should be noted that all of the above is based on the observation of our cases and refers to *cortical* lesions, i.e., to the cerebral cortex involved in the activity of the sensory systems and thus in the development of the sensory field corresponding to each system. In contrast, *subcortical* lesions (of the deep white matter) seem to correspond to a very different behavior. They leave the sensory field intact with all its varied characteristics, and the sensory systems seem practically unharmed; instead, they maximally disrupt complex activities, i.e. complex functions of mixed sensory-motor character (handling, writing, speaking, etc.). There is then a radical contrast between the severe affection of these higher functional complexes and the normality of the sensory structures strictly speaking; not being here applicable the functional unity for the whole of it. Deep (subcortical) lesions in the parieto-occipital-temporal region give rise to serious behavioral disorders in the sense of classical brain pathology. At the same time the sensory excitability and physiological level for any type of sensory function is normal, there is also no primary motor disorder but the connection between the sensory and motor domains is hindered, leading to a considerable impairment of the actions in which the anterior motor region and the posterior sensory region must intervene in unison.

At first glance, the above behavior may appear to be a major limitation to the fundamentals of the brain dynamics presented here, but although it precludes a generalization to all types of cases or brain lesions, it does not undermine the dynamic principles as long as the difference between cortical and subcortical lesion is taken into account. The first type of lesion is related to the *disorganization of the sensory field* due to altered nervous excitability (excitability deficit, summation permeability, desynchronization, spiral reduction by inversion and narrowing, etc.). Depending on the position of the lesion, it may affect all sensory systems equally (central syndrome) or some more than others (paracentral syndrome). The second type of lesion is related to the *brain disarticulation* that disrupts the coupling of the higher sensory-motor complex



involved in the above mentioned complex activities, while preserving the own motor and sensory activities, which do not undergo alterations in excitability and in the own structure. It is understandable that admitting the latter type of disorders means a consolidation of certain ideas of classical brain pathology, whereas the first type of disorders means a major pathophysiological and nosological transformation. The difference between the two types is justified by the type of anatomical lesion: nervous centers (gray matter) in the first type, long intracerebral pathways (white matter) in the second type. Their different nature is not an obstacle for both types to complement each other in the whole brain activity, leaving open the question of the close relationship between both types; for example, the possibility of a slight deficit of cortical excitability in a subcortical lesion, diaschisis effects or others that we have not yet studied with precision.

As an illustration of subcortical lesions, we present the following cases studied in 1947-48.

*Deep left parieto-occipital glioma.* General symptoms: headache, very frequent vomiting, pain on percussion of the left parietal region, pain on pressure on eyeballs and trigeminal points, especially on the left side. There is no papilledema. Local brain symptoms: homonymous hemianopsia (explored without perimeter, in bed), amnesic aphasia and some sensory aphasia (great deficit in naming and evoking and poor comprehension), marked ideational apraxia, complete agraphia, acalculia, dyslexia, finger agnosia, difficulty with transitive movements (difficulty in left-right, etc.), and minimal visual agnosia (partial failure with overlapping figures).

As for complex activities, the following stands out: when writing, the patient has great difficulty in handling the pencil, which he places inverted, and finds it difficult to hold it between his fingers, as well as to place it on the paper [agnosia of utilization (Morlaàs 1928)], he strongly deviates towards the left edge (visual side preserved) of the sheet of paper, placing the tip of the pencil on the fingernail of the finger that holds the paper. When trying to write his name, there is perseveration of the initial line of the first letter, which prevents any further action. He fails all tests of naming, handling, etc., although he is able to perform simple tests on command such as sticking out his tongue, closing his eyes, raising his hand (therefore, there is no innervatory apraxia). He is also unable to lift his head off the pillow or does so slowly and clumsily. He is totally unable to perform more complex commands such as sending a kiss with his hand or simply putting his lips in a kissing position. In the two-handed "thumbing one's nose" test, after much insistence and watching someone else do it, he attempts to bring both outstretched hands together, but without any relation to his face. In many praxis tests there is a strong tendency to perseveration, much less in language. Thus, there may be pseudoparaphasia. Spontaneous automatic language (ordinary stereotyped conversation, greetings, farewells, etc.) is correct, but more voluntary and selective language reveals an amnesic defect as well as a partial defect in comprehension. No paraphasia nor jargon aphasia.

The special examination on dynamic phenomena (excitability, asynchrony, etc.) did not give any positive results, although it was carried out while the patient was in bed, without the use of special equipment and without great precision. In any case, any dynamic deficit, even moderate, was ruled out with reasonable certainty. Some of the results of the dynamic tests were as follows. For the vision of the two healthy halves there was no delay. He perceives winks and fast movements even in low illumination or at some distance, there were no color and orientation disorders of the visual image (no slight deviation from the vertical of a suitable test object, even by moving it away or dimming its illumination to the point of barely perceiving it, and in monocular vision). In accordance with all this, there was no facilitation effect by strong contraction of the whole musculature. There was also no disturbance of tactile sensitivity in its various aspects including gnosis.

The clear clinical diagnosis was confirmed by ventriculography (disappearance of the posterior horn of the left lateral ventricle).

Another very similar case, although with a more inferior deep lesion, is that of an otogenic left temporal abscess, confirmed by surgery in the middle somewhat posterior part of the temporal gyrus (temporo-occipital region), quite deep and with a large purulent discharge. Symptoms in the acute phase, of general type (of intracranial hypertension, irritation) were: intense headache, fever, meningismus, slight papilledema, paresis of the left third cranial nerve, involvement of the left trigeminal nerve, etc. Local symptoms were: homonymous right upper quadrantonopsia and very marked amnesic aphasia with very slight difficulty in comprehension and occasional paraphasia. There was no dyspraxia, perhaps very slight signs. Dynamic tests on vision and other sensory systems gave no result, or it was so weak that it was within the error margin.

After several drainage punctures, symptoms remitted almost completely, except for residual deficits in previously blind quadrants.

Twenty days later, there was a relapse with the same symptoms that slowly increased, and further interventions were necessary. Over time, he developed pronounced intracranial hypertension with tendency to cerebral herniation. Brain involvement was still limited to the blindness of the quadrants mentioned above and to language in which the naming defect predominated in addition to some semantic and comprehension alteration. There was no apraxia, not even in the copying of drawings.

In new dynamic tests, it was not possible to clearly find any visual defect (no delay nor small inclination with respect to the vertical, no improvement with muscular effort, etc., even overlapping figures were well recognized). There were no problems in touch either. However, perhaps there was some deficit of excitability in hearing; the right ear showed some hearing loss on audiometry. In the moderate comprehension deficit there was a certain lag because a word was heard well but its meaning was not immediately understood, even in the case of very common words; however, there was no facilitating action. The abscess became deeper and deeper and more difficult to drain. He had strong trigeminal pain (pain in the middle of the face, especially in the teeth, which were healthy).

In summary, the exposed subcortical cases showed fundamentally no defect in perception (apart from "marginal" blindness), nor in comprehension, but the patients were unable to perform complex tasks (praxis disorder in the first case and verbal naming deficiency in the second) although they lacked a true motor lesion. It is not the time now to give further explanations, and we merely indicate the agreement between our observations and the point of view of the neurologist Pierre Marie when he considers that the neuropathology of war is a "polio-pathology" or pathology of the gray matter, whereas during peacetime there is rather a "leuko-pathology" or pathology of the white matter.

## 28 Schema in manual touch

### 28.1 Tactile object recognition

After having studied the body schema in our brain-injured patients, we shall now deal with the tactile schema itself, i.e., the schema in manual touch (tactile gnosis). It is possible to establish relationships between both types of schema, the body schema and the manual schema, and to consider the latter as a particular aspect of the former, so that the manual schema is the body schema referred to the hand. Some authors have already pointed out the affinities of tactile gnosis with somatognosia (Schilder 1931), as well as with praxis in a broad sense (Lange 1936), considering especially tactile agnosia as a constructional agnosia. In our study we shall see the close link between manual recognition and body schema.

For a complete study of the present issue in our brain-injured patients, we shall first examine the characteristics of tactile manipulation of objects, and secondly, tactile recognition of objects (tactile gnosis).

#### 28.1.1 Tactile manipulation

The study of tactile manipulation is the starting point for tactile object recognition. In view of what has been said about tactile localization and especially about the body schema, the first question that arises is how manual activity (manipulation and recognition) is possible given that the hand and especially the fingertips are preferential sites of distal involvement in the concentric reduction of the body schema.

For subject M, it is easy to understand that ordinary conscious manipulation of objects (gentle hand contact) is of little use in obtaining tactile information about them. However, in such a situation the subject uses spontaneously, and without receiving any external indication, various procedures that tend to counteract the disorder. These procedures are of two types; a muscular effort more or less generalized to the whole body (as in any voluntary action) which activates the somatic body model, and a touch with energetic pressure of the hand and fingers in addition to rubbing the object, which entails a considerable increase in the tactile sensations aroused. This spontaneous correction of the preferential distal affection of touch exists in subject M since he was wounded, and is independent of the finding of the phenomenon of facilitation by muscular effort, although later this facilitation is applied with more strength and security. The adequate manipulation of objects depends on the body scheme of the hand in which somatic, postural and praxic aspects of the hand are integrated.

It is clear that the correction of the defects, as indicated above, varies according to the degree of muscular effort and contact pressure. The hand may turn out to be more or less enlarged and sensitive in the body schema, as can be deduced from the errors in object manipulation in the following tests with patient M.

One of the tests is to count cards from a deck of cards with eyes closed and applying maximum facilitation by muscular effort. He acts slowly to ensure that he passes the cards from one hand to the other to count them. Out of 15 cards he only counts 14 in these conditions because one of the times he puts two of them together. This small error disappears with the eyes open. But if the facilitation by muscular effort is less intense in such a way that the body scheme makes the hand very incomplete (according to the subject's sensation), then more mistakes are made: out of 15 cards, only 11 are counted because more than one card is taken at a time. If the facilitation is even more moderate and the hand is probably only felt as "sketched", he counts only 7 cards out of 15, as he sometimes takes up to four cards together as if they were a single card.

A finer test, applying maximum facilitation by muscular effort, consists of counting the pages of a notebook with one hand. Out of 15 pages, he counts 12 because he puts two of them together several times. The same happens when leafing through a book with thin pages or a large-sized magazine. The difficulty can be so great that instead of grabbing the page by brushing the edge of the page with the fingertips, he decides to put his hand in the center of the page, trying to scrunch it up and grab it by the relief formed by the creases.

The defect in manipulation, which is never completely erased, can be very diverse depending on the possibility of counteracting the distal deficit. Depending on the type of objects, it is sometimes possible to use maximum facilitation and contact pressure, as in the case of cards in a deck of cards where the cardboard is resistant. In contrast, with the thin pages of a book, the subject can only apply facilitation by muscular effort, as tactile pressure plays no role and the pages cannot be handled as independently as the cards in the previous test. It should be borne in mind here that the patient is a manual worker with rather rough hands, and although this has some influence it cannot be the cause of the errors made, as demonstrated by the degree of error in relation to the facilitation applied.

The test now consists of counting cigarette rolling papers. Out of 8 papers he only counts 5. He performs the test applying maximum muscular effort and also great tension in his hands to achieve maximum facilitation. At the same time, he uses all kinds of controls, such as rubbing the papers to make sure if there are several or just one, and putting the papers in one hand carefully to extract them with the other. However, as can be seen in the result, he has pulled out two papers together, and the most remarkable thing is that he does not take the last one because he believes they are finished.

These types of tests can be varied indefinitely, and a similar result is always obtained. For example, another test is to take toothpicks from a toothpick holder by the tip. From a total of 11 toothpicks he picks up 8 of them (sometimes he picks up two together, and throws away another one without noticing it). In another test he has to pick up (and count) with one hand the pins he has in the hole of the other hand. Out of 35 pins he only counts 31. Here he makes less mistakes than in other tests because after picking up the pins he takes the time to touch them with three fingers to check if there is more than one, but if

he does it with only two fingers, he always picks up several pins at once. As in the case of the cigarette paper, he considers the test finished when he still has two pins left in his hand; when warned, he then picks up one of them and drops the other without realizing it. As in the previous tests, he applies maximum facilitation and is left in full freedom to act except for opening his eyes, although sometimes sight does not help much in such subtle tests. Subject M performs all these tests with remarkable slowness despite maximum facilitation. Under weak facilitation it is almost impossible for him to perform them.

According to what has been said above, it is easy to understand that any daily manual activity must always be undertaken under the action of maximum facilitation including maximum muscular tension in his hands. He usually presses hard objects with his fingers or rub them repeatedly. This is what happens when he tries to button his vest; he uses muscular effort facilitation and both hands, although it is also possible for him to do it with one hand but with great difficulty and slowness. With weak facilitation he has great difficulty finding the buttonhole, less trouble finding the button, but then putting the button through the buttonhole is very cumbersome, taking altogether about 30 seconds.

The Schneider patient of [Goldstein and Gelb \(1919\)](#) used a tactile manipulation for object recognition analogous to that of patient M in the above test with pins, and in addition to multiple fingertip contacts, probably exerted some pressure on the object and was also aided by some diffuse muscular effort throughout the body, although his alteration was not as pronounced as in our patient M.

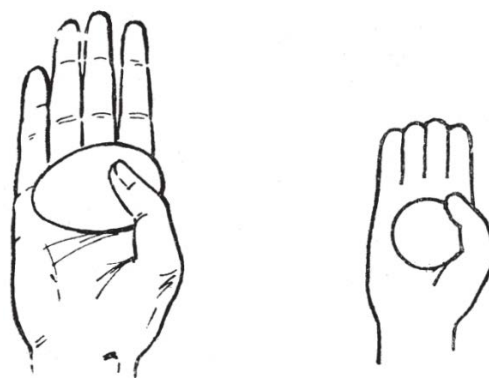
### **28.1.2 Tactile object recognition**

Tactile recognition of objects is pathologically conditioned by a reduction in stereognosis that includes the aforementioned manipulation defect. Recognition is also affected by a defect in the tactile schema that hinders a synthetic conception of the object. This leads to a successive recognition of partial aspects of the object, a fragmentation that is typical of all types of disturbance of the schema (visual schema, body schema, etc.).

