Brain Dynamics

by Justo Gonzalo

Volume 1

Edited by Isabel Gonzalo Fonrodona

Optics Department. Faculty of Physics. Complutense University of Madrid

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Translated from Spanish by Isabel Gonzalo Fonrodona and Fernando Aymat Olasolo

BRAIN DYNAMICS

The brain activity according to the dynamic conditions of nervous excitability

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

Preface

This book is the English translation of Volume 1 of the book written in Spanish by Justo Gonzalo Rodríguez-Leal (Barcelona 1910 – Madrid 1986), first published in 1945. At present, there is only the Spanish facsimile edition of 2010, including two supplements[a]. The interest of the research described in this book lies 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, or have only been observed in the last decades, and that the functional dynamic unity of the cortex proposed by the author is closely related to the current trends in the study of the brain. Some singular phenomena are described with extreme detail, such as inverted vision, facilitation, influence of stimulus intensity, delocalization of colors, reversal of motion, and orientation disorder, among others.

Once the author finished his studies of medicine, he completed his training in neurology at the University of Vienna (1933-34) and in brain pathology at the University of Frankfurt (1934-35). The research described here began in the middle of the Spanish Civil War (1936-1939) at a brain injury center in Valencia (Spain), and was later continued at the Ramón y Cajal Institute and at the Spanish National Research Council in Madrid.

From the study of brain-injured patients with unilateral lesion in an association area in the left parieto-occipital cortex, equidistant from the visual, tactile and auditory primary areas, the author characterized a multisensory, bilateral and symmetrical disorder that he called *central syndrome.* This is a multisensory (visual, tactile, auditory) alteration with the lesion not involving the specific areas, all functions being affected, from simple excitability to more complex functions, bilaterally and symmetrically, all of which being incompatible with the rigid traditional theory of brain localization. A phenomenon of clear dynamic character, which depends on the intensity of the stimulus, is the separation or disaggregation of a sensory function into partial functions or qualities that are united in normal perception. Different qualities are gradually lost according to their demands of nervous excitability when the intensity of the stimulus decreases, thus revealing the different functions that make up the sensorium, including visual image orientation as an unexpected function. Thus, inverted or tilted vision appears, among other disorders. The first in-depth study of tilted or inverted vision is part of the present research (pages 142-185). A related dynamic phenomenon is partial disappearance of the disorders by intensification of the stimulus, or by the striking phenomenon of facilitation according to which the perception of a stimulus improves by the presence of another stimulus of the same or of a different sensory modality (cross-modal effect), or by a motor stimulus,

[a] Gonzalo J. "Dinámica Cerebral", Edición facsímil, Vol. 1 (1945), Vol. 2 (1950) y Suplementos I y II. Red Temática en Tecnologías de Computación Artificial/Natural (RTNAC) and Universidad de Santiago de Compostela (USC), Spain 2010. (In Spanish). Open Access at the official repository of USC: http://hdl.handle.net/10347/4341

muscular effort being one of the most efficient and less known means. For example, a strong muscle contraction can improve perception by straightening the image in the disorder of inverted or tilted visual image. This capability is greater the greater the brain excitability deficit. The first detailed study on multisensory and motor facilitation is also part of this research. This study provides novel observations and an interpretation of this effect. It is remarkable that this type of effect is an extremely current research topic, as evidenced by the large number of scientific articles.

The observation of all these phenomena required a meticulous and careful examination of the patients, since in addition to the fact that these types of patients are usually unaware of their own anomalies, they unconsciously develop facilitation mechanisms that improve their perception.

From the new approach that the author gave to the research, his conception of brain dynamics emerged. To the best of my knowledge, this was the first time that the term *brain dynamics*, so widely used today, was introduced to describe brain mechanisms in relation to sensory organization. As the author pointed out, this research filled the gap then existing between brain pathology and the physiology of the nervous system since the phenomena described find explanation on a physiological basis governed by the laws of nervous excitability. This provided a dynamic solution to the rigid theory of brain localization, and established a continuous transition between lower and higher sensory functions, both being based on the same physiological laws.

Two brain injured patients who differ in the intensity of their disorders are especially studied here. The deep and detailed analysis of these patients allowed the author to get to the root of the issue and to generalize the concept of brain dynamics. In subsequent research, the author found 35 cases that also fit the above-mentioned central syndrome [b]. A reference case in this research is also the famous Schneider case of Goldstein and Gelb studied in 1918, which deserves publications even at present, and which the author interpreted under the central syndrome. As the author pointed out, the scarcity of cases of central syndrome is not due to their exceptionality, since they would be numerous, but rather to the fact that they remain hidden due to the difficulty in the examination of these types of patients.

In this Volume 1, a first part deals qualitatively with general aspects of the research (findings, new syndrome, methodology, etc.), and a second part focuses on the quantitative and experimental aspects concerning visual functions. This part covers electrical and light excitability, color vision, visual field, visual forms, delocalization of colors, acuity, motion perception, motion inversion, orientation of the visual image, and finally visual schema, where some remarkable phenomena concerning spatial orientation are described. A striking example is that these patients were able to read a text whether it was in a normal position or upside down without noticing any difference; another example is the disorder in allocentric orientation. In the study of all these topics, a dynamic analysis is performed taking into account facilitations applied, among other

[b] Gonzalo J. (1952), *Trabajos del Instituto Cajal de Investigaciones Biológicas*, Vol. **XLIV**: 95-157. English translation Open Access at the repository of Complutense University of Madrid: https://eprints.ucm.es/30931/

variables, and always establishing a comparison between the two brain injured patients who presented different loss of neural mass in the same area. Volume 2 (1950) deals with tactile functions and extension of concepts.[c] For example, among other noteworthy phenomena, tactile inversion is observed. Inverted perception is thus generalized in the central syndrome to all sensory systems of a spatial nature. This volume is in process of translation into English.

It is worth mentioning the rich bibliographic documentation on various currents of thought, clinical data, experiments and precedents of the phenomena observed, which adds interest and amenity to the book.

This research received the attention of relevant authors. For example, in a letter to Gonzalo in 1946, W. Köhler (representative of the Gestalt theory) wrote[d]: "*The book contains many observations which are both entirely new and very important. I also believe that at several points your interpretations are more convincing than those of Gelb and Goldstein*". Also, R. Bing (Prof. of neurology at the University of Basel, Switzerland) wrote to Gonzalo in 1946[c] : "*It is a work of the utmost importance and originality, worthy of the traditions established by the immortal sage* (Ramón y Cajal) *to whose memory it has been dedicated*" (translated from French). Soon after, Bender and Teuber, in a work on visual functions[e] stated in 1948: "*Thus far, the American and English literature has failed to produce a monograph similar in scope to Gonzalo's Dinámica Cerebral which was based on experiments with brain injuried casualties of the Spanish Civil War*". Other authors such as J. Ajuriaguerra and H. Hécaen 1949, P. Guiraud 1950, McD. Critchley 1953, devoted attention to this research in their specialized books.

In a later publication[f] 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. Thus, he found allometric relationships between different sensory qualities. These last aspects are briefly presented in the Spanish facsimile edition (Supplement II), and are exposed more recently by other authors[g] placing them in the current context, and

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[c] Engish translation of Volume 2, Open Access at the repository of the Complutense University of Madrid: https://eprints.ucm.es/id/eprint/72118/

[d] Family archive: Letter from W. Köhler to J. Gonzalo, 1946; letter from R. Bing to J. Gonzalo, 1946.

[e] Bender M. B., Teuber H. L. (1948), Neuro-ophtalmology, in: *Progress in Neurology and Psiquiatry*. Spiegel E.A. (Ed), **III**, Chap. 8: 163-182, p 171.

[f] Gonzalo J. (1952), *Trabajos del Instituto Cajal de Investigaciones Biológicas*, Vol. **XLIV**: 95-157. English translation, Open Access at the repository of the Complutense University of Madrid: https://eprints.ucm.es/30931/

[g] Gonzalo Fonrodona I., Porras M. A. (2014), Nervous excitability dynamics in a multisensory síndrome and its similitude with normals. Scaling Laws, in: Costa A., Villalba E. (Eds.) *Horizons in Neuroscience*, Vol. **13**: Chap.10: 161, and references therein, 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

including new developments involving power laws of perception based on clinical data from this book (see also references in that work of 2014).

This research was later echoed in the field of cybernetics and artificial intelligence where some authors[h] add this research to that of K.S. Lashley and A.R. Luria as a neurophysiological basis for the functional organization of the nervous tissue and behavior. 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 the Spanish facsimile edition mentioned above, and whose free online version maintains a significant rate of visits since its publication.

The author conducted the research under the adverse conditions of the Civil War and the time that followed, with great shortage of resources in spite of some financial support from the Cajal Institute and the Spanish National Research Council, the institution to which he belonged. There was no re-edition of the two volumes of the book and, except for two long publications in the journal of the Cajal Institute, there were no subsequent publications in international journals, the author having the project of concluding a new book which no longer came out in spite of the large amount of material prepared for it. However, the author exposed his research in the PhD courses on Brain Physiopathology that he was teaching at the University of Madrid (1945-1966). More data on the author's research are easily accessible through the references contained in an open digital support[i].

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 should be understood in the context of the time it was written, particularly the expressions of a temporal character. The text in the figures has been translated while maintaining the original figures even if some of them are not of high quality.

I acknowledge the permissions obtained from the RTNAC and the University of Santiago de Compostela (publishers of the Spanish facsimile edition in 2010), and I am also especially grateful to Miguel A. Porras for his continued and valuable advice.

Isabel Gonzalo Fonrodona
Optics Department, Faculty of Physics
Complutense University of Madrid
Madrid, Spain, 2021- 2022

[h] 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.

[i] https://en.wikipedia.org/wiki/Justo_Gonzalo

Contents

Color vision

Visual forms

Visual image orientation

Schema in visual perception

General aspects. Fundamental findings and the conception of brain dynamics

In this part, we study the fundamental findings that constitute the starting point for the new conception of brain activity. These findings are the various phenomena of *dynamic action*, found at the end of 1939, which lead to an understanding of brain functions completely different from that sustained by classical theory.

Significant new data lead not only to a new general point of view but also to innovation in working and research methods. We must therefore deal with three outstanding aspects: dynamic action phenomena, a new conception of brain activity and research methods followed. These aspects are completely interdependent since they are based on the same principle, which is developed systematically.

This part on general aspects is necessary both to show the theoretical meaning of this research and to present basic phenomena which will constantly appear in a second part when studying in detail the different sensory functions[1]. In this way, it is possible to maintain a certain balance between a deductive and an inductive exposition that allows a better understanding of the present work.

This exposition on general aspects and their theoretical meaning will find its completion in a third part[2].

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[1] Such second part deals with visual functions in this volumen 1, and with tactile functions in volume 2 (1950), English translation (2022) Open Access: https://eprints.ucm.es/id/eprint/72118/

[2] This third part was not published in spite of the material prepared for it. In addition to Vol. 2 (1950), there are these two later publications by the author:

Gonzalo J., *Trabajos del Instituto Cajal de Investigaciones Biológicas*, **XLIII**: pp. 209-260 (1951). **XLIV**: pp. 95-157 (1952), English translation Open Access: https://eprints.ucm.es/30931/

A brief exposition of the third part is included in Suplemento II of the Spanish facsimile edition (2010). There are more recent works on that part, for example: Gonzalo Fonrodona I., Porras M. A. (2014), *Horizons in Neuroscience* Vol. **13**, Chap.10, pp. 161–189, and references therein. 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

1 Fundamental findings

1.1 Development of the research

The elaboration of the topics we are going to expose has taken a long time; the studies began in the summer of 1938 and continued without interruption up to now, that is, six years. The research was initially directed towards the complex issue of the agnosia syndrome, but starting from new phenomena of agnosia, the initial orientation gradually changed as the mechanism of these phenomena was elucidated in depth. A broader base has been established by achieving a transition from brain pathology to brain physiology. The latter has been developed in an entirely systematic way, since by starting from a small number of fundamental facts it has been possible to carry out a development that encompasses all types of brain activity. Thus, a human brain physiology is established which is directly based on the laws of nervous excitability, and due to the special dynamic conditions that determine such excitability, it can be called *Brain Dynamics*.

New syndromes have constituted the first starting point. Thus, in a certain subject affected by a left parieto-occipital brain injury, who initially seemed to present only a concentric reduction of the visual field, a series of singular disturbances were found in the summer of 1938, such as inverted vision, loss of visual perception of motion, *flat color* vision (Gelb 1920), as well as other more elementary alterations such as greenish *chromatopsia*, etc. The first remarkable phenomenon found in that subject at that time was the perception of flat colors; the last one was inverted vision, a couple of months later. This proves how hidden or ignored these kinds of disturbances can remain for the reasons we shall indicate further on. The study of these phenomena, providing new findings, gradually became more problematic. Thus, a consequence of the inverted vision was the finding of the remarkable phenomenon consisting of the loss of *orthogonal*[3] function in visual shapes. Due to this latter phenomenon, the subject is able to read letters and numbers both in their normal position and upside down, and claiming that the position in both cases is completely identical and normal. Along with these unusual or totally unknown phenomena there were the usual disturbances of visual agnosia in a very diffuse and unstable way. This was a motley complex of bizarre phenomena which, despite giving rise to very significant disturbances, went completely unnoticed at first sight, since they

[3] Orthogonal function means correct orientation on the plane orthogonal to the line of sight.

did not seem to alter the ordinary behavior of the affected individual, who was almost the most unaware of his disorders. However, it was soon discovered that agnosia and other sensory changes were not limited to visual functions; touch and praxias were equally affected, therefore we were confronted to a very special disturbance.

There were precedents for some of these agnosia phenomena, especially concerning the flat color disorder, first described by Gelb (1920) in two patients with occipital damage occurred in World War I, although it has not been studied in detail since then. Some cases of inverted vision had also been observed, already at the beginning of the 20th century, but these were simple notes about this disorder suffered sporadically by certain patients with a brain pathology, without being directly observed and objectively examined by the authors.

Initially, no matter how much interest might raise the issue of the brain localization of those disorders in line with the usual conception of brain pathology, much more attention was paid to the study of the pathogenesis, since very favorable conditions were offered for that study to be carried out. Thus, in the case of inverted vision, it was discovered that an object appearing in a normal position in near vision, was seen gradually tilted when moving away, to the point of getting totally inverted at a certain distance. It was also found that the bigger the size of the object, the greater the distance needed to reach the inversion. These tests, among many others, made the research focus on the physiopathological study, which made possible, after many detours and patient observations, the discovery of nervous phenomena of greater scope and scientific meaning, which gave a new course to the research.

During the summer of 1938, a large amount of different observations on the brain-injured patient in question had been gathered, although their systematization and, above all, their theoretical explanation presented great difficulties. In 1939, attention was paid to the relationships of brain excitability, which were so evident in various experiments about inverted vision. Thus, a strong stimulation, i.e. near vision, resulted in a correct and normal image, whereas a weak stimulation, i.e. far vision, resulted in an inverted image, according to the especial physiological conditions of the patient – what we shall later call functional asynchrony –. Although a decisive step had been taken, there were certain difficulties that needed to be solved before a proper and conclusive explanation could be reached. An important issue to be addressed was the great instability of the phenomena in a short period of time and even within the same examination. A large margin of variation made every meticulous determination difficult and any quantitative assessment useless.

The persevering and in-depth analysis of the aforementioned brain-injured patient, undertaken by the end of 1939, had as a most important result the unexpected discovery of the nervous phenomena of *dynamic action*, leading to a profound transformation of all viewpoints prior to this finding. The physiological criterion of excitability became indispensable, acquiring a fundamental significance since, as it will be seen, the dynamic action phenomena are based on special changes in brain excitability. Consequently, the research was set up on a strictly physiological basis, addressing the study of brain activity in a very different way from that followed by brain pathology from the research of Wernicke (1874) to present time.

Having shown that a single small left parieto-occipital lesion had caused a generalized and uniform alteration of the whole brain, i.e. of all the sensory systems, in both halves of the body, and that the different disorders responded to special dynamic conditions of nervous excitability (cerebral repercussion, asynchrony, summation, etc.), it was no longer possible to look for brain localizations against all logic. This is the negative aspect of the problem, and as for the positive aspect, it was necessary to take advantage of the great prospects offered by the dynamic manifestations of nervous excitability to unravel the fine nervous mechanism of brain functions.

Finally, in 1940, a systematic research plan was established in view of the new facts, which made it possible to successfully address several disciplines which, although close to each other, had always been quite isolated from each other, such as sensory physiology, physiological psychology and brain pathology. All of them should be derived from the physiology of the nervous system and, above all, from the laws of nervous excitability.

In 1941, it was finally possible to establish a conceptual framework that gave rise to the *Brain Dynamics* presented here. However, due to insufficient working resources, a huge task remained to be done in the experimental domain. This task has been performed during the last three years (1942, 1943 and 1944), in which, by means of limited instrumental resources, it has been possible to develop the experimental analysis and make a quantitative assessment of the phenomena. Thus, where before there were only simple descriptions from the clinical observation of patients, now a varied experimentation is carried out, whose results, expressed by means of graphic representations, show in the simplest and most accurate way the physiological laws that govern brain activities.

1.2 Singular precedents on agnosia research

Before setting out the fundamental finding of dynamic action, it is necessary to discuss certain precedents that will help to understand the meaning of the findings. Thus, we must refer to the works of Goldstein and Gelb (1918, 1919) on the well-known case of *Schneider*, a patient with brain injury from the First World War. These authors carried out, around 1918, two important research works on agnosia in this patient. These works have been significant both for the singular observations and for the theoretical consequences that the authors claim. The first one deals with visual function: *Zur Psychologie des optischen Wahrnehmungs und Erkennungsvorganges* (Goldstein and Gelb 1918), a very detailed and extensive study, also highly cited and discussed; the second one addresses tactile functions: *Über den Einfluss des vollständigen Verlustes des optischen Vorstellunsvermöges auf das taktile Erkennen* (Goldstein and Gelb 1919).

With regard to the first of the works, on visual function, the patient with a left parieto-occipital lesion showed an *apperceptive visual agnosia* (blindness to visual forms), a type of visual agnosia supposed by Lissauer (1890) and demonstrated for the first time by Goldstein and Gelb (1918,1919). The greatest novelty of this work is that the patient, being totally blind to visual forms, should not see more than amorphous spots, but thanks

to a special mechanism discovered by these authors in this patient, he was able to behave apparently in a quite normal way with regard to his visual functions.