We shall therefore deal with the two defects mentioned, astereognosis and tactile asymbolia. The first has already been studied in part when dealing with the reduction in manual (active) touch (Sec. 23.5.2), and we shall now complete it in relation to the issue at hand. In the *stereognostic defect* there is no complete loss of the spatial characteristics of the object, nor of its form, but rather a reduction and a transformation are involved. *Stereognosis reduction* is a natural consequence of the gradual disorder of the sensory function, and is manifested in modifications of the hardness, size and texture of the object, due to a reduction of the sensory dimensions of intensity, space and time. The object is then perceived as softer, smaller, smoother, etc., than it really is. Connected to this disturbance is the *stereognosis transformation*, a metamorphosis illusion that signifies a change towards more stable forms and that follows certain laws of configuration.

We have been able to find these characteristics in other brain-injured patients, previous to the ones we are now dealing with. These other patients always feel in the affected hand that the examined object has decreased in size compared to when they examine it with the healthy hand. They also feel that there are certain changes in the

conformation of the objects as they may seem to be deformed in a certain direction, etc. An illustrative example in subject M is as follows. Subject M holds a 7 x 5 cm wooden egg in any one hand (Fig. 158). If he applies maximal muscular effort facilitation, he perceives it as oval and feels that he is touching it along the fingers, although the fingers may perhaps be felt as slightly incomplete at their distal end. However, if the facilitation is much weaker, the egg is perceived as spherical, smaller in size and more localized towards the hollow of the hand or maybe more proximal, depending on the circumstances of facilitation, palpation, etc. (Fig. 158). If the facilitation were too weak and the palpation of the object too soft, all sensation of size and shape would vanish, and there would be even greater proximal deviation. In conclusion, in diffuse perception, forms tend to be gradually simplified in accordance with the reduction of the organization. That is, it is not a matter of perceptual errors but of a regular change towards simpler and more stable forms, just as we have found in visual forms and in tactile forms. Thus, the modifications felt correspond to the state of the tactile field, and in this case, of the tactile field of the hand. If the size of the object is felt to be reduced, this is an expression of a similar decrease in the size of the hand, and the same is true for the change of form. Ultimately, it can be said that gnosis of the object is gnosis of the hand.



**Figure 158.** Schematic representation to illustrate the change in size and shape of a wooden egg. On the left, practically normal stereognosis with maximum facilitation by muscular effort. On the right, reduced stereognosis, with weak facilitation, perceiving a sphere of smaller volume and located more towards the center of the hand, corresponding to a certain reduction of the hand. (In the test, the hand is closed over the object. Here it is shown open for clarity of illustration).

With regard to the *disturbance of the tactile schema* itself (tactile agnosia or tactile asymbolia), our patients, M and T, only seemed to have, at the beginning of being examined, the defect of loss of meaning of the manipulated objects. A few months after subject T was injured in April 1938, he identified a shell as a snail with the left hand, and as a “hollow thing” with the right hand, thus showing a tactile asymbolia mainly of the hand opposite to the brain lesion. But this asymbolia was not pure, since the subject already presented some defect of postural sensitivity and particular disorders of spatial sensitivity, among others. Patient M, studied extensively during the summer of 1938 following the finding of inverted vision, orthogonal disorder, etc., also showed bilateral tactile agnosia. A key held in either hand was interpreted as “a piece of iron with a handle

up here and a point down below”, although this was not a pure asymbolia either, since at that time he was found to have very pronounced disorders of deep and postural sensitivity throughout his body.

However, failure in recognition was not always the rule in patient M. In some cases he got it right, albeit indirectly, recognizing objects such as a buckle, a fork, a brush, a pair of scissors. In other cases he assigned a very different meaning, as in a sink stopper with a chain or a rifle bullet. Finally, in other cases he only indicated its shape or some character, without taking a decision. With respect to the different objects, his answers in quotation marks are indicated as follows:

Buckle with belt: “I don't know ..., with corners”, (touches the buckle) “looks like a buckle...”, (pulls the belt, touches the holes) “a belt, it has here something to hook” (speaks very slowly).

Fork: “It has points here ..., a fork”.

Nail brush: “Soft here” (the bristles) “and hard here” (the wood). It makes the bristles sound, but by making noise at the same time it cannot recognize the nail brush, and only when it stops making noise can it recognize the nail brush by repeatedly moving the bristles.

Pocket scissors: “A piece of iron with two handles”, (he recognizes them by making noise with them, otherwise he doesn't know it).

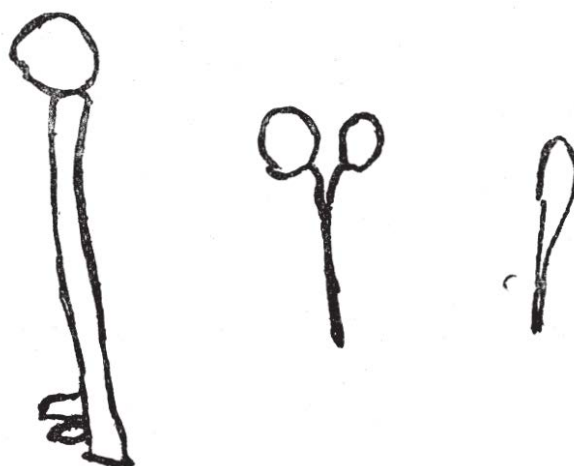
Sink stopper with chain: “clock with chain”.

Rifle bullet: “A nail ..., here the head and here the tip, a round nail”.

Key: “A piece of iron with a handle up here and a spike down here”.

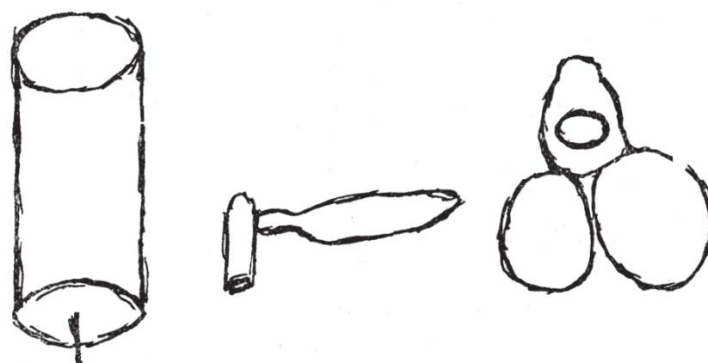
Candle: “A piece of wood ..., a stick ..., it has a rope here”.

In the cases in which he responds correctly, he needs much time and the procedure is not correct and simple, since he uses an indirect analytical-deductive method instead of the immediate and synthetic method of the normal subject. This indirect way of proceeding was mainly emphasized by [Goldstein and Gelb \(1918, 1919\)](#) in their patient Schneider of whom they said that he did not acquire a simultaneous representation of the object since he only provided a description as a sum of parts. Such tests show the need to pay attention to the smallest details and to the entire process, and not to be satisfied with just recording the final effect, which could give the false idea that tactile recognition is normal. This is true for many other functions, as we have repeatedly pointed out throughout this book.



**Figure 159.** Drawings by patient M in 1938. Original size drawings of objects (key, scissors, bullet) drawn with his right hand and looking at the drawing while the objects are manipulated with his left hand. At the end, he does not recognize the meaning of his own drawings which, although deficient, convey the main ideas.

Our patient M behaves in all these tests like patient Schneider. In the case where neither subject was able to recognize an object by touch, they had a fairly good idea of its shape. If the object was offered to them again much later under the same conditions, they came to identify it as the same object as before, even though they did not recognize it. However, in the case of an object that they had not recognized by touch (e.g., a key in the case of subject M) they were of course able to recognize it immediately afterwards by sight, but were reluctant to believe that it was the same object of which they had only recognized the shape by touch a few minutes before seeing it. One of the most remarkable phenomena consists in being able to draw an object with one hand while the object is tactilely examined with the other hand, although not recognized. The drawing, rather acceptable although schematic, does not allow the subject to recognize the object either. Examined subject M in this respect in 1938, while he was manipulating with his left hand an object he was unable to recognize, with his right hand he was drawing its approximate shape by looking only at the drawing (see Fig. 159). When finished he still was unable to recognize the object. The same happened with subject Schneider for several objects, in spite of drawing much better than our subject, as can be seen in Fig. 160. The work of [Goldstein and Gelb \(1918, 1919\)](#) contains other similar drawings of a key, a printer's stamp, etc., very well drawn in spite of their difficulty.



**Figure 160.** Drawings by patient Schneider of [Goldstein and Gelb \(1918, 1919\)](#). Original size drawings of objects (candle, percussion hammer, cigar scissors) drawn with one hand looking at the drawing while the objects are manipulated with the other hand. He is not able to recognize the object either tactilely or visually.

Apart from these similarities between the phenomena, it is not possible to accept the authors' explanation of the method followed by their patient (regarding the way of obtaining sizes, proportions, etc.) to make the drawings. Their explanation is aimed at supporting the idea that their patient is completely unable to acquire spatial relations. On the contrary, it is clear that subject Schneider, like subject M, was able to achieve some spatial perception, albeit more or less reduced. We already know that functional reduction, which affects complex and constructional activities, hinders the idea of the whole. The subject is drawing step by step, without having much idea of the unity of the whole but being able to conceive different parts. The disorder thus lies in the asymbolia of the object, in the inability to integrate the whole process of tactile recognition. The fact that the drawing made of the object is also not visually recognized is due to the fact that it is extremely schematic and even deformed, as occurs in subject M, according to what has been said about schema in visual forms (Sec. 15 in Vol. 1). This also applies to the Schneider patient. A year later, when the facilitation by maximum muscular effort was discovered in subject M, he was able under such facilitation to recognize the drawings of the key and the scissors (Fig. 159) although not that of the bullet because it was very deficient.



The finding of facilitation by muscular effort introduced relevant changes, and nowadays, the defect in the manual touch schema (tactile agnosia) can be counteracted to a great extent allowing subject M to easily recognize many objects. Difficulties or confusions arise only in special tests. For a complete study of the issue we shall first deal with the behavior of subject M in his usual state, that is, with moderate facilitation which he already applied involuntarily before discovering the phenomenon, and the possibility of considerably intensifying it. Afterwards, we shall deal with the changes introduced by maximum facilitation (by maximum muscular effort).

Subject M, whatever his state, uses a series of tactile aids in the tactile recognition of objects, such as pressing them with the fingers, rubbing them with the tense hand, exploring them successively in multiple directions, thus trying to discover their various aspects, and all this is done with remarkable speed. However, the subject in his usual state takes a certain time until he considers the process finished, a process that is rather an inspection than a true recognition. This time is variable depending on the familiarity of the object, the more or less moderate degree of facilitation by diffuse muscular effort, etc., and can range from 7 to 20 seconds, whereas a normal subject reaches the meaning at once and immediately. Thus, in a first phase he makes an analytical examination of the object, which does not go beyond providing a certain idea about its shape, spending 5 to 7 seconds, sometimes much more. In a second phase, the subject, who has only a descriptive conception of the parts (not simultaneous like that of the normal subject) passes from the analytical to the deductive phase and wonders what the object in question can be. Sometimes he succeeds in finding out by indirect means, sometimes he remains in suspense without being able to give more than a description of the shape found before. In this second phase he needs another seven seconds. Finally, sometimes a third phase is necessary to find the name of the object when somehow its meaning has already been found, but this is a process that goes beyond the simple recognition of the object. These phases are similar to those of visual object recognition (Sec. 11.3 in Vol. 1), although the circumstances are not quite the same, since tactile recognition is already somewhat less simultaneous than visual recognition in a normal situation. All of the above is illustrated below with examples of recognition of the following objects by subject M in his usual condition; (his answers are in quotation marks).

Ordinary metal spoon: after handling it a lot, he says "... it has a bowl, it must be a spoon". He has taken 20 seconds because he has applied little facilitation by muscular effort.

Easily recognizable objects, such as a thimble, a ring, etc.: he usually takes five to seven seconds to recognize their shape. In the case of the thimble, he takes five seconds to know that "it is a round thing, hollow at one end". A few seconds later he names it correctly.

Pocket watch: "this looks like a box, but it has a ring up here. It's most likely a watch".

Hat brush with handle: "... a toothbrush".

Metal medal: "a round thing equal to ten centavos".

Open safety pin: "here together and here apart, if a piece were removed it would be a hairpin".

(See other examples above in this section).

The way to *deductively* find the meanings lies, more than in joining the various aspects, in relying on some very typical detail of the object, such as the concavity of the spoon, the hollow of the thimble, the ring of the watch, or being able to insert a finger in the case of a pipe or a flask, etc. When this is not possible, the meaning is greatly altered, as in the case of the medal, or even more so in the case of the safety pin. At other times, the integration of the whole is more necessary, even if there are characteristic details, as in the case of the key. When it is not possible to achieve the synthetic meaning, it is not possible to go beyond a purely additive description of the parts, which is characteristic of tactile agnosia. The resulting conception is then fragmentary and diffuse. That is why changes of meaning or approximations occur, such as taking small watchmakers' pliers for scissors. The subject also does not get a correct idea of the size, which leads him to take the brush with handle for a toothbrush. Other times the defect of recognition borders on astereognosis, for example, when taking a fork for a spoon, a button for a coin or a screw for a nail. In short, tactile recognition is very slow, requires a lot of successive tactile inspection and, above all, fails to grasp the whole and the specificity of the object. It must therefore be concluded that, although the correct answer is sometimes given, the recognition is somewhat deceptive and, in any case, different from the normal one.

*Maximal facilitation* by maximum muscular effort considerably reduces such defects, and then only abnormal signs are found in some tests. The recognition time is reduced to one second as a minimum. If there are difficulties, due to the smallness of the objects, the time may be increased to three or four seconds (this remarkable brevity is due to being quite familiar with repeatedly tested objects). He also applies strong finger pressure and rubs the object, but in any case, the examination of the object is much faster and safer, without the need to inspect in multiple directions and repeatedly check the details. Thus, for the previously described case of recognizing the spoon, now with maximum facilitation it is enough for him to hold it with one hand by the handle and place a finger in the concavity next to the handle to know what it is in a second, without the need to resort to other manipulations. There is therefore now a synthetic (normal) conception, but with a facilitation due to a maximum contraction of all the musculature including that of the hands, which for a normal subject is totally unnecessary. Under such conditions, both astereognosis and asymbolia defects are erased, and he quickly recognizes the shell, the buckle, the medal, the cap, the chain, the glasses, the candle, the brush, the key (whole or without teeth), the bullet, the smoking pipe, etc. However, some insecurities or confusions may still arise, such as taking a button for a coin, or a screw for a nail; but a small warning is enough for him to correct the error. He recognizes small sculptures of human and animal figures; however, he almost failed with a porcelain elephant of about 18 cm, and only after much thought was he able to recognize it. We already know that the maximum facilitation cannot erase all the defect, and also now, some lability can be evidenced in special recognition tests.