These authors found, after many persevering and meticulous observations, that the patient needed to move his head to see shapes or recognize them visually. He needed to contour the object in order to realize its configuration, otherwise he only perceived a chaos of shapeless spots. But the most surprising thing is the authors' explanation that visual recognition is fictitious because the patient is only able to recognize the object by a motor process and not by sight, which must remain unaltered. That is to say, if the subject is able to find out the configuration or form of objects is not due to his sight but to the representation of the set of movements he makes with his head by contouring the diffuse forms of the objects. Such movements are so insignificant that they go unnoticed by the most attentive observer, as happened for a long time to these authors and also to the patient who was initially completely unaware of them because he performed them involuntarily.

The mentioned authors consider that their Schneider case shows a kind of motor imagination and a surprising disposition for that kind of perception. If all spontaneous head movement was prevented, the patient in question was totally unable to see or visually recognize any object.

Such characteristics represent the most outstanding feature in visual perception and shape recognition by a person who is blind to visual shapes. In addition, various psychological processes related to the visual disorder in question are discussed, and all kinds of evidences are provided in order to maintain the above explanations.

In the second work of these authors, also very extensive and detailed, published shortly after the first one, they maintain that the loss of visual spatial sense and visual representation entails secondarily a disorder of the same nature in the sense of touch. In this way they aim to explain the disturbances of tactile space in both sides of the body (location of the stimuli, spatial discrimination of two stimuli, posture, etc.) as well as those of tactile recognition, since the patient presented a very special tactile asymbolia or agnosia. They think that tactile functions are basically intact and if they can be so severely affected it is due to the loss of the visual influence that would normally be exerted on touch, which by itself would be unable to organize spatial and gnosic structures. Appealing to this hypothesis of visual influence on touch, they renew a theory that was in vogue long time ago among psychologists and philosophers, in which it is claimed that there is no space other than that provided by sight, and that tactile space is generated secondarily by the influence or support provided by sight.

All these considerations refer to the general thesis; as for the particular disturbances of tactile function, there is again a disorder very similar to that of visual function, with very similar motor help phenomena. The authors find that thanks to certain muscular jerks that the subject makes unconsciously, it is possible to supply the functional deficit and achieve the localization of stimuli or even to perform other more complex functions. In short, a series of mechanisms allow the patient to reach a behavior of rather normal appearance, without which the patient presents important disturbances in all the spatial and gnosic structure. According to the authors, such motor mechanisms only apparently accomplish the spatial tactile function, and the indirect way of achieving this function is

a subterfuge that in no way changes the true state of the patient, who lacks spatial sense in both sight and touch.

Such works, as much cited as discussed, have been the seed of a number of more or less theoretical conceptions. Thus, Schilder (1923/1935), for example, relies on these works on the Schneider case for his conception on *body schema,* and likewise Stein (1928, 1930) for his motor theory of perception.

On the other hand, several contradictions were soon indicated, and there are very important works devoted to refute the whole building of hypotheses of the authors, especially the work of Poppelreuter (1923), although in fact nothing positive has been established. Here, in the reduced space that we have to deal with many issues, we cannot expose in detail the research of Goldstein and Gelb (1918, 1919) and the criticisms it has promoted, and we will expose only some general ideas. Aside from the important criticism by Poppelreuter (1923), the Schneider case has been discussed in various aspects by authors such as Pötzl (1928), Kleist (1922), Stein (1928), etc., as well as by many other scholars who have exposed it in detail.

Certain contradictions are indicated which make the Schneider case difficult to understand and, in general, it is thought that alongside remarkable and interesting observations, there are extremely hypothetical ideas.

The Schneider case was later subjected to further psychological research on general intelligence (Benary 1922), and language peculiarities (Hochheimer, 1932), and after twenty years after his brain injury, Schneider was still the focus of conferences and diverse studies, maintaining the characteristics initially described by Goldstein and Gelb (1918, 1919). It can be stated that no other brain patient has so far given rise to as many works and comments as the Schneider case.

After twenty years, in 1939, the veracity of certain findings in the Schneider case of Goldstein and Gelb was confirmed for the first time in other cases, thanks to the evidence provided by the M and T cases, whose detailed study has provided the material for the present work. It should be noted that, although the great attention to detail of the mentioned authors has revealed very special phenomenological aspects of the Schneider case, they have not taken the decisive and indispensable step for a complete understanding of the disorders. It can be said that they have just arrived to the borderline. As is often the case, a certain number of well observed facts are confirmed and maintained in subsequent investigations by other authors, whereas other facts of decisive nature remain latent and go unnoticed due to the influence of theoretical prejudices always present in any investigation, which make speculation alien to reality and further prevent the discovery of significant facts. But all this is inherent to the scientific development whose progress is the result of the sum of several authors' efforts.

From the viewpoint of clinical and symptomatic manifestations, the Schneider case is the only precedent we can indicate to our current investigations on the M and T cases. This is not because such cases are exceptional; on the contrary, we think they manifest a modality of a very general nature, which, if it has remained hidden is due to an insufficient examination of the patients with brain injuries. However, the Schneider case is more a starting point in our research than a true precedent, since all the work presented here starts precisely from the experimental rebuttal of the theoretical explanations of Goldstein and

Gelb (1918, 1919) about motor recognition and the alleged visual influence on touch. This is due to the finding of the phenomena of dynamic action, which in addition to offering a solution to the problems posed by the Schneider case, they open up new and wide-ranging perspectives in brain research.

1.3 The finding of the dynamic action phenomena

At the beginning of this research, in August 1938, the M case began to be significant, initially for the discovery of the phenomenon of *flat colors*, and especially later for the series of experiments on the curious disorder of inverted vision. But all this did not seem to indicate a possible similarity between the M case and the Schneider case, since neither of these two phenomena had been described in the Schneider patient. It was a year later, at the end of 1939, when trying to rationally explain the series of syndromes found in the M case, an attempt was made to find a certain parallelism between the disorders of both subjects. In fact, although the M case had initially attracted all our attention to certain very remarkable symptoms never mentioned in the Schneider case, they had a good number of disorders in common, some of them extremely rare such as the loss of visual perception of motion and, in a more general sense, both presented, in very special conditions, the disorders of visual and tactile agnosia. The fruitful approach of the Schneider case was the result of a very careful examination of subject M to solve certain difficulties that were making the results of his examination very changeable.

1.3.1 The singular functional disaggregation

As a frequent occurrence, some phenomena encountered at the beginning of an investigation go almost unnoticed as regards their main significance, and only stand out in a preferential place at the end of many detours, without anything essential having occurred in them. It all depends on the right choice between what is essential and what is accessory. This is what happened to us with the issue of functional disaggregation, which is the first phenomenon found of dynamic action, which we will be examined in detail when we study *nervous asynchrony.* Only by combining it with the other phenomena, it is possible to understand the true state of brain activity in the M case.

In the summer of 1938, by studying experimentally the disorder of inverted vision in the M case, we had enough data to establish, as a general rule, the remarkable functional disaggregation caused by the brain excitability disorder. The sensory response obtained depended entirely on the intensity of the stimulus used. Thus, if an object was seen at a short distance, its initially correct perceived position was progressively tilted as the distance to the object increased, until a total inversion occurred. The larger the size of the object, the greater the distance required to achieve the inversion. Likewise, even if the object was located at a short distance where it was seen correctly, the object could be seen inverted or strongly tilted if the exposure time of the object was only about one second. Therefore, it was possible to study with great ease the transition from the normal function

(correct vision) to the pathological one (inverted or diversely tilted vision) just by changing the experimental conditions. This phenomenon was evidently based on especial alterations in brain excitability, with a different response to stimuli according to their intensity. The stimulation was weak in far vision due to low luminous intensity and small visual angle; the opposite for near vision. In the case of inversion or strong tilt in near vision but with a brief exposure of the test object, another stimulation factor took part besides the intensity: the exposure time of the stimulus. This fact was already an indication that the reaction time was extremely increased in this patient. For all this, the state of brain excitability gave the real explanation for all these disorders. However, the full importance of the excitability process did not become evident until a year later when the necessary maturity was reached by observing other dynamic action phenomena.

The functional disaggregation is the phenomenon that has allowed the important study of the physiological structure of the various sensory organizations. This disaggregation is based on an asynchrony of the nervous elements as a consequence of the special dynamic alteration in brain excitability. The characteristics mentioned above in inverted vision were common to all types of functions; thus, for example, in the case of tactile sensitivity, a weak stimulus was only perceived without producing any sensation of localization, whereas a more intense one was perceived in a localized manner. The results were dependent on the intensity and duration of the stimulus applied; hence, a tactile stimulus of a certain intensity but short duration was felt only as a simple contact, whereas a more prolonged excitation of the same stimulus caused localization.

These partial effects, out of phase with each other, form a single function as an all-or-nothing effect in a normal individual. In these partial effects it is clearly shown that the simplest elements of the function are the most easily excitable (have the highest excitability), so that more complex organizations are delayed because they need more excitation. These are only activated by stimulation of a certain intensity and of longer duration than for the most elementary activities. Thus, if the stimulus is weak, even if it lasts a long time, the level of excitation of the more complex elements (little excitable) is not reached and the only response obtained is that of the simpler elements of the disaggregated function.