In the same way as overlapping drawings constitute a very selective visual test for discovering defects of agnosia or schema (Fig. 76), we can employ a similar procedure with touch by trying that the subject recognizes two or more objects together in one hand. Indeed, subject M with maximal facilitation recognizes well separately a belt buckle and

a pin to fix shirt collar, but if given both together hooked to each other in some way, he fails completely. With four or five objects together he takes a long time and makes mistakes and omissions.

At the beginning of the test with several objects at the same time, the subject tries to select them by removing them from the palm of the hand one by one to explore each one with the fingers, but then the test is like on a single object and does not correspond to the required demand. He takes a long time, but all the objects (4) are recognized correctly. Instead, if he is not allowed to make that selection and is asked to handle them all at the same time, he makes mistakes. From five small objects that he must pick up all at once with one hand (two buckles, a pin to fix shirt collar, a button and a 3 cm key) he claims to recognize "two buckles and two buttons" in 20-30 seconds, unable to resist making a certain selection. Therefore, he has omitted the key and has mistaken the pin to fix shirt collar for a button.

In another test with larger objects, such as a small pliers, a branch of scissors, a key, a medal, he names: medal, key and scissors. In another test with the same objects he answers: key, scissors leg, medal and bullet (this last word probably refers to the sharp head of the pliers).

It may be objected that these simultaneous recognition tests with the hand may be very difficult for many normal subjects, but in fact they are within the reach of any normal subject, at least after some training in a few trials. Such is the case with our patients who have been repeatedly subjected to a multitude of tests in long sessions, and end up adapting quite well to the tests, within their sensory deficits.

The tactile reduction that occurs when an object is covered with a handkerchief causes the perception time to lengthen even under maximum facilitation. For example, for each of the separate objects: medal, buckle, etc., the time increases from one second without the handkerchief to twelve seconds with it, since a single contact is not sufficient for the subject, and he must use rubbing and so on to make up for the loss of tactile finesse due to the interposition of the handkerchief. In perception speed tests, the tactile recognition deficit can also be evidenced. In this type of tests subject M is only allowed to hold the object in his hand for a short time (the object is put in his hand, touched in some way, and then the object is immediately removed). The results may be difficult to be accurately assessed. Subject M seems to need a second for recognition under the best possible conditions, what the normal subject does in much less time, perhaps a third of a second or less, at least for usual objects. Subject M always fails in quick tests, for example in the case of the button, which takes it by a coin, he needs almost two seconds to have time to rub it between his fingers.

Case T, much less examined but very similar to case M when subject T is under maximum facilitation, usually recognizes all types of objects (last exploration in 1944), but tends to use successive touch and an analytical-deductive procedure, generally taking two seconds and sometimes much longer in the tactile recognition of objects. If the objects are not very easy, he takes up to five seconds and more. This recognition is therefore not equivalent to that of the normal subject even though the objects are identified, since a certain degree of initial synthesis is missing, and this occurs in both hands although the right hand is somewhat more affected.

Summarizing all this section on tactile recognition of objects, we see that the alteration of this process involves a complex of disorders including distal reduction, astereognosis and schema alteration. The latter is the most evident and consists in a fragmented conception, i.e., in the inability of simultaneous activity, characteristic of any type of alteration of the schema (visual, praxic, etc.). Normal simultaneous comprehension is replaced with successive comprehension.

## 28.2 Critical examination of tactile agnosia

The nature and individuality of the classic syndrome of tactile agnosia raises similar problems to those of visual agnosia (Sec. 15.3 in Vol. 1) and perhaps with a longer tradition of controversy. We shall first examine the historical background, then the interpretation of the brain dynamics presented here, and finally the rebuttals.

### 28.2.1 Historical background

The question at hand begins with the old Wernicke - Strümpell - Dejerine controversy, in which the first of these authors tries to maintain the independence of the tactile agnosia syndrome whereas the other two refute such autonomy because they consider that tactile agnosia, erroneously called tactile paralysis ('Tastlähmung' in German) of [Wernicke \(1895\)](#) is only the result of a more elementary sensitivity disorder.

This controversy deserves a careful examination since it is the origin of several tendencies and also of many misunderstandings. [Wernicke \(1895\)](#) described two cases of cortical lesion in which the subjects were unable to recognize objects by touch, despite showing scarce alterations of tactile sensitivity. There was then a remarkable disproportion between such alterations and object recognition; that is, the recognition defect went far beyond what could be expected from the small alteration of the simplest tactile functions. Wernicke also pointed out that in cervical tabes with significant alterations in sensitivity, tactile recognition is relatively well preserved, whereas on the contrary, in cortical lesions, slight defects in sensitivity cause an almost absolute impossibility of recognition. Thus, this disproportion led him to establish tactile recognition disorder (tactile asymbolia) as an independent nosological entity. Shortly thereafter, [Strümpell \(1918\)](#) in Germany and [Dejerine \(1907\)](#) and [Dejerine and Dejerine-Klumpke \(1914\)](#) in France took a position against the independent existence of tactile agnosia. They were followed by many authors who refused to admit a manifestation of pure agnosia without any other sensitivity disorder. [Vouters \(1909\)](#), following [Dejerine \(1907\)](#), went so far as to affirm that among all the cases of tactile agnosia published up to that time, there were none authentic. It is remarkable to note that while all the authors of the time accepted the hypothesis of anatomical localization according to the specificity of the nervous centers, mutual associations, corresponding nosological entities, etc. (anatomical and associationist theory of brain functions), Dejerine who was no exception in this respect for aphasia and so on, adhered to a very different unitary approach for cortical tactile functions, more appropriate to current times. In two cases of [Wernicke \(1895\)](#) the presence of elementary sensitivity disorders was quite patent. The question is then centered on the disproportion of alteration of the various functions. For Wernicke, agnosia cannot come entirely from the most elementary defect, whereas for Dejerine, agnosia would be explained by the elementary defect and does not care much about the lack of parallelism in intensity between elementary sensitivity disorder and agnosia. Subsequently, the meaning of this lack of parallelism was highlighted by different authors such as [Heilbronner \(1905, 1910\)](#), [Bonhoeffer \(1918\)](#) and [Goldstein \(1915, 1925\)](#). Further on in this section we shall see its scope and modalities.

Certainly, in cortical lesions the most frequent cases are mixed forms of astereognosis and tactile agnosia, with diverse disorders of the simplest sensitivities. In most cases, astereognosis predominates. Of this type are almost all the war wounded with parietal lesions studied by us.

In about 40 injured subjects with parietal brain lesions, studied in detail by the usual clinical procedures, a global alteration of the tactile system was always found, sometimes with a great astereognosis disorder easily explained by the deficit of more elementary functions, these cases being the most abundant. Other cases had a greater disproportion between the astereognosis defect and the elementary deficit in tactile sensations, spatial functions of localization, movement, spatial discrimination, etc. Rarely there was astereognosis disorder mixed with tactile agnosia and with minor impairment of elementary functions. In this type of injured subjects, it was possible to determine all kinds of forms of alteration, e.g., partial astereognosis of the hand or limited to a few fingers, in very circumscribed parietal lesion. To diagnose these cases in 1938 we considered the reduction of critical sensitivity, which affected the whole set of tactile functions but always in much greater proportion to the higher functions, for example, spatial functions more impaired than elementary qualities, and the meaning of forms even more affected than spatial functions. It was impossible to establish the disorder of a single function, and the most that could be done within this general syndrome was to determine the predominance of astereognosis or of agnosia. It is noteworthy that in the few cases that could be framed between astereognosis and agnosia (perhaps more of the latter), the elementary and spatial functions appeared almost normal at the beginning of the ordinary clinical examination, but in the course of such examination an easy fatigue and functional lability appeared for Weber's test, posture, small passive movements, etc. In addition, whereas the patients recognized the shape of large objects quite well, they failed with smaller objects; many errors were also made in the meaning of objects and only an approximate meaning was possible, such as confusing a fork with tweezers or a lighter that opens with scissors, this being a disorder of the type "disjunctive" agnosia of [Liepmann \(1909\)](#). In such cases with great disproportion between the elementary defect, which was minimal (only lability), and the very patent defect of shape and agnosia, it should be noted that the patients were in the recovery phase and shortly before had presented significant hypoesthesia.

Cases that at first only appeared to be of pure asymbolia are our two patients, M and T, but a more precise analysis showed in the long run much more widespread disorders. This occurred before the discovery of facilitation by muscular effort, which masked the degree of abnormality, and despite the fact that the subjects were in the recovery phase.

Although pure cases of tactile agnosia are certainly very rare (admitting that they may exist), there is a tendency to accept that some cases have indeed been described, in spite of Dejerine and his followers. The cases of [Kutner \(1907\)](#), [Gerstmann \(1918\)](#), [Bonhoeffer \(1918\)](#), [Raymond and Egger \(1906\)](#), [Guillain and Bize \(1932\)](#) (among many other authors) are often mentioned as the most representative cases of pure tactile agnosia. Thus, we would have to add here somehow our two cases M and T, or at least the second one in which disorders other than agnosia are more difficult to be detected, and also the Schneider case presuming the view of [Goldstein and Gelb \(1918, 1919\)](#) that touch is not basically affected but only by the loss of visual influx. But it should be noted at once that while many authors consider the above cases as being of totally pure tactile agnosia, others, more cautious, consider them to be the *relatively* purest cases, since certain more elementary alterations can be detected in special determinations in very pure cases, and other cases already show at first glance small elementary defects (see the following paragraphs).

Indeed, in the case of [Kutner \(1907\)](#), considered as transcortical tactile paralysis, the subject was suffering from epileptic seizures and had some general mental disorders, which already clouded the diagnosis of tactile agnosia. In addition, a considerable functional variability was noted in the tests, i.e. a remarkable fatigability not only in tactile recognition but also already in sensitivity tests, showing slight disturbances of joint sensitivity and of localization. Thus, there was no absolute purity.

The case of [Gerstmann \(1918\)](#), one of the purest among all those mentioned, was a war wounded man with a parietal lesion presenting tactile agnosia as a residual form of a preceding astereognosis; therefore, at least the course of the disorder was not pure. In addition, it is noted, as in the previous case, a great fatigability of the subject in tactile recognition tests and a variability of the results from one day to another. Such patent fatigability in these cases makes them object of the criticism of [Weizsäcker \(1923, 1931\)](#) (see below).

Other cases are much less pure. In that of [Bonhoeffer \(1918\)](#), also a war wounded man, there was agnosia only in four fingers of one hand (the thumb was not), and there was also a very small defect in temperature and pain sensations, as well as an increase in Weber's test. The case of [Higier \(1916\)](#) also belongs to this type of cases, although more impure than the previous one, with alterations of sensitivity and localization errors. These partial-type cases also pose difficulties for the classical conceptions of the gnostic areas and their transcortical action, as indicated by [Kleist \(1934\)](#), although [Bonhoeffer \(1918\)](#) claims the existence of special connections between the gnostic area and each of the fingers. We have also found these partial types in war wounded, although much more impure than those mentioned, and more mixed with astereognosis and elementary disorders of sensitivity. Similar to the case of [Bonhoeffer \(1918\)](#), although extended to the whole hand, is the case of [Poggio \(1908\)](#), a subject who after surgery for cysticercosis presented a slight astereognosis and subsequently tactile agnosia and a small increase in Weber.

Rather less pure is the case of [Raymond and Egger \(1906\)](#), erroneously considered by these authors as a case of "tactile aphasia", although it would only correspond to a tactile asymbolia. The patient was a 64-year-old woman who had suffered a stroke with language disorders and monoplegia of one hand. In addition to the typical defect of recognition of the meaning of objects with the affected hand, she presented many other defects of sensitivity, mainly of spatial nature (increased Weber, tactile localization errors, decreased vibratory sensitivity, decreased sensations of weight and pressure). Other cases sometimes mentioned in the literature on tactile agnosia, besides showing small defects of other kinds that make them impure, were transient forms of deeper alterations of sensitivity, residual states of earlier more elementary astereognosis, or conversely, early stages of progressive disorders that were disintegrating more deeply the cortical tactile system. To the latter type belongs the observation of [Marie, Bouttier and Bogaert \(1924\)](#) in a prefrontal tumor with various mental disturbances, and also the case of [André-Thomas and Courjon \(1917\)](#) with a parietal tumor. To the type of residual agnosia belongs the brain-injured patient of [Magnus-Alsleben \(1924\)](#). Already [Villaret \(1916\)](#) rightly understood the tactile manifestations in brain-injured patients as stages of recovery from deeper disorders. However, a more modern author such as [Delay \(1935\)](#) still tries to interpret the various modalities along the course of the disorder as changes in the association of syndromes independent of each other. The latter cannot be accepted after all that has been said in the above-mentioned series of cases, since it is seen that the disorder affects the tactile organization as a whole. Only in seemingly pure cases the defect in elementary functions is very small in comparison with the defect in higher functions because the rule that higher functions are affected in greater proportion is always fulfilled, this being an experimental fact independent of any theoretical orientation.

At this point it is unnecessary to mention more cases, since with those already mentioned it can be seen that tactile agnosia is not pure since it is always more or less clouded with more elementary tactile defects. However, we shall mention again the case of [Guillain and Bize \(1932\)](#) because of its special circumstances, since it was later subjected again to a long and detailed study by [Delay \(1935\)](#) using very precise psychophysiological methods. This case is that of a young woman with a parietal wound caused by a bullet resulting in hemiparesis, transient speech defects, etc., with easy recovery. Given the type of injury, she must have also presented hypoesthesia, which would allow considering this case as residual agnosia, but this is not indicated in the summary we have used. A year later this case was presented by [Guillain and Bize \(1932\)](#) as 'pure astereognosis due to a traumatic parietal cortical lesion', and two years later she was thoroughly examined by [Delay \(1935\)](#). The tactile sensitivity of her hand was as follows: intact qualities, very small defect of tactile localization, clear defect of simultaneous and successive Weber (the first increased five times the normal value), small defect of shape appreciation and complete loss of object recognition, although she can slowly recognize stereometric objects by deduction. From all this it can already be seen that the case is far from being pure; in fact, there is a rather global

impairment of the tactile organization, although the maximum defect is in the higher function (gnosis).

It is notable that the very accurate pressure, pain and temperature tests in this case, which had provided normal results with mechanical methods that only measured intensity, showed significant defects with electrical methods capable of measuring intensity and time. The chronaxie of the qualities, determined by Bourguignon (1933) himself, was increased in the affected hand about three times above the normal value for mild pain and for temperature, but pressure was shown to be intact. The chronaxie measured was for the palm of the hand; perhaps pressure was affected in the fingertips. In short, the more complete and precise the study of the cases, the better the impurity of tactile agnosia can be demonstrated; the disorder is of the whole organization, according to certain characteristics of physiological hierarchy.

From a careful examination of the circumstances of the so-called pure cases, the Wernicke-Dejerine controversy cannot be considered over. The so-called pure cases being really impure somewhat favors Dejerine's interpretation. But at the same time, the defects of elementary sensitivity in some cases are so minimal that these cases could fit Wernicke's interpretation of the real existence of a tactile asymbolia not derived exclusively from simpler defects. What is evident in these last cases is the great disproportion between the weak alterations of the elementary sensitivity and the total lack of capability for tactile recognition of object meaning.

Let us now address the current state of the issue. An author like Foerster (1936), always in line with classical brain theory and not at all suspicious of sharing more modern anti-localist conceptions, recognizes the limitations of the classical approach. With regard to tactile agnosia he states that the more precise the physiological methods for the determination of thresholds in hand sensitivity tests are, the better we can demonstrate almost without exception that along with tactile agnosia there is an alteration of the pressure points on the skin, an elevation of their intensity thresholds for mechanical and electrical stimuli, an increase in the chronaxie of the pressure points and a fatigability of these points under multiple repeated tests in a short time. He also states that he fully agrees with Weizsäcker's criticism on the concept of pure tactile agnosia and especially on the thesis that sensitivity as such might be completely intact. Weizsäcker (1923, 1931), in part associated to Stein (Stein and Weizsäcker 1927, 1928), considers the usual clinical examination of tactile sensitivity to be insufficient. Where the usual methods fail to discover elementary disorders, appropriate physiological methods (chronaximetry, functional analysis, etc.) can reveal the existence of clear disorders of excitability, a functional change ('Funktionswandel' in German) expressed in threshold lability, increase of the characteristic time, etc. In short, there is a modification in the excitability state which alters tactile perception and would provide an explanation for the agnosia disorder. This leads Weizsäcker to declare that the Wernicke-Dejerine controversy is over, and he denies of course the existence of the tactile agnosia postulated by Wernicke (1895).

Another aspect of the current status of the question concerns the phenomenological characteristics of tactile asymbolia. Since the analysis of the Schneider case mainly, we have not only paid attention to the lack of understanding of the meaning, but also to the indirect way of arriving at it in the tests in which the agnosic subject gets it right.

From very diverse perspectives, attempts have been made to understand the mind with agnosia, either philosophically by Cassirer (1923, 1925, 1929), or psychologically, especially by Révész (1926) in the case of tactile agnosia, or physiopathologically by Delay (1935). From all these approaches, the result is always what Goldstein and Gelb (1918, 1919) had already indicated with respect to their patient Schneider: replacement of simultaneity with addition (see Sec. 12.2).

For Cassirer (1923, 1925, 1929), the world of the agnosic subject (tactile, visual, etc.) is threatened to break up into fragments. The superior unity is lost, the syntactic structure of the whole phenomenon disappears, a sort of “agrammatism” of the rules of perception prevails, similar to the defect studied by Pick (1905) in language. Delay (1935) goes so far as to say that tactile asymbolia is characterized not so much by null recognition as by a radical change in the recognition process, and includes tactile asymbolia within the semantic alterations, while still admitting the autonomy of tactile agnosia.

Half a century after Wernicke, the theories about the brain have undergone many transformations, but the Wernicke-Dejerine controversy is not entirely satisfactorily resolved by what has been said so far, even if it seems otherwise, and at least the arguments of the great German author retain their full value.

It is now up to us to develop the issue by means of the brain dynamics presented in this book.

### 28.2.2 Interpretation according to the brain dynamics

The findings and considerations of Weizsäcker (1923, 1931) seem to put an end to the controversy on tactile agnosia, this being admitted to a certain extent by authors as prominent as Foerster (1936) and Lange (1936), among others. However, going back to the roots of the issue and being consistent, it must be admitted that the problem remains more or less as it was raised by Wernicke (1895) with his two cases, i.e., the great disproportion between the defects of elementary sensitivity and the abolition of tactile recognition of the meaning of objects is still the problem to be solved. The two cases of Wernicke (1895) were already impure, then came other purer ones (Kutner 1907, Gerstmann 1918) among others, which presented some doubts. Finally, Weizsäcker (1923, 1931), in more precise determinations, found that all the cases that can be studied are perfectly impure. This has two important consequences. One is that we should not expect to find a pure case. The other, which is essential, is that tactile agnosia would derive from the disorder of elementary excitability leading to a disorganization in perception, since it is the tactile system as a whole that would be impaired. Although all this is important, the problem of the mentioned disproportion is not solved. As valuable as the conception defended by Weizsäcker (1923, 1931) on the unity of the sensory system is (experimentally confirmed in the brain dynamics presented here), it does not explain the mechanism by which a very small disturbance of elementary excitability leads to a suppression of higher functions (much more complex).

The above-mentioned disproportion (non-parallelism in the intensity of alteration), which corresponds to the principle of Jacksonian ascendancy on the greater vulnerability of the higher functions, constitutes a gap in the ideas of Stein and Weizsäcker (1927, 1928) and Weizsäcker (1923, 1931). Already at the beginning of this book we warned that the “Funktionswandel” (functional change) of these authors was not able to specify



sufficiently the mechanism of perturbation of the different functions. This instead is possible here in our study thanks to the experimental finding of nervous asynchrony (Sec. 3.5 in Vol. 1) which increases the excitability thresholds of the different brain functions differently. Excitability characteristics are also determined here not only for the most elementary activity, such as Stein and Weizsäcker, but also for many other more complex functions and in a systematic way. Taking into account the *quantitative* characteristics of the different altered functions, the problem to be solved becomes clear suddenly, since the mentioned disproportion comes from the fact that the desynchronization follows a Fechner-type logarithmic law which governs the change in the sensory system.

The curves on desynchronization of functions, either sensory recruitment curves or the asynchronous bundle of curves of various sensory levels, illustrate accurately the physiological basis of the non-parallelism in the intensity of alteration between elementary and more complex functions. For example, with respect to tactile functions, in Fig. 118 or 120 it can be observed in subject M that for the sensation of *simple touch or pressure*, the chronaxie increases about ten times the normal value, whereas for a *near-normal localization* sensation, the chronaxie increases about fifty times, which results in a considerable abnormal separation between the respective intensity-duration curves of these two extreme types of sensation. From the localization recruitment curve (proximal deviation as a function of stimulus intensity, Fig. 113) and also from the orientation recruitment curve (Fig. 129), it can be seen that the most affected states (intense proximal deviation and strong inversion, lower part of the curves) are recruited with a weak increase of the stimulus whereas the states closer to normal (high part of the curves) require a considerable increase of the stimulus. The same occurs in vision, for example in the recruitment of the visual image orientation: in order to go from almost 180° of deviation (inversion) to 90°, a small increase in stimulation is sufficient; but to go from 90° to 0° (normal), the increase in stimulation must be very much greater.

In a given sensory sphere, the whole system is impaired, all its functions are affected, but in greater proportion the complex activities (the most physiologically demanding). Thus, the Jacksonian principle of disproportion in functional alteration is physiologically explained by generalizing the law of Fechner (1860), for sensory growth, to pathological manifestations. Considering more broadly the problem of the mentioned disproportion, we see that it is related to the question of the so-called clinical dissociations, which we have addressed more than once when studying tactile functions, especially sensory qualities (Sec. 20.1 and 20.2) where we stated that there are no *absolute* but *relative* dissociations since the impairment is global although some functions are much more severely affected than others according to their physiological characteristics (see also Sec. 23.1 on simultaneous and successive Weber test). Dissociations correspond to the phenomenon of asynchrony that takes place in the series of differentiations of sensitivity (i.e. in the sensory organization), which leads to a dynamic reduction of the system.

It is understandable that the activity of the sensory system is a whole involving the entire system, and that the various syndromes that are presumed to be well-defined and independent entities (agnosia, astereognosis, individualized disturbance of this or that special function) are in fact predominant alterations but not the only ones, thus the usual nosological classifications have at best only a relative value. In fact, what is found is a concrete state of excitability and organization of the system (Sec. 3.6 in Vol. 1), corresponding to a particular stage of development of the sensory field in which the

different functions, apparently independent, are only a consequence of the level of organization (see final part of Sec. 22.4), and therefore do not have a reality of their own. Thus, it does not make much sense to pay attention to the multitude of seemingly independent sensory phenomena, nor to broader classifications of phenomena such as the usual one into sensations, perceptions and gnosis. To specify in a simple and accurate way the new ordering of phenomena we must refer to the perception of sensory dimensions (intensity, space and time), which can be expressed numerically and reflect the underlying physiological state. As for the manifestation of the phenomena, it is framed within the spiral development of the field, an immediate result, as we know, of neuro-sensory isomorphism.

Restricting ourselves to the question of tactile recognition, we state that it is the final result of the organization of the tactile field (of the hand). It is thus not necessary to resort to theories about secondary identifications, inter-sensory supports or visual associations for the object's meaning to emerge. Otherwise, those blind from birth would necessarily remain in a state of tactile agnosia, and this does not happen. More important now is to highlight the characteristic fragmentation of the recognition process that we have repeatedly observed in both vision and touch. This is an agnosic, semantic, categorical, schema disorder, very commented within the brain pathology by [Goldstein and Gelb \(1918, 1919\)](#), [Gelb and Goldstein \(1922, 1923/1925\)](#), [Gelb \(1925/1926, 1937\)](#), [Head and Holmes \(1911/1912\)](#), [Woerkom \(1925\)](#) etc., and outside of it by [Cassirer \(1923, 1925, 1929\)](#), [Benary \(1922\)](#), [Delacroix \(1930\)](#), among others. What needs to be specified is the origin of such *fragmentation* in recognition, i.e. the successive recognition process followed by a subject with agnosia as opposed to the immediate (synthetic) process followed by a normal subject. We have already said that this type of abnormal recognition derives from a deficit in organization (e.g., see Sec. 15.3 in Vol. 1 regarding visual agnosia), and this is what we intend to define more precisely. By carefully examining the type of perturbation in the schema function, we see that if there is fragmentation it is because there is still some hint of interpretation, that is, some vestige of schema. The schema subsists but reduced, as happens in any other type of function. This means that the understanding of the meaning is poor, that the schema has lost intensity (development), thus it cannot encompass the whole but only parts or fragments. In such a situation only the successive (discontinuous) interpretation appears, which in reality is nothing more than a juxtaposition of partial interpretations independent of each other. A subject with agnosia cannot integrate or construct (as in simultaneous visual agnosia, praxis discontinuity, constructional apraxia, etc.), cannot conceive of the whole and the higher unity, and therefore remains in a series of unconnected details.

According to what has been said, we think that the origin and basis of all this behavior in agnosia is that the schema has been reduced, i.e., there is a *reduction in the dimension of the schema*. In this way, we believe to have solved the proposed problem. It could be objected that such a solution was already implicit in the idea of fragmentation in recognition, but it can be replied that it was not clearly expressed. In any case, the importance of the mentioned solution lies mainly in the consequences it has, orienting the issue of agnosia in a physiological direction very different from the psychological one followed so far. Taking into account the schema dimension implies to address the issue

in a strictly physiological framework since the mentioned dimension reduction is not different from the reduction of the sensory dimensions (dimensions of the sensory field), as has been said. Therefore, what occurs for all functions is a *change of scale* (another dimension value). Everything is thus established on a physiological basis since the dimension depends on the active brain mass. This simplifies the issue, gives it a firm basis and, consequently, excludes many sterile discussions. For example, the disputed question of the so-called “noetic” function as to whether it is a disorder of the general psychic (noetic) activity or a disturbance of the physiological mechanism is meaningless in our conception, since we have to refer only to the dimensions of the sensory field and its organization, specified precisely by its dimensions. Moreover, this means that there is the same pattern of organization for all phenomena, there being no opposition between sensory functions and gnostic functions, but rather a continuity, as indicated in Sec. 27.5, for example. Thus, the change that occurs in schema by replacing simultaneous perception with successive perception is analogous to the change from simultaneous Weber to successive Weber. Let us recall that when the simultaneous Weber is almost abolished, the perception of duplicity with the successive Weber is still possible.

### 28.2.3 Discussion. Refutations and assertions

Finally, we must consider several questions about the nature of tactile agnosia, which will lead us to refute or assert some ideas, thus defining more precisely the scenario we have arrived at.

First of all, there is the question of the Schneider case, of which numerous indications and criticisms have been made throughout this book. Regarding the singular tactile disorder in this case, [Goldstein and Gelb \(1919\)](#) literally state: “clinically, we must speak here of a *transcortical sensitivity disorder*, a type of sensitivity disorder whose possibility had not been considered until now, and which is observed and clarified here for the first time”. As is known, the authors think that touch itself is basically intact in all its functions, the loss of visual influence (due to a greater visual impairment) being what determines such a transcortical tactile disturbance. For all that we have exposed, this idea falls apart and it is no longer possible to accept the general conceptions of these authors about their patient. However, the facts discovered in that patient have been verified in our patients (M and T) and in many others later on, demonstrating the true nature of the disorder. It should be noted that with the hypothesis of visual influence and the consequent transcortical defect of touch, Goldstein and Gelb proceed entirely within the classical theory of associations between cortical centers. Also, a transcortical alteration of tactile agnosia had already been proposed ten years earlier by [Kutner \(1907\)](#) in the case he reports, although it refers to a frontal lesion. In contrast, we dynamically interpret the whole set of sensory alterations in the Schneider case as an effect of the central syndrome. In this syndrome, dynamic repercussion is a mechanism that could be assimilated to transcortical action if the latter were understood not as an influence of specific centers (e.g., visual centers) that shape psychological functions, but rather as a nonspecific nervous influence that determines the physiological state of general brain excitability. But this idea is not even suspected in the classical conception of transcortical disturbance nor

in the interpretation of the Schneider case. Such an idea belongs entirely to the brain dynamics presented here.