No indication of all this is found in the detailed descriptions of the Schneider case by Goldstein and Gelb. They only emphasize that the behavior in the various visual and tactile activities is completely different depending on whether or not motor recognition intervenes in visual activities and certain muscular jerks in tactile ones. The meaning of this difference will be seen later on.

This singular disaggregation was progressively observed in all kinds of altered functions, greatly facilitating how to address the research within the multiform set of symptoms that changed at every step due to circumstances that went easily unnoticed. It was precisely by trying to obtain the highest possible accuracy in quantitative determinations, that we reached the true understanding of the special state of brain excitability in subject M, in whom the functional asynchrony indicated above was only a particular aspect.

1.3.2 Facilitation (reinforcement[4]) or summation phenomenon

The great instability of the symptomatology in neurological patients is a well-known fact, but in our case, the lack of constancy of the phenomena when we tried to determine quantitatively with precision the effect of the stimuli was so sudden and of such a wide range that it could not be considered due to fatigue or changes in the recovery from the pathologic syndrome, as could be admitted in other cases. Perhaps special mechanisms were involved, and for a long time they were searched for. At the same time, since certain analogies had been found between the general symptomatology of subject M and that of subject Schneider, even the special muscular jerks that appeared spontaneously in some examinations of the tactile function, we tried first to determine whether the motor recognition phenomena for sight existed in subject M, and then to see whether such phenomena were the cause of such remarkable variations in the behavior of the subject. This muscular jerks had been clearly observed in the second individual studied here, subject T, who was examined before subject M and before knowing in detail the studies of Goldstein and Gelb (1918, 1919).

Whereas for the sense of touch (tactile localization for example, etc.), muscular jerks were evident on occasions and in an irregular manner, for the sense of sight they could not be verified in very repeated and detailed observations, and nothing seemed to confirm the existence of a motor recognition and a full analogy with the Schneider case. All these new analyses were undertaken by the end of 1939 in order to clarify and complete a number of details. It happened by chance that the patient M, lying down and in complete inactivity, he made so many errors when objects were presented to him for their visual recognition that it was possible to conclude that he barely had vision, which appeared to improve noticeably as soon as he sat up and returned to his ordinary attitude. Such an unexpected change made attention focus again on the determination of motor phenomena or something similar. When trying earlier to examine the existence of motor recognition, it was not possible to find an active movement of the subject's head, even though it was known, from the indications of Goldstein and Gelb (1918, 1919), that such recognition movements went easily unnoticed due to their small amplitude. When the patient was told not to move his head at all, no significant influence on his functions was observed, or at any case, its effect was not very important, but a new test with the head kept fixed did give decisive results. With the patient's head firmly fixed, his visual perception of objects worked as badly as in the previous position, lying down and inactive. However, when the head was released, the usual behavior appeared again, that is, very slow but quite viable perception. Our patient became very surprised but did not seem to give much importance to such results, and only when he failed the visual tests with his head fixed, he insisted on having his head released and said that it was because the blood did not reach his head and he was not able to see well. If such movements of his head were there, they had to be extremely slight, rather small muscular tensions producing insignificant displacement.

[4] Reinforcement is `refuerzo´ in Spanish, term used in the original work, and has been translated in most cases as `facilitation´.

For a moment it was thought that the motor recognition of Goldstein and Gelb (918, 1919) had been found, thus confirming for the first time that phenomenon in another subject; but in a multitude of further tests performed to study the issue accurately, very remarkable divergences began to appear, since our patient behaved as follows: he was able to recognize objects visually by just moving his head in any direction without corresponding in any way to the shape of the object presented. Therefore, such behavior should not correspond to the contouring referred to by the mentioned authors. In addition, when the subject's head was immobilized and he was lying down in complete muscular relaxation and immobility, if objects were presented to him, his visual perception was initially unclear, but after ten to fifteen seconds the perception had improved considerably. In his ordinary state of total freedom and sitting up, this time was reduced to about half, although he was still very slow compared to a normal subject.

All these results led to the opinion that there was something that influenced the brain activity of visual perception, modifying the nervous excitability. Other tests showed in the same way that the motor recognition according to Goldstein and Gelb (1918, 1919) was here inadmissible; in fact, keeping the subject's head immobile and asking him to clench his fist strongly, his visual perception worked as well as with the free movements of the head. No movement of any kind was required; tension or a strong muscular contraction of any part of the body was enough.

In the Schneider case, visual perception was rather slow, as in our patient in his ordinary state, and the authors thought that this delay was due to the time spent on performing the contouring movements, without which the subject was not able to see but shapeless spots. Therefore, in tachistoscopic exposure of figures, he was not able to see defined shapes because of the brevity of the exposure. But having ourselves achieved the above mentioned results on changes in excitability due to a possible extra-visual facilitation, we tried to determine the effect of an intensification of the facilitation on latency time. The result was that a strong contraction of all the voluntary body musculature reduced so much the sensory time that it seemed to completely normalize the visual function. The subject thought he was cured, he stated that he felt as before suffering the brain wound, although later, when he experienced that on finishing the intense muscular effort he returned to his usual precarious state, his enthusiasm diminished, but he kept on thinking that healing would be a matter of a few days. As expected, the patient was impressed by all these tests. In fact, when in previous experiments the effect of keeping his head still was found, and he had to finally discard the unfounded explanation of the circulatory difficulty, he fell into a state of prostration because he perceived the severity of his disorders.

By studying in this way the possible existence of the motor recognition of Goldstein and Gelb (1918,1919), we ended up discovering that sensory activity is considerably influenced, in its own excitability, by nervous actions alien to itself, as is the case of muscular tension. This type of facilitation, which represents a nervous summation phenomenon, can show gradations. Thus, it happens that already the upright attitude entails a natural muscular tension causing a certain summative action to which can be added the action due to slight tensions of the neck muscles (performed unconsciously during visual perception), reinforcing the previous summative effect. When the subject is

in complete inactivity (lying down and in a state of muscular relaxation), all facilitating action ceases and the true basal state of brain excitability is achieved. Finally, by means of maximum contraction of the whole musculature, the effect of summation is so considerable that the visual behavior of the subject becomes quite normal. Although in the latter case, we shall see that by examining the subject thoroughly, he still shows a certain deficit, though very small in comparison with the two previous situations: complete inactivity and moderate muscular effort in ordinary life.

In summary, from what is exposed in this section, it can be inferred that for the proper understanding of the pathological manifestations of our subject, it is necessary to take into account, besides the intensity and duration of the applied stimulus, the summation effect by muscular action, which increases brain excitability. With this last finding of facilitation by muscular effort, the problem of instability and inconstancy of the experimental results, which had hitherto made all attempts at a quantitative determination of the disorders so arduous, was solved. It was then possible not only to obtain precise and stable results, but also to obtain a large number of them, since a new procedure was available to easily experiment on nervous excitation relationships.

However, the investigation on summation did not stop here since, given that the effect of muscular contraction seemed to be so non-specific, there was reason to believe that perhaps any other type of nervous action would produce the same result. Indeed, the result of the tests performed was entirely in favor of this hypothesis. Both acoustic and tactile stimulations were able to improve visual function, however the effect obtained was much smaller than that produced by an intense contraction of the whole musculature, as is easily understandable. Certain phenomena were to be chosen to clearly show the summation effect. To this end, the inverted vision disorder is very appropriate. Thus, an object placed at a convenient distance is seen inverted (upside down) if the subject is free from any facilitating action, but by means of an intense contraction of all his musculature, the image of the object rotates quickly becoming correctly oriented and at the same time the sharpness of its shape improves considerably. Under these same conditions of object presentation and being free of any facilitation, a tactile excitation of a certain importance, such as brushing or rubbing his back, without causing any pain to avoid defensive contractures, has the effect of re-inverting the image of the object in a certain number of degrees, i.e. to change from the inverted position to a tilted or even horizontal one. This change lasts only as long as the tactile stimulation is applied. Similarly, a strong acoustic excitation by means of an intense whistling at the subject's ear causes the inverted image to oscillate by causing a small re-inversion.

This means that any type of nervous action, both muscular contraction (in a constant tension or performing movements) and any sensory excitation, modifies the state of brain activity as a whole when it reaches the cortical centers, reinforcing by a summation action the excitability of the brain. There is no specificity in the effects of the various types of facilitation mechanisms. It should be note that there are also significant types of summation phenomena in certain situations, and their knowledge is of great importance for the full understanding of the behavior of the brain-injured people we shall study. Thus, especially in vision but also in hearing, the result of the tests is very different depending on whether the person uses only one eye or one ear keeping the other one shut, or whether

he uses both eyes or both ears. In the latter case, a significant improvement of the corresponding function occurs due to the resulting summation effect. This effect will be called *bi effect* and is of great importance in vision and in any other sensory system as well. For example, in the sense of touch, a weak contact can be made perceptible by means of a simultaneous stimulation in another region of the skin. In this way it is also possible to reduce, or even suppress, the pathological time lag between the sensation of mere contact and that of localization.

Several types of facilitation can act simultaneously and thus produce a very important summation effect; for instance, binocular vision and maximum muscular contraction. Therefore, within nervous reinforcement, different types of facilitation must be considered; otherwise, important gaps would remain in the accuracy of the experimentation and the theory of nervous summation.