It is convenient to bring up Schneider's case because the hypothesis of visual influence on touch has given rise to a certain state of opinion. The observations of [Stauffenberg \(1914\)](#) on visual agnosia have also been considered in the same sense by modern authors such as [Kleist \(1934\)](#), [Lange \(1936\)](#) or [Delay \(1935\)](#). Stauffenberg found that of 20 cases of visual agnosia, 15 also showed impaired tactile recognition without major sensitive defects that could be responsible for it. However, Stauffenberg himself makes no reference to specific influences (his observations predate the Schneider case). He rightly considers, following the ideas of his master [Monakow \(1905\)](#) that visual agnosia represents an “asemic disorder of the highest level” which never constitutes a single symptom, being accompanied by other tactile, language, orientation, intelligence, etc., alterations. For our part we must add that such multiple alterations are only an expression of the central syndrome, as already indicated at the beginning of the study of tactile functions (Sec. 18.3), as is the case in our patients and in Schneider's case. The objection of [Thiele \(1928\)](#) to [Stauffenberg \(1914\)](#), based on the fact that the referred multiple alterations can be explained by considering that in most of such cases there were vascular alterations (often with multiple cerebral foci), could reduce the value of Stauffenberg's interpretation. But this means nothing in our patients and in Schneider, in whom the lesion was only one and well circumscribed, which leads us to fully accept the point of view of [Stauffenberg \(1914, 1918\)](#).

The issue of influences still finds in [Lange \(1936\)](#) a very particular point of view. After interpreting Stauffenberg's observations from the point of view of visual influence (like Goldstein and Gelb in the case of Schneider), he wonders about the reverse question, i.e., in what way a tactile disorder should influence vision. He believes that this problem is of great importance even though it has hardly been raised. To this we reply that influences of any kind are non-existent; if the influence of vision on touch has come from an inconsistent psychological domain, for the reverse influence (touch on vision) not even that reason can be found, and ultimately it is the central syndrome that provides a solution for all these cases. In this regard, it should be noted that our patient T seemed to be only a “tactile case” in the early examinations of 1938; and much later on, visual disturbances were discovered (moderate concentric reduction, polyopia, etc.) which, according to the usual conceptions, could not be related to the same cortical lesion causing the tactile defects. Was it necessary then to think that touch was affected primarily and vision secondarily? Not at all; both types of disorders existed by themselves and had to be attributed to a common fundamental disorder since they were caused by the same lesion. The fact that the defect in touch was found first is explained by the fact that the lesion was in a parietal region of the skull quite far from the occipital pole and the subject did not complain of any visual defect. Thus, clinical attention was somehow directed to touch, particularly when considering that just after being injured, he had presented a clear loss of sensitivity on the contralateral side of the lesion (although the lesion did not correspond to the tactile projection area). However, in patient M, the visual defect attracted attention first since the patient complained of visual symptoms and also the entry orifice of the projectile was in the occipital area, whereas the tactile defects were brought to light much later. In both patients (M and T) the disorder of elementary excitability (tactile and visual) was demonstrated when the dynamic action was discovered a year later, which also led to finding the auditory defect in them, as we exposed in the first part of this book (Sec. 1 and 2 in Vol.1).

In connection with this, it must be said that in the Schneider case the visual defect was the first to be discovered and only much later did the authors become aware of the existence of a

similar defect in touch. In fact, the first publication on vision (1918) makes no reference to tactile disorders. When tactile defects were later found (second publication in 1919), it seems natural that they were put in relation to the earlier study on vision, and perhaps this order in the discovery of visual and tactile disturbances has had something to do with the idea of visual influence on touch. In any case, it is clear how much important data can remain hidden in the usual clinical observations that seem to exhaust the question, and therefore the impossibility of a theoretical interpretation in such a situation. A theory is viable only when the data are complete, the examination is exhaustive in all directions, and the results are consistent with each other so that they can be interpreted from a common and simple point of view without the need to continually appeal to new hypotheses or principles.

The existence of multiple disorders poses a profound theoretical problem, and attempts to resolve it within the classical conceptions have been futile. Hesitations and contradictions are not infrequent in a same author. Thus, [Delay \(1935\)](#), at the beginning of his detailed study on astereognosis, when he addresses the question of the lack of parallelism between the alteration of the higher and lower tactile functions, considers that in the combined alterations of anesthesia and astereognosis there is only a fortuitous coincidence, explainable for reasons of anatomical proximity and not for functional reasons. This is not admissible after all that has been exposed. However, in the final part of Delay's work, when referring to the recovery of tactile syndromes in which some are transformed into others, he rightly thinks that it would be justified to wonder whether tactile asymbolia represents only the most superficial and minimal alteration of the cortical tactile region. He goes on to say that to a higher degree would be alterations in spatial perceptions, shape agnosia, etc.; and to an even higher degree, cortical anesthesia. But then he declares that it is impossible to decide, and finally he sticks to the hypothesis of a "gnostic center" in the brain different from that of the most elementary functions. Thus, he again moves infinitely far away from the functional and dynamic conception to accept the fundamental features of the classical theory. However, we know from our research that such a "gnostic center" corresponds to a central region that is responsible both for gnosis and for the most elementary excitability of contact sensation, as it results from the central syndrome.

All that has been exposed so far: the impurity of the so-called pure cases, their revision in the light of the "Funktionswandel" (functional change), the logarithmic desynchronization in our study, the multiplicity of sensory spheres affected in agnosia, the data of the Schneider case and the research on our brain-injured patients, clearly demonstrate from multiple perspectives the shortcomings of classical conceptions. We could state that there are no pure cases of tactile agnosia or anything else, but rather a global impairment that, when the lesion is central, affects all sensory systems equally. What is usually diagnosed and isolated in brain cases is only the most pronounced alteration, the predominant disorder, which will be the disorder of the most complex function. This is at the expense of neglecting other simpler and less evident alterations in the usual examinations, but which demonstrate the true nature of the disorder and the true mechanism of brain activity. The classical conception is therefore only a rough approximation, which in many cases does not meet all the requirements, and fails completely when dealing with *privileged cases* of the dynamics of the brain, such as our two brain-injured patients and Schneider.

Therefore, our dynamic conception not only interprets special cases but any other case, and fully satisfies the requirements of a coherent and simple explanation.

## Compilation of tactile functions

Tactile functions have been systematically investigated in all their aspects, resulting in a large number of new facts and new approaches derived from them. The global alteration of the tactile system in our brain-injured patients is, as we know, only one particular aspect of the multisensory alteration in the already exposed central syndrome. Given the properties of this syndrome, the tactile disturbances are of the same quantitative order as those previously studied in vision. As for the phenomena, the finding of spatial inversion in touch in 1946 (subsequent to inversion in vision) makes it possible to establish a complete analogy between tactile and visual sensory manifestations, and also leads to new advances in the research and to broad generalizations. Therefore, in the central syndrome, the general characteristics of the visual functions compiled at the end of Vol. 1 are now equally applicable, with the appropriate specific modifications.

1) *General excitability* shows an increase in chronaxie and rheobase, as well as permeability to spatial summation (facilitation) and temporal summation (iteration) of the same order of magnitude as in vision, which corresponds to the homogeneous repercussion of the central syndrome. The tactile sensation presents a remarkably slow evolution, and in the extinction phase (where there is persistence) the sensation can be reactivated by summation. It is worth noting the underestimation of weights. Vibration sensitivity shows loss of high frequencies and overestimation of rhythm, due to the effect of increased fusion of intermittent stimuli, phenomena that can be explained by the decrease in excitability speed (increased chronaxie, increased refractory period, etc.). This is similar to what happens in vision with flicker-fusion frequency.

2) *Sensory qualities of touch*, of a much simpler nature than colors in vision, show a notable decrease in sensitivity and a marked heterotactile interval by functional disaggregation (asynchrony). The sensation of pressure or contact appears as the primary activity of touch. The tactile quality of pain presents a considerable enlargement of the pressure-pain (tango-algic) interval. Similarly, temperature sensation is easily decomposed into pressure sensation and thermal sensation giving rise to the pressure-thermal (tango-thermal) interval. The increase in the thermal threshold, heat and cold, explains the significant enlargement of the neutral range of temperature.

Clinical dissociations of sensitivity are relative, not absolute; the explanation is functional and within the overall disturbance of the system. Sensitivities are dynamically classified into tangibility (pressure as primary tactile activity) and true qualities (pain and temperature). The functional nexus between the former and the latter is suggested.

3) *Tactile space* presents phenomena of special importance. In tactile localization, asynchrony causes a large abnormal interval between the simple sensation of contact and specific localization. The stimulation intensity determines intermediate effects such as various phases of spatial localization, which are: primitive projection (contact sensation without any localization), medial deviation, inversion, homolateral proximal deviation, and specific (normal) localization. Each of these phases has several particularities. Such effects derive from two general phenomena: irradiation and proximal deviation. The weaker the stimulus and the more early the localization phase, the greater the irradiation and proximal deviation. Tactile irradiation is homologous to chromatic irradiation

(pathological flat color vision). The elongated shape of tactile irradiation is related to the elongated shape of the tactile field or body schema. Proximal deviation is a manifestation of a concentric reduction of the body schema, with a preferential impairment of the distal regions and a functional privilege of the medial line. All this implies a structural change of the sensory field, giving rise to a field of residual activity, i.e., a poorly defined field (irradiation) and constricted field (centripetal deviation).

As for quantitative relationships, both irradiance and deviation (the latter easier to quantify) vary as a function of stimulus intensity, following logarithmic recruitment curves. Iteration curves are determined electrically as a function of the number of stimuli and as a function of the time interval between stimuli. Mechanical iteration with tuning forks only generates recruitment at low frequencies, due to the aforementioned characteristics of vibration sensitivity. These relationships are ratified in the asynchronous beam of intensity-duration curves corresponding to the different localization phases (Sec. 22.2), where the higher levels suffer a disturbance several times greater than that of the lower levels. In addition, it is worth noting the post-stimulus summation effect that facilitation by muscular effort exerts on the residual trace of a tactile stimulus. The reactivation varies depending on the delay in the application of this facilitation, and is measured by the degree of deviation of the localization sensation (Table 26). The theory of tactile localization and sensory space in general is discussed, and the empiricist theory of the associative-kinetic local sign is refuted. Abnormal localization phenomena (deviation, irradiation, inversion) are the expression of a sensory field of residual function, in correspondence with the central nervous substrate, prior to any experience. Nor is there a local sign as a functional individuality, but rather multiple gradations according to the degree of organization of the nervous centers.

Several functions concerning spatial discrimination are studied. Spatial acuity (Weber) shows, as in vision, a considerable increase in the spatial threshold (reduction of acuity), which is interpreted as a spatial shrinkage (shrinkage of the body schema). The motion sensation produced by a moving object on the surface of the skin reveals a subjective acceleration and spatial shortening in inverse relation to the intensity of the pressure of the moving object. This is a result of spatial and temporal shortening. The spatial phases that arise are identical to those arising in the visual perception of motion. In the perception of figures drawn on the skin, systematic errors, illusions, dysmorphisms, etc., are produced due to the instability of the sensory field. In joint sensitivity (deep movement), as in the perception of a moving object on the surface of the skin, there are also intrinsic modifications (acceleration and shortening of the perceived movement) together with changes in joint localization by deviation and inversion. All this depends on the type of passive stimulation, which can be iterative (multiple oscillations of the same joint) or single (in this case very energetic and large, since an ordinary oscillation does not elicit sensation). Finally, it is worth mentioning the alteration of one's own corporeality regarding reduction in size, weight, continuity, etc., as well as the reduction in stereognosis, "flat touch" due to tactile irradiation which erases the microstructure of the object being handled, etc.



4) Perceived *tactile orientation* presents phenomena of the utmost importance, which complement the study on tactile space. The finding of the inversion of tactile space (and also of auditory space) entails the generalization of the orientation issue and the establishment of new dynamic postulates of brain activity. The general laws are the same as for the orientation of the visual image, and now the process is studied more completely by combining the development of the inversion with that of the proximal deviation leading to a spiral development. The spiral trajectory of the inversion is determined by varying the intensity of a point stimulus in the three areas of the body (head, thorax with upper limbs and abdomen with lower limbs) that are autonomous regions of inversion. The most complete study of the gradual changes in perceived orientation and localization is performed by varying the pressure of a rectilinear stimulus on a distal part of a limb. The lower the pressure of the rectilinear stimulus on the hand, the greater the proximal deviation and tilt until reaching the inversion of the orientation with contralateral localization over the trunk while reducing the size of the line. The characteristic curves of sensory recruitment are obtained quantitatively. Tests on changes in the direction of motion on the skin serve to ratify and complete the previous ones. In the perception of joint movement, there are complicated changes in both the direction of movement and proximal deviation of localization depending on the amplitude and energy of the passive joint oscillation, at the same time as the perception of the intrinsic aspects of the movement (speed, amplitude) is changing. Particularly notable are the phenomena of sensory-motor incongruence in the direction of movement in certain circumstances due to sensory inversion in voluntary movement. The orientation disorder is also manifested in the whole body schema, but only appears as a moderate tilt, since the body figure does not subsist (fades away) beyond a certain tilt.

Orientation disorder is also studied in more complex processes, the multiple sensory alterations that arise in gait being very demonstrative. The successive steps act iteratively on the nervous centers giving rise to a sensory recruitment of the walking direction depending on the number of steps, their frequency (addition effect in fast steps and no addition effect in very slow steps), and also on the step energy, with effects similar to those mentioned in joint sensitivity. Space and time in walking are reduced by the aforementioned spatiotemporal shrinkage. The subjective walking trajectory follows a spiral path. Typical sensory recruitment curves of the direction of walking by iteration as a function of the number of steps are obtained, and are analogous to the curves in vision on the perceived direction of motion as a function of the number of metronome oscillations. Finally, alterations in the localization of the movement of the lower extremities, the sensation of translation on the ground, the subjective orientation of the whole body during walking (turns, subjective slope, etc.) are also studied. Of another type are the singular sensory-motor phenomena in objective postural deviations, static (head, trunk, limbs) and in movement (finger-pointing, walking, etc.), interpreted as an induced deviation secondary to a primary sensory deviation.

Orientation theory is a topic of general significance, and poses very fundamental brain questions. It can be seen that both proximal deviation and inversion are involved in spatial localization, and when both are combined in the developmental process by increasing stimulation, a spiral development of the sensory field is obtained (which

constitutes a law of sensory organization in the brain dynamics presented here). Within asynchrony, the origin of inversion lies in the marginal areas of the cerebral cortex, with inverted and contralateral sensory projection, whereas re-inversion is the result of what we have called central areas (see Sec.2.2.1 in Vol.1). There is thus a direct correspondence between the brain field and the brain state: reduced and inverted sensory field corresponds to brain reduced to marginal activity; enlarged field with normal orientation (reorganized) corresponds to an enlarged brain with the mass of the central areas. It should be noted that the process of sensory organization involves, to different extent depending on the case, both the anatomical configuration and the action of the mass (maneuvering mass of the mentioned central areas). Ultimately, the spiral development of the sensory field reveals a psychophysical isomorphism.

5) *Tactile schema* comprises a set of functions that are in continuity with the previous ones. The body schema presents different levels of organization: somatic model, postural model and praxis model. The somatic model is the most rudimentary level and corresponds to the simple corporeality that only gradually emerges through tactile stimuli or through facilitation by muscular effort, since it is originally vanished. Due to this absence of somatic model, the initiation of praxis is hindered, the facilitation by muscular contraction appears in a quasi-reflex form, activating the model to varying degrees; it is a disorder of asomatognosia type. The postural model means further development, which is indispensable for the correct spatial localization of tactile stimuli. The general posture of the body suffers a subjective deviation due to the orientation disorder. More important is the disorder of segmental postures, which are perceived as deviated towards the neutral posture (sensory reduction). This phenomenon presents diverse degrees of reduction, and also certain illusions in a normal subject. The praxis model is considered in a broad sense, and many different types of action alterations have been addressed. Dyspraxia in the rudimentarily developed model consists of automatic actions guided by illusions about one's own body (phantom body, anosognosia, etc.). There are also several inversions in praxis that cause autotopagnosia by sensory inversion of the direction of movement, as well as the peculiar phenomenon of contralateral canalization of movement initiation under special conditions. Dyspraxia in the coupling of the praxis model with external space refers to defects in spatial orientation (pointing with the finger to oneself and to external directions), and to instability in left-right differentiation, which is a general visual and tactile spatial disorder (mirror writing, etc.). Very noticeable is the egocentric prevalence due to the failure of allocentric orientation and action. This is interpreted as a result of taking as a reference only the neutral model of the body (illusion of neutral position). The dyspraxia in the requested actions comes from the instability of the body schema. It is worth mentioning the defect in the initiation of praxis as well as the defect in constructional praxis, this varying according to the degree of automation of the actions, praxis discontinuity being the most characteristic phenomenon. The various disorders of the praxis model correspond to various forms of apractognosia with certain general defects such as neutral illusion, unawareness of the defect and constructional deficit. The gradual series in apractognosia shows functional unity, there is continuity between phenomena of different functional levels and there is no opposition between sensory and

gnostic functions, but they all show the same pattern of organization and are part of a functional growth (growth of the sensory field).

The schema in manual touch constitutes a particular aspect of the body schema. Tactile recognition of objects is impaired due in part to distal tactile disorder affecting manual activity, being spontaneously compensated by facilitation by muscular effort and pressure in handling objects, although errors in manipulation remain in special tests. Object recognition can undergo stereognosis reduction and stereognosis transformation, and at a higher level, a defect in the tactile schema (asymbolia). Tactile recognition goes through different phases (analytical, deductive, etc.), and there is fragmentation of the function, indirect recognition, etc. The typical fragmentation (discontinuity), constructional inability, etc. of the agnostic condition is the result of a reduction in the dimension of the schema, schema that becomes “small” and does not encompass the whole, but only independent parts. This is no different from the alteration of other dimensions of the sensory field; there is in fact a change of scale.

Finally, several controversies and problems about tactile agnosia are easily clarified by the facts and concepts of the brain dynamics presented here. Thus, the disproportion of impairment between the higher and lower functions is consistent with a logarithmic asynchrony, and the multisensory disorders with the central syndrome described here. Therefore, this concept of brain dynamics is supported by its own data and by issues raised outside of it.

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## References of volume 2

- Adrian E. D. (1928), "The Basis of Sensation". London: Christophers 1928.
- Adrian E. D. (1932), "The Mechanism of Nervous Action". Oxford: Milford, London and Oxford University Press 1932.
- Adrian E. D. (1947), "The physical background of perception." Oxford: Clarendon Press 1947.
- Altenburger H. (1933), Sensible Chronaxie. *Deutsche Zeitschrift für Nervenheilkunde (Journal of Neurology)*, **129**: 219-247; Chronaxie. *Deutsche Zeitschrift für Nervenheilkunde*, **129**: 269.
- Altenburger H. (1936/37), Electrodiagnostik einschliesslich. Aktionströme und Chronaxie. *Handbuch der Neurologie* III. Bumke, Foerster (Eds.), Berlin: Springer.
- André-Thomas J., Courjon J. (1917), Hémianesthésie par lésion du lobe pariétal. *Revue Neurologique*, **2**: 269-273.
- Anrep G. V. (1923), Irradiation of conditioned reflexes. *Proceedings of the Royal Society of London*. B, **94**: 404-425.
- Anton G. (1893), Beiträge zur klinischen Beurteilung zur Localisation der Muskelsinnstörungen im Grosshirn. *Z. Heilk.*, **14**: 313-348.
- Anton G. (1898), Über Herderkrankungen des Gehirnes, welche von Patienten selbst nicht wahrgenommen worden. *Wiener Klinische Wochenschrift*, **11**: 227-229.
- Anton G. (1899), Über die Selbstwahrnehmung der Herderkrankungen des Gehirns durch den Kranken bei Rindenblindheit und Rindentaubheit. *Archiv für Psychiatrie und Nervenkrankheiten*, **32**: 86-127.
- Aristoteles (384-322 B.C.), "De Anima", II, III. In: McKeon R. (Ed.), "Introduction to Aristotle". New York: The modern Library, 1947.
- Aubert H. (1865), "Physiologie der Netzhaut". Breslau: Morgenstern 1865.
- Babinski J. (1914), Contribution à l'étude des troubles mentaux dans l'hémiplégie cérébrale (anosognosie). *Revue Neurologique*, **27**: 845-847.
- Babinski J. (1918), Anosognosie. *Revue Neurologique*, **31**: 365-367.
- Benary W. (1922), Studien zur Untersuchung der Intelligenz bei einem Fall von Seelenblindheit. *Psychologische Forschung*, **2**: 208-297.
- Benussi V. (1913), Kinematohaptische Erscheinungen, *Archiv für die gesamte Psychologie*, **29**: 385-388.
- Benussi V. (1916), Versuche zur Analyse taktil erweckter Scheinbewegungen. *Archiv für die gesamte Psychologie*, **36**: 59-135.
- Beringer K. (1923), Experimentelle psychosen durch mescaline. *Zeitschrift für die gesamte Neurologie und Psychiatrie*, **84**: 426-432.
- Beringer K. (1927), Der Meskalinrausch: seine Geschichte und Erscheinungsweise. Monographien aus dem Gesamtgebiete der Neurologie und Psychiatrie. Berlin: Springer. **49**: 1-315.
- Bogaert L. van (1934), Sur la pathologie de l'image de soi. (Etudes anatomo-cliniques). *Annales Médico-Psychologiques*, **92**: 519-555.
- Bonhoeffer K. (1918), Partielle reine Tastlähmung. *Monatsschrift für Psychiatrie und*

- Neurologie*, **43**: 141-145.
- Bonhoeffer K. (1923), Zur Klinik und Lokalisation des Agrammatismus und der Rechts-Links-Desorientierung. *Monatsschrift für Psychiatrie und Neurologie*, **54**: 11-24.
- Bourguignon G. (1923), "La Chronaxie Chez L'Homme". Paris: Masson 1923.
- Bourguignon G. (1929), Chronaxie sensorielles cutanées chez l'homme normal. *Compt. Rend. Acad. des Scienc.*, **189**: 305.
- Bourguignon G. (1932), La chronaxie du nerf et du point moteur musculaire dans les repercussions en pathologie humaine et expérimentale. Deuxieme Congres international de Pathologie comparée 1932.
- Bourguignon G. (1933), Interpretation des sensibilités thermique et douloureuse a l'aide des chronaxies sensitives cutanées normales et de leurs variations dans la syringomyélie. *Compt. Rend. Acad. des Scienc. (Paris)*, **197**(15): 792-794.
- Brecher G. A. (1937), Die Momentgrenze im optischen Gebiet. *Z. Biol.*, **98**: 232-247.
- Bremer F. (1930 a), Nouvelles recherches sur la summation centrale. *Compt. Rend. Soc. Biologie*, **14**: 810.
- Bremer F. (1930 b), La summation d'influx nerveux dans l'arc réflexe spinal. *Compt. Rend. Soc. Biologie*, **13**: 508.
- Buscaino V. M. (1946), "Neurobiologia delle percezioni". Napoli: Ed. scientifica italiana 1946.
- Cassirer E. (1923, 1925, 1929), "Philosophie der symbolischen Formen": 1. Die Sprache, 1923; 2. Das mythische Denken, 1925; 3. Phänomenologie der Erkenntnis, 1929. Berlin: Bruno Cassirer.
- Conrad K. (1932), Versuch einer psychologischen Analyse des Parietal-syndroms, *Monatsschrift für Psychiatrie und Neurologie*, **84**: 28-97.
- Conrad K. (1933), Das Körperschema. Eine kritische studie und der versuch einer revision. *Zeitschrift für die gesamte Neurologie und Psychiatrie*, **174**: 346-369.
- Czermak J. N. (1852), "Über den Raumsinn". Berichte über die Verhandlungen der Koniglich-Sachsichen Gesellschaft der wissenschaften zu Leipzig. Mathematisch-Physische Klasse.
- Dejerine J. J. (1907), A propos de l'agnosie tactile. *Revue Neurologique*, **15**: 781-794.
- Dejerine J. J., Dejerine-Klumpke A. M. (1914), "Sémiologie des affections du système nerveux", Paris: Masson 1914.
- Delacroix H. (1930), "Le langage et la pensée". Paris: Librairie Félix Alean 1930.
- Delay J. (1935), "Les Astéréognosies: Pathologie du Toucher". Paris: Masson et Cie 1935.
- Deny G., Camus P. (1905), Sur une forme d'hypocondrie aberrante due a la perte de la conscience du corp. *Revue Neurologique*, **13**: 460-467.
- Dusser de Barenne J. G. (1913), Zur Kenntnis der Alloasthesie. Experimente und Betrachtungen. *Monatsschrift für Psychiatrie und Neurologie*, **34**: 523-540.
- Dusser de Barenne J. G. (1924), Experimental Researches on Sensory Localization in the Cerebral Cortex of the Monkey (Macacus). *Proc. Roy. Soc. Lond. B*, **96**: 272-291.
- Egger M. (1899), De la sensibilité osseuse. *Journal de Physiologie et de Pathologie Général*, **1**: 511-520.

- Exner S. (1894), "Entwurf zu einer physiologischen Erklärung der psychischen Erscheinungen". Wien: Deuticke 1894.
- Fechner G. T. (1860), "Elemente der Psychophysik". Leipzig: Breitkopf und Härtel 1860.
- Fischer B., Pötzl O. (1924), Zur Symptomatologie der Sensibilitätsstörungen von cerebralem Typus Eine noch nicht bekannte cutane Sensibilitätsstörung von zentralem Typus. *Zeitschrift für die gesamte Neurologie und Psychiatrie*, **88** (1): 58-76.
- Foerster O. (1903), Ein Fall von elementärer allgemeiner Somatopsychose. (Afunktion der Somatopsychie). Ein Beitrag zur Frage der Bedeutung der Somatopsychie für das Wahrnehmungsvermögen. *Monatsschrift für Psychiatrie und Neurologie*, **14**: 189-205.
- Foerster O. (1916), Die Topik der Sensibilitätsstörungen bei Unterbrechung der sensiblen Leitungsbahnen. *Neurologisches Zentralblatt*, **35**: 807-810.
- Foerster (1930), Klinisches. *Journal of Neurology*, **115** (4-6): 248-314.
- Foerster O. (1936), Die Bedeutung des Hinterstranges für die taktile Sensibilität. *Handbuch der Neurologie*, Bumke, Foerster (Eds.), Berlin: Springer. p 359-363. Motorische Felder und Bahnen: sensible corticale Felder. *Handbuch der Neurologie*, Bumke, Foerster (Eds.), Berlin: Springer. IV: 49-56.
- Foix CH. (1922), Sur une variété de troubles bilatéraux de la sensibilité par lésion unilatéral du cerveau. Anesthésie par agnosie (anesthéso-agnosie) avec prédominance des troubles de la notion position (atopognosie) constituant, par leur association a une hémiplegie et une aphasie d'intensités variables et à l'apraxie idéo-motrice, un syndrome caractéristique des lésions profondes du lobe pariétal. *Revue Neurologique*, **1**: 322-331.
- Frey M. von (1910, 1913), Physiologie der Sinnesorgane der menschlichen Haut. *Monatsschrift Kinderheilkunde*, **9** (1): 351-368 (1910); **13** (1): 96-124 (1913).
- Frey M. von (1916/1917), Zur Frage der Kraftempfindungen. *Zeitschrift für Biologie*, **67**: 484-487.
- Frey M. von (1918), Über Bewegungswahrnehmungen und Bewegungen in resezierten und in anästhetischen Gelenken. *Zeitschrift für Biologie*, **68**: 339-350.
- Frey M. von (1928), Die Gliederung des Tastsinns. *Deutsche Zeitschrift für Nervenheilkunde*, **101**: 155.
- Fuchs A. (1908), Neurologische Kasuistik: Eigentümliche Sensibilitätsstörung (Tastlähmung) bei Polyneuritis. *Wiener Klinische Wochenschrift*, **21**: 1180-1183.
- Gelb A. (1925/1926), Die psychologische Bedeutung pathologischer Störungen der Raumwahrnehmung. 9 Kongr. exp. Psychologie.
- Gelb A. (1937), Zur medizinischen Psychologie und philosophischen Anthropologie. *Acta Psychologica*, **3**: 194-271.
- Gelb A., Goldstein K. (1922), Psychologische Analysen hirnpathologischer Fälle auf Grund von Untersuchungen Hirnverletzter: VII. Über Gesichtsfeldbefunde bei abnormer "Ermüdbarkeit" des Auges (sog. "Ringskotome"). *Albrecht von Graefes Archiv für Ophthalmologie*. (*Graefe's Archive for Clinical and Experimental Ophthalmology*), **109**: 387- 403.
- Gelb A., Goldstein K. (1923), Über eine eigenartige Sehstörung (Dysmorphopsie) infolge