So far, we have referred to the visual system. As regards tactile functions, the results were entirely similar with respect to the conditions of functional asynchrony depending on the intensity and duration of the stimulus applied (as mentioned above), as well as with respect to the facilitation effect. Therefore, the muscular jerks referred to by Goldstein and Gelb (1918, 1919) were not indispensable to localize stimuli and reach other more complicated functions. Complex functions like localization of stimuli were achieved both by increasing the intensity of the stimulus without any facilitation, and by weak stimulus and facilitation by strong muscular contraction.

At the same time, all these relationships and excitability phenomena showed that the sensory disturbance affecting vision and touch was global, from the simplest elementary sensation to the most complex perceptual and gnosic functions. By contrast, in the Schneider case, disturbances had been indicated only in the latter two. The rebuttal of the explanations of motor recognition for vision, and of muscular jerks for touch, as well as the study of excitability relationships, and the fact that the sensory disturbance was extended to all kinds of functions of these two sensory systems, led to questioning the hypothesis of the supposed visual influence on touch. Thus, we proceeded to complete the study of the dynamic action.

1.3.3 Cerebral repercussion

This is about the finding of the disturbance of all brain functions and on both sides of the body, produced by a single not very extensive lesion in the left parieto-occipital region, against all that could be expected from the theory of brain localization.

Excitability relations and central summation phenomena, although generally ignored in brain pathology, are on the contrary well-known in the general physiology of the nervous system. Thus, it is enough here to extend them for the first time to the domain of human brain functioning. However, the effect of nervous repercussion due to a small lesion in an *association* area, which is ultimately what the general brain disturbance consists of, is perhaps of much greater theoretical interest. This is because the nervous repercussion phenomena or remote effects of lesions, although they had been revealed some time ago, their research had not involved the human brain; but now in our case they have given rise to issues of fundamental importance, radically modifying current trends and, above all, compromising the theory of brain localization.

The difficulty in understanding a tactile disorder equally in both sides of the body caused by an injury in an area that has nothing to do with the brain region ascribed to touch, may have led in the Schneider case to the hypothesis of the visual influence mentioned above. But now, having shown that the disorder in our M case was much deeper since not only spatial functions (localization, two-point discrimination, motion, position, posture, etc.) were involved, but also the simplest sensitive manifestations (pressure, pain, temperature, etc.), the thesis of a secondary disorder of tactile space did not answer the whole question. In order to explain the tactile disturbance, the aforementioned visual influence should then have been extended to more elementary functions; but, to think that the simple sensation of contact or pressure needs visual influence is meaningless. Therefore, the question that tactile function was affected in both halves of the body following a brain injury on the left side and outside the tactile area was still a problem. Nevertheless, the same problem arose with regard to visual function, since the brain damage did not involve the calcarine cortex, but the patient suffered from a very large concentric reduction of the visual field, that is, the two halves of the field were involved. This patient as well as a large number of our wounded subjects with concentric visual field reduction of different severity, presented brain lesions outside the calcarine cortex or visual field projection area, and so did the Schneider case as well. This disorder, without any possible explanation within the theory of brain localization, in spite of being sufficiently known, has not led to the necessary research. The difficulties to explain this type of disorder in both visual and tactile systems were the same, and it was not possible to progress by trying to understand a disorder by means of another disorder, since both disorders were equally in need of further research.

On the other hand, the finding of facilitation by muscular effort and the evidence that it exerted a dynamic action “at a distance” on brain excitability, opened the way to consider these disorders from a dynamic point of view. If facilitation acts “at a distance”, the lesion could also do so; in this way nervous activity should offer a much more unitary character than what might be assumed within the generally accepted anatomical and localization concepts of brain pathology.

If such a hypothesis of action “at a distance” of the lesion (dynamic repercussion) was correct, it seemed conceivable that other hitherto unexplored sensory systems might have been equally impaired, and due to the special characteristics of the disorder they would have gone unnoticed. Based on this, we decided to determine the patient's hearing status in all its aspects, and found that he showed a kind of alteration completely identical in intensity and characteristics to that shown in vision and touch. The summation effect due to the upright attitude and other weak muscular efforts, as well as the habitual slight examination had made all these hearing disorders unnoticeable. The hearing impairment was in all respects in line with the characteristics already indicated for other sensory systems with regard to intensity-duration relationships, sensory function delay (functional disaggregation), summation effect, etc. The disorder also affected both ears, which showed severe deafness when the subject was inactive and the acoustic stimulation was weak and, above all, of short duration. There was also inability to perceive sound rhythms from a metronome, to distinguish musical tones, etc.

Therefore, our view that there was a general alteration of the brain by a dynamic repercussion effect became more definite by empirical confirmation of the theoretical deductions. The three most important sensory systems, vision, touch and hearing, were deeply altered on both sides of the body, that is, in their full extent, and also in all their functional aspects from the simplest elementary sensation to the more complex gnosic functions. The disorder showed the exceptional feature, within the usual concepts, of being the effect of an action "at a distance" of a brain injury located in the association ("central") area. This establishes a new type of brain disturbance that we shall study later on under the name of *central syndrome* of the cerebral cortex, and which also constitutes a starting point for the fundamentals of brain dynamics.

Other sensory disturbances, although much less important, appeared in lower functions such as taste and smell, to which disturbances in orientation and body position corresponding to a function of the labyrinth had to be added. All these fundamental findings about the dynamic action were carried out, as mentioned above, at the end of 1939 in the M case, and confirmed much later in the second patient, the T case, and were extended and refined in a series of details over successive years. The general disturbance of the sensory systems has made it possible to study in a single individual a multitude of phenomena, such as the complete series of sensory activities, whose physiological nature has been uncovered by a sensory lag of functions (functional disaggregation). Furthermore, by means of the summation effect, an important experimental resource has been provided to expand and control this type of analysis.

1.4 Rebuttal and experimental extension of the two research studies of Goldstein and Gelb on agnosia

The two previous sections have dealt, firstly, with the origins of these studies in the research on the Schneider case and, secondly, with the finding of new data on dynamic action that have profoundly changed the initial focus of the research. Now is the time to specify conclusions and considerations on these matters.

Long time after the finding of the dynamic action in the M case, the other injured patient was sought, subject T who, because of the symptoms he showed in 1938, had certain analogies with the M case. The purpose was to submit him to the same analysis to which the M case had been submitted. The brain lesion was in the same region as in the M case, but it was less deep. According to these circumstances, the behavior of subject T was found to be entirely analogous to that of subject M, except that all the excitability disorders were much less pronounced. Thus, less slowness, less asynchrony (less sensory lag) and less summation effect. As for the general disorder of sensory systems (cerebral repercussion), the distribution was entirely identical to that of case M.

In short, we are dealing with two cases with a dynamic disturbance of the brain, different in their intensity but identical in their essential characteristics. Concerning the Schneider case, in view of its most pronounced manifestations, it is feasible to ascribe this case to the type of dynamic disturbance exposed here, and in view of certain data, to consider it as intermediate between the T and M cases as regards the intensity of the brain

disorder. In this way, it is possible to know approximately the true state of alteration of sensory functions in the Schneider case on the basis of a small number of indications, and it is also possible to predict the existence of a number of disorders that have been completely unnoticed by the authors.

In their final comments, Goldstein and Gelb (1918, 1919) insist on the inalterability of the symptoms in their case Schneider, but after all that has been said about the changes according to the conditions of excitation and facilitation, their statement can be considered to be the result of an incomplete observation. On the other hand, they found that certain complex functions of a spatial nature (form, motion, localization, etc.), as well as the higher functions of visual and tactile recognition, failed or were missing in the face of stimuli of ordinary intensity. But a more attentive examination of the conditions and relationships of excitability is enough to know that the disorder originates, above all, from a general alteration of the excitability of the brain, encompassing all kinds of sensory functions, both simple and complex.

For the same reasons that the authors think that the Schneider case is a man who lacks spatial perception for both sight and touch, they could have argued that he was blind or anaesthetic (without sensation), since stimuli of a certain intensity but of very short duration (easily detected by a normal individual) would have gone completely unnoticed by him, as in the M and T cases. However, increasing the intensity or the excitation time is enough for even complex functions to appear.

Also, the special modification of nervous excitability leads to the generation of iterative excitation, i.e. a weak stimulus sufficiently repeated at short intervals can produce not only elementary sensory responses (which do not appear under a single excitation), but also sensory responses to complex functions. This iteration capability is very notorious in the M case and much less in the T case, in line with the intensity of the other nervous excitability disorders. Hence, the intermediate degree of iteration that the Schneider case should present can be found with a certain approximation.

The research on the Schneider case remains exclusively at a psychological level, while the mere description of the symptoms is at a clinical level. As interesting as the results are, the decisive step towards a physiological rationale of the disorders, the only way to explain fully and correctly the important questions raised by the Schneider case, has not been taken.