- von Gesichtsfeldeinengung. *Psychologische Forschung*, **4**: 38.
- Gelb A., Goldstein K. (1925), Psychologische Analysen hirnpathologischer Fälle: X. Über Farbennamen-Amnesie nebst Bemerkungen über das Wesen der amnestischen Aphasie überhaupt und die Beziehung zwischen Sprache und dem Verhalten zur Umwelt. *Psychologische Forschung (Physiological Research)*, **6**: 127-186.
- Gerstmann J. (1918). Reine taktile Agnosie. *Monatsschrift für Psychiatrie und Neurologie*, **44**: 329-343. [Pure tactile agnosia. *Cognitive Neuropsychology*, **18**: 267-274 (2001)].
- Gerstmann J. (1924), Fingeragnosie: Eine umschriebene Störung der Orientierung am eigenen Körper. *Wiener klinische Wochenschrift*, **37**: 1010-1012.
- Gerstmann J. (1927), Fingeragnosie und Agraphie, ein neues Syndrom. *Zeitschrift für die gesamte Neurologie und Psychiatrie*, **108**: 152-177.
- Gerstmann J. (1930), Zur Symptomatologie der Hirnläsionen im Uebergangsbereich der unteren Parietal- und mittleren Occipitalwindung (Das Syndrom: Fingeragnosie, Rechts-Links-Störung, Agraphie, Akalkulie). *Nervenarzt*, **3**: 691-695.
- Gerstmann J. (1931), Zur lokaldiagnostischen Verwertbarkeit des Syndroms: Fingeragnosie, Rechts-Links-Störung, Agraphie, Akalkulie. *Jahrbuch für Psychiatrie und Neurologie*, **48**: 135-143.
- Gerstmann J. (1940), Syndrome of finger agnosia, disorientation for right and left, agraphia and acalculia. *Archives of Neurology and Psychiatry*, **44**: 398-407.
- Goldscheider A. (1886), Zur Dualität des Temperatursinnes. *Pflüger's Archiv (Archiv für die gesamte Physiologie des Menschen und der Tiere)*, **39**: 96 -120.
- Goldscheider A. (1889), Untersuchungen über den Muskelsinn. *Archiv für Anatomie und Physiologie*, **3**: 369-502.
- Goldscheider A. (1917), Weitere Mitteilungen zur Physiologie des Sinnesnerven der Haut. *Pflüger's Archiv*, **168**: 36-88.
- Goldscheider A. (1925), Beitrag zur Lokalisation der Tastempfindungen. *Archiv für die gesamte Physiologie*, Berlin **209**: 518-525.
- Goldstein K. (1915), Ein Beitrag zur Lehre von der Bedeutung der Insel für die Sprache und der linken Hemisphäre für das linksseitige Tasten. *Archiv für Psychiatrie und Nervenkrankheiten*, **55**: 158-173.
- Goldstein K. (1925), Das Symptom, seine Eutstehung und Bedeutung. *Archiv für Psychiatrie und Neurologie*, **76**: 84-108.
- Goldstein K. (1926), Neuere Erfahrungen zum Problem der sogenannten induzierten Tonusveränderungen. Gleichzeitig ein Beitrag zur Symptomatologie der Zerebellar und Frontaler krankungen. *Deutsche Zeitschrift für Nervenheilkunde. (Journal of Neurology)*, **89** (1-3): 72-77.
- Goldstein K., Gelb A. (1918), Psychologische Analysen hirnpathologischer Fälle auf Grund von Untersuchungen Hirnverletzter. I. Abhandlung: Zur Psychologie des optischen Wahrnehmungs und Erkennungsvorganges. *Zeitschrift für die gesamte Neurologie und Psychiatrie*, **41**: 1-142.
- Goldstein K., Gelb A. (1919), Psychologische Analysen hirnpathologischer Fälle auf Grund von Untersuchungen Hirnverletzter. II. Abhandlung: Über den Einfluss des

- vollständigen Verlustes des optischen Vorstellungsvermögens auf das taktile Erkennen. Zugleich ein Beitrag zur Psychologie der taktilen Raumwahrnehmung und der Bewegungsvorstellungen. *Zeitschrift für Psychologie*, **83**: 157-250.
- Gonzalo J. (1935), Sobre la localización y fisiopatología del tálamo y del subtálamo. *Archivos de Neurobiología* **XV** (4): 625–668.
- Grünbaum A. A. (1930), Aphasie und Motorik. *Zeitschrift für die gesamte Neurologie und Psychiatrie*, **130**: 85-412.
- Guillain G. (1905), Revue générale sur la pathologie du système nerveux. *L'année psychologique*, **12**: 624-635
- Guillain G., Bize P. R. (1932), Astereognosie pure par lésion corticale pariétale traumatique. *Revue Neurologique*, **39**: 502-509.
- Hagen F. W. (1846), "Psychologie und Psychiatrie". Wagners Handwörterbuch der Physiologie. Braunschweig.
- Hahn H. (1928), Über den Erregungsvorgang der Temperaturnerven. *Archiv für die gesamte Psychologie*, **65**: 41.
- Hartmann H., Schilder P. (1927), Körperinneres und Körperschema. *Zeitschrift für die gesamte Neurologie und Psychiatrie*, **109** (1): 666- 675.
- Head H., Riddoch G. (1920), Sensory disturbances in the hand following injuries of the cerebral cortex. *British Medical Journal*, **2**: 782-783.
- Head H., Rivers W. H. R., Holmes G., Sherren J., Thompson T., Riddoch G. (1920), "Studies in neurology". 2 vols., London: Oxford Univ. Press 1920.
- Head H., Holmes G. (1911/1912), Sensory disturbances from cerebral lesions. *Brain*, **34**: 102-254.
- Heilbronner K. (1905), Zur Frage der motorischen Asymbolie (Apraxie). *Zeitschrift für Psychologie und Physiologie der Sinnesorgane*, **39**: 161- 205.
- Heilbronner K. (1910), Die aphasischen, apraktischen und agnostischen Störungen. *Handbuch der Neurologie*, Lewandowsky M. (Ed.) Berlin: Springer 1910, p. 982-1092.
- Helmholtz H. von (1896), "Handbuch der physiologischen Optik", Leipzig: Leopold Voss. Published in parts from 1856 to 1866, later published in full in 1867 as: Vol. IX Allgemeinen Encyclopädie der Physik, Gustav Karsten (Ed.), second revised edition, Leipzig: Leopold Voss 1896.
- Henri V. (1898), Die Raumwahrnehmungen des Tastsinnes. *Mind*, **7** (28): 547-552.
- Henri V. (1900), La notion de l'espace et le sens du toucher. *Revue Neurologique*, p.74.
- Henschen S. E. (1923), 40 jähriger Karapf um das Sohzentrum und seine Bedeutung für die Hirnforschung. *Zeitschrift für die gesamte Neurologie und Psychiatrie*, **87**: 1-617.
- Henschen S. E. (1926), Die Vertretung der beiden Augen in der Sehbahn und in der Sehrinde. *Graefes Archiv Ophthalmologie*, **117**: 419-459.
- Hering E. (1874/1878), Grundzüge einer Theorie des Farbensinnes. Sitzungsber Wiener Akad. 15 Mai, 1874. In "Zur Lehre vom Lichtsinne. Sechs Mitteilungen", Wien 1878. ("On the Theory of Sensibility to Light". Vienna 1878).
- Hering E. (1880), Zur Erklärung der Farbenblindheit aus der Theorie der Gegenfarben. *Lotos* (Prague), NF 1880, **1**: 3-34.



- Hermann G., Potzl O. (1928), "Die optische Allaesthesie". Berlin: Karger 1928.
- Higier H. (1916), A paralytic equivalent of genuine epilepsy. *Deutsche Zeitschrift für Nervenheilkunde*, **55**: 95-100.
- Hoefler P., Kohlrausch A. (1924), Über die Schwellenempfindung an Schmerzpunkten der Haut. *Archiv für die gesamte Psychologie*, **205**: 438-442.
- Hoff H., Schilder P. (1927), "Die Lagereflexe des Menschen". Wien: Springer 1927.
- Hoff H., Pötzl O. (1935), Über ein neues parieto-occipitales Syndrom (Seelenfilung des Schauens Störung des Körperschemas Wegfall des zentralen Sehens). *Jahrbuch für Psychiatrie und Neurologie*, **52**: 173- 218.
- Hoorweg J. L. (1892), Über die elektrische Nervenerregung. *Archiv für die gesamte Physiologie*, **52**: 87-108.
- Jackson J. H. (1884), The Croonian lectures on evolution and dissolution of the nervous system. *Br. Med. J.*, **1**: 591-593, 660 - 663, 703-707.
- James W. (1890), "The Principles of Psychology" (2 vols.). New York: Henry Holt 1890.
- Jones E. (1908), The precise diagnostic value of allochiria. *Brain*, **30**: 490 -532.
- Kappers C. U. A. (1921), On structural laws in the nervous system: The principles of neurobiotaxis. *Brain*, **44**: 125-149.
- Katz D. (1920/1925), "Die Erscheinungsweisen der Tasteindrücke". Rostock 1920; 2nd ed.: Der Aufbau der Tastwelt. *Zeitschrift Psychologie Sinnesorgane*, **2**: 1-270 (1925).
- Kiesow F. (1924), Zur Frage nach der Giltigkeit des Weberschen Gesetzes im Gebiete der Tastempfindungen. *Archiv für die gesamte Psychologie*, **47**: 1-13.
- Kleist K. von (1907), Corticale (innervatorische) apraxie. *Jahrbuch Psychiatrie Neurologie*, **28**: 46-112.
- Kleist K. von (1911), Der Gang und der gegenwertige Stand der Apraxieforschung. *Ergebnisse der Neurologie und Psychiatrie*, **1**: 342-452.
- Kleist K. von (1934), "Gehirmpathologie". Leipzig: Barth J. 1934.
- Kleist K. von (1937), Bericht über die Gehirmpathologie in ihrer Bedeutung für Neurologie und Psychiatrie. *Zeitschrift für die gesamte Neurologie und Psychiatrie*, **58**: 159-193.
- Kleist V. K., Gonzalo J. (1938), Über Thalamus und Subthalamussyndrome und die Störungen einzelner Thalamuskern. *Monatsschrift für Psychiatrie und Neurologie*, **99**: 87-130.
- Koehnlein H. (1934), Über das absolute Zeitgedächtnis. *Zeitschrift für Sinnesphysiologie*, **65**, 35-57.
- Koffka K. (1919/1935), "Beiträge zur Psychologie der Gestalt". Bd., 1, Leipzig 1919 ("Principles of Gestaltpsychology". New York 1935).
- Köhler W. (1930), "Gestalt Psychology". London 1930.
- Kramer F. (1915), Alloästhesie und fehlende Wahrnehmung der gelähmten Körperhälfte bei subvertikalem Hirnherd. *Neurologisches Zentralblatt*, **34**: 287-288.
- Kramer F. (1917), Bulbarapoplexie (Verschluss der Arteria cerebelli posterior inferior) mit Alloästhesie. *Zeitschrift für die gesamte Neurologie und Psychiatrie*, **14**: 58-60.
- Külpe O. (1893/1909), "Grundriss der Psychologie", 1893 ("Outlines of Psychology").