The need for a physiological basis becomes even more peremptory with regard to phenomena such as the supposed motor recognition and the alleged secondary tactile alteration by abolition of the visual influence. These two explanations are completely psychological: in one, it all comes down to a singular facility for motor representation; in the other, it is admitted that the only true space is that provided by sight and that spatial structures of touch are an effect provided by sight. The authors went so far as to pose the problems, but the psychological orientation has perhaps blocked the way to their proper solution. A physiological orientation would have led to logical arguments capable of explaining all kinds of phenomena in a unitary and simple way. For all these reasons, we can say that our studies rebut the research of Goldstein and Gelb (1918, 1919) on agnosia in the Schneider case, and simultaneously extend it by continuing it under a more

experimental aspect, even though the result of this deepening denies the explanation of these authors.

In particular, with regard to the motor aids that all these subjects use unconsciously, the physiological point of view becomes irreplaceable; the only explanation is that of the nervous summation effect. Moreover, it is necessary to take into account nervous summation in order to know the true state of the brain disorder, since the weak summation generated in the ordinary state of these injured patients is enough to improve their functions and cover up the severity of their impairments. We know that depending on the intensity of the muscular effort, the intensity of the nervous excitability disorder can be very different, and the subject can then present different types of states. This makes it possible that different cases regarding the intensity of their nervous disorder can be equated to some extent. As an example, the M case under maximum facilitation by strong muscular contraction can be very similar to the T case free of all facilitation, and M with somewhat less muscular effort becomes similar to the Schneider case. The psychological conceptions of Goldstein and Gelb (1918, 1919) that have induced them to leave aside such important questions about nervous excitability, show how far brain pathology research is from its true physiological basis.

The other fundamental result of the second work on the Schneider case is quite paradoxical. It is remarkable that Goldstein (1910), particularly known for his justified attacks on the classical theory of brain localization (based on the anatomical-clinical point of view), did not take advantage of the exceptional opportunity he had with the Schneider case to provide an observational fact as simple as decisive. That fact was much more eloquent than all kinds of theoretical arguments, always very diffuse in this matter. By resorting to the theory of inter-sensory influence, Goldstein (1910) moves within the associationist psychology, one of the mainstays of the doctrine of brain localization. The development of this issue has led him to establish primary alterations for sight and secondary alterations for touch. However, in both our cases and in the Schneider case, vision, touch and hearing are basically altered at their origin, in all their functions, with the simplest functions being altered on both sides of the body, a fact that is totally incomprehensible according to the theory of brain localization. The solution to the problem of a general disturbance of sensory systems affecting the whole body, due to a left parieto-occipital brain lesion (which does not involve visual, tactile and auditory projection areas at all) is to radically abandon the usual concept of a rigid and static anatomical localization, since brain lesions affecting the truly central areas (the so-called association areas) disturb equally, by dynamic action, the entire brain system that is in functional relationship with those centers.

The cerebral cortex can be considered as a dynamic structure in which the functional level depends on the amount of nervous mass, mass that would act as an unspecific activator of brain excitability. From this point of view, it is easy to understand that the exclusion of nervous mass due to destructive injuries should reduce functional capacity, i.e. impair the excitability level of activities subordinated to the dynamic action of the nervous centers. The effect of facilitation is then an added activity to the remaining brain activity, thus partially replacing the excluded brain mass by the injury.

In short, within the physiologic criterion, the brain disorder of the three cases, Schneider, M and T, can be explained by a unitary dynamic disorder. By virtue of this functional dynamic unit, the disorder of nervous excitability spreads to the entire brain, with regard to sensory functions. This disorder is characterized not only by a necessary increase in the stimulus intensity and duration, but also by *permeability* to iteration and to facilitation (summation effect), and additionally by a functional asynchrony (leading to a functional disaggregation) arising from the excitability deficit, as we shall study further on.

1.5 Special features in the examination of these wounded patients

At first glance, although it might seem paradoxical, these cases present a scarce symptomatology. The M case spontaneously complains only of a great loss of vision, which otherwise does not bother him greatly in his daily life. Apart from tests and clinical determinations, nothing in his ordinary behavior would suggest such serious disorders as those shown by his different sensory systems. Even more scarce are the manifestations shown at first glance by the second subject (T), who in principle has not shown any particular brain disorder outside the usual general discomfort typical of brain-injured convalescents. This means that the finding of the numerous disorders that these subjects have is almost exclusively due to very patient examinations which have made clear, over the years, the condition of these subjects, without them explicitly cooperated to the knowledge of their disorders.

Thus, disorders such as flat color vision or the inverted vision phenomenon have been unexpected results in examinations aimed at other purposes, and when they were found, the patient initially did not seem to give great importance to any of them. With respect to the important phenomenon of inverted or strongly tilted vision, subject M merely commented: “these are things that sometimes appear in my sight”. He also explained that sometimes, before we found his disorder, he had been surprised by his abnormalities, for instance, seeing some men working upside down on a scaffold. In general, the alterations go completely or almost unnoticed by the injured subjects themselves, and even when they are discovered, these subjects do not seem to be concerned and rather consider them as something transitory that does not affect their daily life.

Throughout the study of these injured subjects, the great difference in behavior during the experimental tests and outside them has been evident. Such divergence has been completely clarified by the special phenomena of dynamic action in these individuals. In fact, when in their daily lives they pay attention only to the most intense stimuli, the disorder of sensory lag that produces a disaggregation of functions is practically excluded; for example, visual stimuli of a certain intensity produce vision that is correctly oriented or very little tilted. But above all, the facilitation action is decisive, since it considerably reduces the brain disorder, as has been said. If out of the tests, binocular vision is used as usual, this is already a very important facilitation due to the double action of the eyes. If a certain muscular activity is added when walking, etc., as

well as a good illumination of natural light, a series of actions are obtained that, when added, produce a considerable reducing effect on the disorder. In the first tests after the fundamental findings, only a few kinds of behavior had been identified; later it became clear that a series of simultaneous combinations of different types of facilitation are possible, allowing for a fairly normal behavior in everyday life.

As for the attitude that the subjects adopt in the face of such singular and numerous symptoms, we should not think that in the long run they are completely indifferent. On the contrary, the finding of facilitation by muscular effort has produced a very deep impression on them, both because of the importance of its effect and because of the inexplicable and strange for them. On the other hand, they have gradually become accustomed to all kinds of tests, although they have certainly found it unpleasant to be affected by so many different disorders. This tolerance or willingness to cope with their situation was very different depending on the type of disturbance. They have adapted fairly well to thorough tests for quantitative determinations of sensory disorders and so on, but they become extremely irritated when a more intellectual activity disorder is demonstrated. In this sense, failures in visual recognition of simple figures or drawings, or certain very subtle defects in understanding language, are very badly received because they realize the great extent of their brain disorder.

Another notable aspect of the research on these injured subjects is the type of interpretation and appreciation they make about their pathological manifestations. In general, brain patients tend to give all kinds of explanations about the symptoms or phenomena observed in them, and since they are usually not very well founded, great errors would be made if one trusted both their explanations and the subjective phenomenology of the patient. It is necessary to submit the patient to an objective control by means of very varied tests, by which we can perfectly realize his functional state by the logical correspondence between different tests. In addition, it must be taken into account that within the instability or lability of the sensory anomalies, a certain equivalence between very different phenomena can be produced. Thus, the patient M states that a piece of writing is in the same position whether it is presented in the correct position or upside down, due to the loss of orthogonal spatial orientation[5], which is to some extent independent of the inverted vision disorder. In this case, to be more precise, the patient should have said that he does not know about positions in reading and does not distinguish the correct position from the inverted position of writing. In this behavior of the patients also intervene sensory organization laws generating the equalization mentioned above. It is likely that these mechanisms are similar to those that cause Babinski anosognosia syndrome (Babinski 1914, 1918) in many brain lesions, according to which the patient tends to involuntarily exclude the existence of the defect. For example, the hemiplegic patient who ignores his paralysis, the receptive aphasic with logorrhea and jargon aphasia who is unaware of the lack of intelligibility of his language, or the brain-injured blind person who obstinately insists on his visual capacity, which is the so-called Anton syndrome (Anton 1898, 1889) of which we have study several very instructive cases in brain-injured patients.

[5] Correct orientation on the plane orthogonal to the line of sight.

What can really hinder the examination, and made it indeed difficult at the beginning of this study for not knowing sufficiently the state of the injured patients, is the lack of vivacity, that is, the great slowness of all the brain processes due to the excitability deficit that gives rise to a long latency for stimuli as well as to an abnormal persistence of them, the latter originating the fusion between successive stimuli or tests very close in time. This excitability deficit, which entails functional slowness, also causes mental clumsiness or viscosity, which obliges to carry out a paused and meticulous examination if useful results are to be obtained. It is very important to keep in mind that patients respond to tests in an approximate way, especially the more pronounced the brain disorder, so quantitative determinations may be of little value unless patients are properly urged and attention is paid to all kinds of details.

Also, given the complexity and extensive nature of the sensory disturbance as well as the sensory state of the patients, tests that do not provide useful information or lose the meaning they would have in ordinary cases, should be avoided. For example, when investigating color vision, it must be kept in mind that it is a very complex disorder extended to very diverse sectors of the color function, namely color blindness or color weakness accompanied by chromatopsia, flat color vision that by irradiation [6] can notably alter the perception of colors located next to others, and finally color agnosia (difficulty to understand color names). Only by considering the disorder as a whole is it possible to establish the examination methods to be followed and how much value should be given to the results.

[6] Diffuse spatial localization of a color.