- New York: Macmillan and Co. 1909).
- Kutner R. (1907), Die transkortikale Tastlähmung. *Monatsschrift für Psychiatrie und Neurologie*, **21**: 191-205.
- Lange J. (1936), Agnosien und Apraxien. *Handbuch der Neurologie*, Bumke, Foerster (Eds.), Berlin: Springer Verlag 1936, **6**: 807-960.
- Lange J. (1930), Fingeragnosie und Agraphie. *Monatsschrift für Psychiatrie und Neurologie*, **76**: 129-188.
- Lashley K. S. (1929), "Brain mechanisms and Intelligence: A Quantitative Study of Injuries to the Brain". Chicago, Illinois: The Univ. Chicago Press 1929.
- Lewinski L. (1879), Über den Kraftsinn. *Archiv für Pathologische Anatomie und Physiologie und für klinische Medizin*, **77**: 134-146.
- Lhermitte J., Trelles J. O. (1933), Sur l'apraxie pure constructive. Les troubles de la pensée spatiale et de la somatognosie dans l'apraxie. *Encéphale*, **28**: 413-444.
- Lhermitte J. (1939), "L'image de notre corps". *Nouvelle Revue Critique, Paris*.
- Liepmann H. (1900), Das Krankheitsbild der Apraxie (motorische Asymbolie) auf Grund eines Falles von einseitiger Apraxie. *Monatsschrift für Psychiatrie und Neurologie*, **8**: 15-44, 102-132, 182-197.
- Liepmann H. (1909), Agnostische Störungen. Taktile Agnosie (Tastlähmung). In "Lehrbuch der Nervenkrankheiten", Curshmann H. (Ed.), Berlin: Springer 1909, p 486.
- Liepmann H. (1920), Apraxie. In "Ergebnisse der gesamten Medizin", Brugsch H. (Ed.), Berlin, Wien: Urban, Schwarzenberg 1920, p 516- 543.
- Lotze R. H. (1852), "Medizinische Psychologie, oder Physiologie der Seele". Leipzig: Weidmann 1852.
- Lüers T., Pötzl O. (1941), Über "Verkehrtsehen" nach Insult. *Wiener klinische Wochenschrift*, **53**: 625-628.
- Mach E. (1886/1906), "Beiträge zur Analyse der Empfindungen". Jena 1906.
- Magnus-Alsleben E. (1924), Über sensibilitätsprüfungen bei Wernickescher Tastlähmung. 49<sup>o</sup> Verhandl. Südwest deutsch. Neurol. u. Psychiatrie zu Baden-Baden 1924. *Zentralb. für die gesamte Neurologie und Psychiatrie*, **37**: 330.
- Marburg O. (1931), Scheitellappenerweichung unter dem Bilde eines Pseudotumor cerebri (Zugleich ein Beitrag zur Frage der Fingeragnosie ohne Agraphie). *Arb. aus. d. Neurol. Inst.*, **33**, f. **1**: 1-13.
- Marie P., Bouttier H. Bogaert L. van (1924), Sur un cas de tumeur préfontale droite. Troubles de l'orientation dans l'espace. *Revue Neurologique*, **31**: 209-221.
- Minskowski M. (1917), Etude sur la physiologie des circonvolutions rolandiques et parietalis. *Schweiz. Archive für Neurologie und Psychiatrie*, **1**: 389-459.
- Monakow C. von (1905), "Gehirnpathologie", Ed. 2. Wien: Holder 1905.
- Morlaàs J. (1928), "Contribution a l'étude de l'apraxie". These de médecine, Paris 1928.
- Mott F. W. (1892), Results of hemisection of the spinal cord in monkeys. *Philosophical Transactions of the Royal Society, London B*, **183**: 1-59.
- Müller G. E. (1903), Die Gesichtspunkte und die Tatsachen der psychophysischen Methodik. In "Ergebnisse der Physiologie" Asher L., Spirok K. (Eds.), Jahrg. 11,

- 1903, **2**: 267-519.
- Obersteiner H. (1892), On allochiria. *Brain*, **4**: 153-163.
- Oppenheim H. (1906), Über einen bemerkenswerten Fall von Tumor cerebri. *Berlin klinische Wochenschrift*, **43**: 1001-1004.
- Pavlov I. P. (1927), "Conditioned Reflexes: An Investigation of the Physiological Activity of the Cerebral Cortex". Anrep G. V. (Transl. and Ed.). London: Oxford Univ. Press 1927.
- Pick A. (1905), "Studien über motorische Apraxie und ihre nahestehende Erscheinungen". Leipzig: Deuticke 1905.
- Pick A. (1908 a), Über eine besondere Form von Orientierungsstörung und deren Vorkommen bei Geisteskranken. *Deutsche medizinische Wochenschrift*, **47**: 2014-2017.
- Pick A. (1908 b), "Über Störungen der Orientierung am eigenen Körper". Arbeiten aus der deutschen psychiatrischen Universitäts-klinik in Prag. Berlin: Karger 1908, p 1-19.
- Pick A. (1915), Zur Pathologie des Bewußtseins vom eigenen Körper. Ein Beitrag aus der Kriegsmedizin. *Neurologisches Zentralblatt.*, **34**: 257-265.
- Pick A. (1922), Störung der Orientierung am eigenen Körper. Beitrag zur Lehre von Bewußtsein des eigenen Körpers. *Psychologische Forschung*, **1**: 303-318.
- Piéron H. (1935), Le Toucher. In "Nouveau Traité de Physiologie normal et pathologique", Dumas G. (Ed.) Paris: Masson 1935, p 1055-1228.
- Piéron H. (1936), Recherches expérimentales sur la sensation vibratoire cutanée. *Année Psychologique*, **3**: 82-102.
- Piéron H., Segal J. (1938), La sensibilité tactile digitale étudié par stimulation électrique du nerve cutanée. *Compt. Rend. Soc. de Biologie*, **228**: 762.
- Pineas H. (1926), Der Mangel an Krankheitsbewusstsein und seine Variationen als Symptom organischer Erkrankungen. *Nervenarzt*, **16**: 238-248.
- Planter E. (1793) "Philosophische Aphorismen". Leipzig: Schwicker 1793.
- Poggio E. (1908), Die kortikale Lokalisation der Asymbolie. *Neurologisches Zentralblatt*, **27**: 817-818.
- Ponzo M. (1910), Intorno ad alcune illusioni nel campo delle sensazioni tattili, sull'illusione di Aristotele e fenomeni analoghi. *Archiv für die gesamte Psychologie*, **16**: 307-345.
- Poppelreuter W. (1917), "Die Psychischen Schädigungen durch Kopfschuss in Kriege 1914-1916. Die Störungen der niederen und höheren Schleitungen durch Verletzungen des Occipitalhirns". Leipzig: Voss 1917.
- Porta G. B. della (1589/1593), "De refractione optices". Naples: Salviani 1589.
- Pötzl O. (1924), Über die Störungen der Selbstwahrnehmung bei linksseitiger Hemiplegie. *Zeitschrift für Neurologie und Psychiatrie*, **93**: 117-168.
- Priestley J. (1777), "Disquisitions relating to Matter and Spirit". London: Johnson J. 1777.
- Ramón y Cajal S. (1898), Estructura del quiasma óptico y teoría general de los entrecruzamientos nerviosos. *Revista trimestral Micrográfica*, **1**, Marzo
- Ramón y Cajal S. (1899), "Textura del Sistema Nervioso del Hombre y de los

- Vertebrados". Tomo II, Madrid 1899.
- Ranschburg P., Schill E. (1932), Über Alexie und Agnosie. *Zeitschrift für die Gesamte Neurologie und Psychiatrie*, **139**: 192-240.
- Raymond F., Egger M. (1906), Un cas d'aphasie tactile. *Revue Neurologique*, **14**: 371-375.
- Redlich E. (1915), Zur Topographie der Sensibilitätsstörungen am Rumpfe bei der zerebralen Hemianästhesie. *Neurologisches Zentralblatt*, **22**.
- Redlich E., Bonvicini G. (1909), Über das Fehlen der Wahrnehmung der eigenen Blindheit bei Hirnkrankheiten. *Jahrbuch für Psychiatrie*, **29**: 1-133.
- Redlich E., Bonvicini G. (1911), Weitere klinische und anatomische Mitteilungen über das Fehlen der Wahrnehmung der eigenen Blindheit bei Hirnkrankheiten. *Neurologisches Zentralblatt*, **30**: 227-235.
- Révész G. (1926), Taktile Gegenstandswahrnehmung und Gestaltbildung. Ber., 8., Internat. Kongress f. Psychol., 1926.
- Rey A. (1947), "Observations sur la douleur cutanée étudiée par la méthode stroboscopique". *Miscellanea psychologica Albert Micholte*, p 254-267.
- Romberg M. H. (1846/1853), "Lehrbuch der Nervenkrankheiten des Menschen". Bd. 1. Berlin: Duncker A. 1846, p 795 (English translation, London: The Sydenham Society 1853).
- Rothmann M. (1914), Über die Grenzen der Extremitätenregion der Grosshirnrinde. *Monatsschrift für Psychiatrie und Neurologie*, **36**: 319- 341.
- Rupp H. (1912), Systematische Uebersicht der Eigenschaften des Gedächtnisses. Ber. Über d. V. Kongress f. Exper. Psychol. 1912, 211-216.
- Scheiner C. (1619), "Oculus". Innsbruck 1619.
- Scheller H., Seidmann H. (1932), Zur Frage der optisch-räumlichen Agnosie. *Monatsschrift Psychiatrie Neurologie*, **81**: 97-188.
- Schilder P. (1923/1935), "Das Körper Schema. Ein Beitrag zur Lehre von Bewusstsein des eigenen Körpers". Berlin: Springer J. 1923. ("The image and appearance of the human body". Psych. Monographs, n. 4, London: Kegan, Trench, Trubner and Co. 1935).
- Schilder P. (1931), Fingeragnosie, Fingerapraxie, Fingeraphasie. *Nervenarzt*, **4**: 625-629.
- Schilder P., Stengel E. (1931/1932), Schmerzsymbolie. *Zeitschrift für die gesamte Neurologie und Psychiatrie*, **132**: 367-370. (Asymbolia for pain. *Archives of Neurology and Psychiatry*, **25**: 598-600).
- Schlesinger B. (1928), Zur Auffassung der optischen und konstruktiven Apraxie. *Zeitschrift für die gesamte Neurologie und Psychiatrie*, **117**: 649-697.
- Schriever H. (1929), Untersuchungen über die wechselseitige Verstärkung von Schmerz. *Zeitschrift für Biologie*, **88**: 487-515.
- Schriever H., Cebulla R. (1938), Über Erregbarkeitsänderungen des Nerven beim Übergang. *Pflügers Archiv für die gesamte Physiologie*, **241**: 1-37.
- Sherrington C. S. (1906), "The integrative action of the nervous system". 2nd ed., New York: Scribner's 1906.
- Siekman W. (1932), Psychologische Analyse des Falles Rat. *Psychologische Forschung*, **16**: 201-250.

- Sitting O. (1931), Über Apraxie. Eine Klinische Studie. *Abh. aus der Neur. Psychiat. Psych. und ihre Grenzen*, Berlin: Karger, Verlag 1931, **63**: 1-248.
- Skramlik E. R. von (1937), "Psychophysiologie der Tastsinne". **1** Auflage, Leipzig: Akademische Verlag 1937.
- Spearman C. E. (1907), An 'economic' theory of spatial perception. *Mind*, **16**: 181-196.
- Stauffenberg W. von (1914), Über Seelenblindheit. *Arbeiten aus dem himanat. Inst. Zurich*, **8**: 1- 212.
- Stauffenberg W. von (1918), Klinische und anatomische Beiträge zur Kenntnis der aphasischen, agnostischen und apraktischen Symptome. *Zeitschrift für die gesamte Neurologie und Psychiatrie*, **39**: 71.
- Stein J. (1928), Über die Veränderung der Sinnesleistungen und die Entstehung von Trugwahrnehmungen. In "Pathologie der Wahrnehmung. Handbuch der Geisteskrankheiten", Bumke O. (Ed.), Bd **I**, Allgemeiner Teil I, Berlin: Springer 1928. p 351-426.
- Stein J. (1930), Physiologie und Pathologie der Sensibilität. *Fortschr. d. Neurol. u. Psychiat.*, **2**.
- Stein J., Weizsacker V. von (1926), Über klinische sensibilitätsprüfungen. *Deutsche Archiv für klinische Medizin*, **151**: 230-253.
- Stein H., Weizsacker V. von (1927), Der Abbau der sensiblen Funktionen. *Deutsche Zeitschrift für Nervenheilkunde*, **99**: 1-30.
- Stein H, Weizsacker V. von (1928), Zur Pathologie der Sensibilität. *Reviews of Physiology, Biochemistry and Pharmacology*, **27** (1): 657-708.
- Stockert F. G. von (1934), Lokalisation und klinische Differenzierung des Symptomes der Nichtwahrnehmung einer Körperhälfte. *Deutsche Zeitschrift für Nervenheilkunde*, **134**: 1-13.
- Stratton G. M. (1896), Some preliminary experiments on vision without inversion of the retinal image. *Psychological Review*, **3**: 611-617.
- Stratton G. M. (1897), Upright vision and the retinal image. *Psychological Review*, **4**: 182-187. Vision without inversion of the retinal image. *Psychological Review*, **4**: 341-360, 463-481.
- Strauss H. (1924), Über konstruktive Apraxie. *Monatsschrift für Psychiatrie*, **56**: 65-124.
- Strümpell A. (1918), Die Stereognose durch den Tastsinn und ihre Störungen. *Deutsche Zeitschrift für Nervenheilkunde*, **60**: 154-168.
- Stumpf C. (1873), "Über den psychologischen Ursprung der Raumwahrnehmung". Leipzig: Hirzel 1873.
- Tastevin J. (1937), En partant de l'experience d'Aristote. *L'Encéphale*, **32**: 57-84, 140-158.
- Thiele R. (1928), Aphasie, Apraxie, Agnosie. In "Handbuch der Geisteskrankheiten herausgeg eben", Bumke O. (Ed.), Bd. **II**, Allgem. Teil 11, S. 243. Berlin: Springer 1928.
- Titchener E. B. (1910), "Text-book of Psychology". New York: Macmillan 1910.
- Villaret M. (1916), Le syndrome nerveux de l'espace rétroparotidien postérieur. *Revue Neurologique* (Paris), **29**: 188-190.
- Vouters L. (1909), "Sur l'agnosie tactile" - travail du laboratoire du professeur Dejerine a

- la Salpêtrière (Paris - G. Steinheil - 1909).
- Wagner W. (1932), Über Raumstörung. *Monatsschrift für Psychiatrie und Neurologie*, **84**: 281-307.
- Weber, E. H. (1834/1846), "De subtilitate tactus", 1834. "Der Tastsinn und das Gemeingefühl", 1846. (Translation: "The sense of touch". London: Academic Press 1978).
- Weizsacker V. von (1931), Kasuistische Beiträge zur Lehre von Funktionswandel bei statoopto-sensiblen Syndromen. *Deutsche Zeitschrift für Nervenheilkunde*, **117**.
- Weizsacker V. von (1923), Über den Funktionswandel besonders des Drucksinnes bei organisch Nervenkranken und über Beziehungen zur Ataxie. *Pfüngers Archiv*, **201**: 317.
- Weizsacker V. von (1924), Über eine systematische Raumsinnesstörung. *Deutsche Zeitschrift für Nervenheilkunde*, **84**.
- Wernicke C. (1895), Zwei Fälle von Rindenläsion. Ein Beitrag zur Localisation der Vorstellungen. *Arbeiten der Psychiatrischen Klinik in Breslau*, **2**: 33-53.
- Woerkom W. van (1925), Über Störungen in Denken bei Aphasiepatienten. *Monatsschrift für Psychiatrie und Neurologie*, **59**: 256-277.
- Wundt W. (1862), "Beiträge zur Theorie der Sinneswahrnehmung". Leipzig: Winter C. F. 1862.
- Wundt W. (1874), "Grundzüge der physiologischen Psychologie". Leipzig: Engelmann 1874.
- Zutt J. (1932), Rechts-Links-Störung, konstruktive Apraxie und reine Agraphie. *Monatsschrift für Psychiatrie und Neurologie*, **82**: 235-305, 355-395.