2 The new central syndrome of the brain

2.1 The M case and the T case

Both the M and T cases were examined for the first time in 1938 and the M case has been, due to various circumstances, the most studied and with which this research work has been initiated. As already indicated, both show the same general characteristics, differing only in the quantitative degree of their disturbances. This allows a more extensive investigation since it can be developed by considering different levels of excitability. The general characteristics of these cases will allow us to describe the new *central syndrome* of the cerebral cortex, which means a considerable transformation in the theories of the brain.

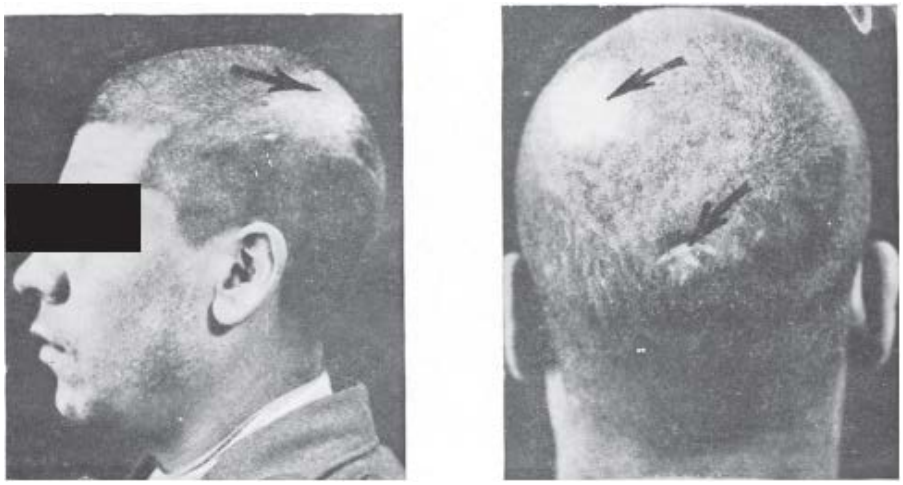
2.1.1 Case M

Subject M suffered a brain wound in May 1938, when he was 25 years old; he has two cranial scars in the left parieto-occipital convexity that corresponds to the entrance and exit orifices of a bullet. A very small entrance hole located 1 cm from the midline and above the external occipital protuberance. Another much bigger one corresponds to the exit, star-shaped and located three fingers from the middle line and two behind the mastoid line (Figs. 1 and 2). Despite the importance of these wounds, the subject healed easily and quickly without the need for surgery or other special care. He only had a little suppuration at the edges of the exit wound, which delayed full healing somewhat.

From the location of the entrance and exit cranial wounds, it can be stated that the brain area destroyed by the bullet corresponds approximately to the angular gyrus, close to the posterior end of the inter-parietal sulcus. Therefore, it corresponds to the parieto-occipital, medial and somewhat upward region, i.e., the area in the middle of area 19 of the Broadmann nomenclature and perhaps the anterior portion of area 18 and the most posterior portion of area 39, both areas around area 19.

The lesion is placed on a fully *associative* brain region whose destruction, given the short intracranial trajectory of the bullet, somewhat tangential to the brain, must not have been very deep although the tear of the cerebral cortex convolutions in that region must be admitted.

He was observed by us a few weeks after being wounded, at which time he presented the usual residual phenomena in brain injured people (severe headaches, slight meningism, etc.). It seems that during the first days after the injury, he completely lost consciousness; later when he regained it, he presented a great loss of vision, especially in the left eye with which he could hardly see.



Figures 1 and 2. M Case. The arrows indicate the two cranial entry and exit scars of the bullet that crossed the left parieto-occipital convexity in its middle and upper portion. The direction of the bullet is from behind forward and from below upward.

Recovery was slow and after some months, he presented an intense concentric reduction of the visual field in both eyes: on the perimeter, with a white test of 0.5 cm in diameter, the visual field did not reach more than 4 to 6 degrees for the right eye and one or two degrees less for the left eye. In addition, he had a very slight paresis on the entire right side of his body that disappeared in a short time. Except for the great loss of vision, he did not complain about anything nor did he present any other significant neurological disorders in the ordinary examinations; his general state was good and the only thing that called our attention was his great need for sleep and his indifference.

He was more carefully examined during the summer months, and several agnosic and apraxic disturbances were found, as well as a remarkable slowness for all sensory and intellectual functions. The most notable findings in the visual system, apart from the marked concentric reduction and a very complex and diffuse agnosia, were: *triplopia* (triple image perception of a single object in monocular vision), a phenomenon that we have also studied in other wounded and which we will address in a special publication; *flat color* vision (a chromatic alteration that makes colors appear swollen and as if they had been detached from their objects); a special chromatic blindness in which, in addition to mixing certain colors, everything was seen with a green tinge, including white (*chromatopsia*), and finally the discovery of inverted vision, intensively investigated during the months of August and September 1938. This anomaly was related to very complex disorders of visual spatial orientation, loss of the *orthogonal* function, etc. As for the sense of touch, apraxic alterations and a certain tactile agnosia were found, as well as an altered perception of posture and movement, etc., affecting the entire body.

He was examined again a year later, and at the end of 1939, the above-mentioned phenomena of dynamic action were found, which revealed a much deeper and more extensive cerebral alteration than that first presumed in 1938. Since then, the research on this patient has become more systematic and experimental, he being subjected to periodic

studies as the research work progressed. With regard to the evolution of the disorders, it can be said that only during the first year after being injured, there has been a certain recovery progressively slower; later, the degree of the disturbances has become more stable and these can now be considered invariable in their evolution.

2.1.2 Case T

The second patient, the T case, was injured in January 1938 at the age of 20, in the left parieto-occipital region, upper third. He was operated the same day he was injured because of the sinking of the cranial vault in that region. During the first month, it seems that he suffered from paresis of the entire right side of the body accompanied by loss or decreased sensitivity on the same side. Two months later, when he began to be observed by us, his general state was very good and he did not present any significant neurological disorder, the paresis and sensitivity disorder having disappeared. Because of the copious suppuration of the head wound, he had to be operated two or three times to remove the infected bone edges until the wound was completely clean. In the surgical interventions, the dura mater was always intact and in good physiological condition. He never presented any complications due to the surgical interventions and his good overall condition allowed him to get up and lead an almost normal life. He never had cerebral vomiting nor meningism, only some headaches and dizziness of the type of small absences. Five months after the injury, the suppuration of the bony edges ceased completely, with the dura always having a good pulsation, healing the wound definitively.

As for the state of the scar, there is now a large hollow of about 3 cm in diameter due to the loss of cranial vault; inside, the scar tissue is thickened over the dura mater, which shows no pulsation because of the thickness and hardness of the scar. The depression is located two fingers to the left of the midline. The anterior prolongations of the scar reach almost the bi-mastoid line, and behind, it is 4 fingers above the cranial external occipital protuberance (Fig. 3).

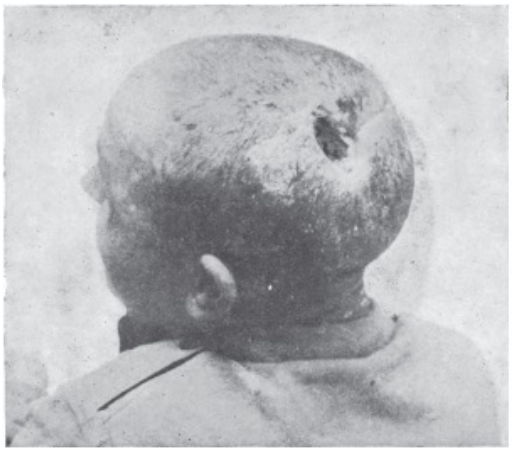


Figure 3. T Case. Aspect of the head injury after healing. Left upper parieto-occipital bony depression that does not directly affect the brain matter.

This is a shrapnel wound that has fractured and destroyed a segment of the cranial vault in the left parieto-occipital area without tearing the dura mater nor causing anatomical subdural lesions, according to the numerous surgical operations in which the dura mater was always found intact and with a good pulsation. For all this, the brain disorders that the patient has presented, and the current residual effects, can be considered to be caused by the brain contusion produced by the bony sinking. This makes the brain disorder in case T much less than in case M; subject T has suffered a contusion whereas subject M has suffered a significant tear in the cortex due to the passage of the bullet. It must also be noted that the recovery from the brain disturbance has been much more important in the T case than in the M case, which is well explained by the respective type of brain injury.

The location of the brain lesion in the T case is very similar to that of the previous case and can be considered practically at the same place. It can be admitted that the brain lesion is located in the center of the intracranial trajectory of the bullet in the M case.

During the examination of patient T in the first months of observation, it was remarkable that such an important wound, even if it was only a contusion, caused so scarce neurological symptoms, even though it could be said that the wound was in an area of the so-called *silent* areas of the brain. He presented besides a concentric reduction of the visual field of moderate intensity in both eyes, a slight hypoesthesia in the right half of the body and a certain insecurity for the other tactile functions (localization, deep sensitivity, recognition, etc.). It is noteworthy in this subject that when he tried to locate tactile stimuli he would slightly shake his body and limbs by means of almost imperceptible muscular contractions, without which he would make many errors.

A few months later, during the summer of 1938, he was subjected to a detailed study for a long time because of the important phenomena found in his visual field. In addition to the concentric reduction and alterations of the type of the so-called *hemianopic weakness of attention* according to Poppelreuter (1917), he presented monocular *polyopia* phenomena in such a pronounced way that it served to unravel much of the mechanism of this phenomenon of multiple vision in a single eye, which we had already observed in many other brain-injured patients. Added to this, there were still very diffuse alterations of visual agnosia, optic ataxia, etc.

Having been discharged when the reversed vision was fortuitously found in case M, and presenting both cases some clinical similarity, subject T was sought and re-examined for a few days. It was not found to present the phenomenon of inverted vision, but rather an important tilt of objects when looking at them at a certain distance. He also presented the curious disorder of the loss of what we call orthogonal function, which allowed him to read just as easily whether the writing was in a normal position or upside down without noticing the change in position. Later we learned that before he was examined by us, after he had been wounded, he was often seen reading the newspaper with the newspaper upside down, and he was very upset when the anomaly was pointed out to him because it went completely unnoticed by him. Also, despite the absence of the phenomenon of fully inverted vision, although with a tendency to it - manifested by the tilt of objects -, we found that after the epileptic seizures he suffered as a result of the cortical contusion, he was so confused by the visual orientation of objects that he preferred to remain in bed

during this state which lasted no longer than 24 hours. It was also observed that his tactile and visual disturbances were more pronounced than initially observed; for example, he had a significant postural disorder due to a deficit in the deep sensitivity and a visual agnosia manifested by slow visual recognition and by the very special efforts he made to see and perceive objects, which could well be considered as the singular muscular jerks mentioned above in order to localize tactile stimuli. This last symptom was observed several months before seeing the M case for the first time and made us turn our attention to the Schneider case of Goldstein and Gelb (1918, 1919).

Due to the finding of the dynamic action in the M case at the end of 1939, and considering the similarity between the M and T cases, subject T was required to verify if he also presented the dynamic phenomena. But due to external circumstances, he could not be found until the year 1942 in which the phenomena of dynamic action were confirmed in the T case, in a similar way to the M case. These verifications allowed us to determine more precisely the extent and depth of the brain disorder. Thus, by studying in detail the excitability state, it was observed that tactile sensitivity was altered on both sides of the body, although slightly more on the right side. For the other sensory systems, the same alterations as those already know in the M case were found.

2.1.3 Comparison between cases M, T and Schneider

From the finding of the dynamic action in both cases M and T we have been able to confirm more and more the similarity between them during the numerous periodical investigations they have been subjected to from then until now. If we compare these two cases with each other and then with the Schneider case, we find that they all represent different degrees of the same type of brain disturbance.

Anatomically, the brain injury occupies approximately the same place in the M and T cases, although it may be in the latter a little more upward. In the first case, considerably more destruction of brain matter can be admitted than in the T case, due to the tear produced by the intracranial trajectory of the bullet, although the damage has been limited to the most superficial zones. In the T case, there is less disturbance because the injury is a contusion type. As for the possibility of deeper or more distant anatomical lesions, there is no reason in the series of examinations of either case to indicate their existence. On the contrary, it can be established with great certainty that in both patients the brain injuries are confined to the site of the wound. In the Schneider case, he also presented a left parieto-occipital injury in its middle and upper portion with direct damage of the brain mass by the bullet.

As far as the recovery from the symptoms and residual signs, subject T has improved much more in relation to his initial condition than subject M. For example, the loss of orthogonal function has disappeared since 1942-43 and polyopia is much less severe nowadays than it was in 1938. In any case, his disturbances have always been quantitatively much less pronounced than in the M case. The different degree of recovery, which can be explained by the different type of brain injury, more recoverable in the T case than in the M case, now further separates these two cases from each other in terms

of the intensity of their brain disturbance. Residual phenomena, especially cortical epileptic seizures, have occurred very rarely in patient M, a total of a couple of seizures in more than five years. Instead, they are more frequent in patient T who suffers them every six months or more, due both to possible cortical irritations of the scar and to the type of irregular life he leads (occasional drinker, etc.). These seizures, at the moment, increase the intensity of the disorders, but it is a transitory state that lasts a very short time. In both cases, it can be stated that the degree of brain disturbance is completely stable and fixed since 1942, so that measurements on sensory functions from one year to the next are usable. The same stability is presented by subject Schneider who after twenty years continues in the same situation as that described in the first two studies dedicated to him.

Concerning the symptomatic manifestations, we already know that the dynamic action, according to the characteristics exposed above, is fulfilled in our two cases and gives the correct explanation to the Schneider case. But at the same time, the two patients studied here are quantitatively very different types of the same brain syndrome. Apart from the cerebral repercussion of the lesion, which causes a disorder common to all sensory systems and in both halves of the body, the excitability disorder is very different due to the different amount of brain mass excluded by the lesion, resulting in two types of physiological level. Thus, considering the visual field whose condition can serve as an index of the degree of disturbance of visual functions, subject M shows an intense reduction since the vision of the usual test of 0.5 cm reaches only up to 6 degrees, whereas in the T case it is possible to reach up to 50 degrees, which represents a small reduction. Similarly with other functions, e.g. with regard to visual image orientation, M presents an inverted vision of 170 degrees, whereas T presents a maximum tilt of about 30 degrees; and so on for all the other visual functions and any other sensory system. The dynamic manifestations of functional sensory lag (functional disaggregation), facilitation effect, etc., which we shall see further on, are also related to the different degree of disturbance. Nonetheless, thanks to the summative effect of facilitation, an approximate quantitative similarity between these two cases can be obtained. For example, with respect to vision, subject M in binocular vision and under maximum muscular contraction can remarkably resemble subject T without facilitation and using only his worse eye.

As regards the Schneider case, several facts we know about it allow us to place it somewhere in between our two cases, the M, which is more severe, and the T, which is less intense. Thus, Schneider reaches up to 25 degrees in the concentric reduction of the visual field. The loss of visual perception of motion was quite remarkable in Schneider, as well as in M, whereas it is extremely small in the T case. For all this, it is perfectly feasible not only to ascribe to the Schneider subject an intermediate position between our two patients, but also to attribute him a series of disorders that have not been found in him and that he should necessarily present. Furthermore, subject M by means of facilitation can easily be equated to subject Schneider, and in this way we can predict with great accuracy and detail the complex brain disturbance that corresponds to him.

2.2 The central syndrome

On the basis of our two cases and the one studied by Goldstein and Gelb (1918, 1919), we have enough reasons to establish a new well-defined type of brain disorder that we shall briefly call *central syndrome*, and more precisely, central syndrome of the cerebral cortex.

It is clear that, according to all the above, the central syndrome is characterized by a new physiological and dynamic conception of brain activity in opposition to current theories of brain pathology.

The central syndrome is defined by the properties that we shall expose in what follows. First of all, the most characteristic feature of the symptomatology is the effect of the cerebral repercussion that causes the typical *symmetric disorder*, that is, the equal alteration of the two halves of the body. Regarding the anatomical lesion, it involves *central* cortical areas that correspond to the so-called association areas, different from the projection areas that occupy a marginal or peripheral position. Added to this is the excitability disorder which, according to dynamic actions, causes a *dynamic reduction* in brain functions. Next, we shall detail the characteristics of this syndrome that, ultimately, in its multiple aspects and developments, represents the topic of this work and the starting point for brain dynamics.

2.2.1 Total and symmetrical repercussion disorder

The lesion of central areas of the cerebral cortex, from which this syndrome takes its name not only because of the anatomical position of the lesion but mainly because it damages brain areas that function as true nervous centers, affects regions that disturb the functional dynamic unit of the brain to a maximum degree. These regions are what were called association areas in the past, which are surrounded peripherally by the projection areas (marginal zones) where the long pathways of the brain end (or start). It could be admitted that these central areas only act by increasing brain excitability in a non-specific way and thus subordinating the activity of all other functionally related brain regions, which means the dynamic unity of all regions. Thus, the destruction of brain matter in central areas causes, by dynamic repercussion, a disorder that affects all the functionally unified territories. In our two cases and also in the Schneider case it has been shown that a lesion in the most central region of the sensory part of the brain disturbs all sensory systems and in all their functions, both the simpler (lower) functions and the more complex (higher) ones. Furthermore, there is something perhaps more special, which is the alteration on both sides of the body and equally, that is, on the whole extension of the sensory field. Thus, the left lesion affects both the contralateral and the homolateral side and both in a fairly equal way resulting in a symmetrical disturbance. This alteration of all the sensory systems and in both halves of the body is due to the dynamic unity of both one brain hemisphere and the whole of the two hemispheres.

It can be said that the more symmetrical the alteration in both halves of the body, the more central is the lesion, and it can also be expected that the alteration is extended to more than one sensory system. Thus, when we find a concentric reduction of the visual

field, we should expect alterations of the same type for other sensory systems (touch, hearing, etc.). In contrast, as the asymmetry becomes more noticeable, i.e. one side is more altered than the other, the central syndrome tends to become a syndrome of marginal or projection zone, and there will be fewer altered sensory systems. In the extreme case, there will be only one alteration with maximum asymmetry and for only one sensory system.

Regarding symmetry, the M, T and Schneider cases can be considered as prototypes of the central syndrome.

2.2.2 Excitability disorder

If we consider a physiological basis and use the concept of nervous excitability, we find very interesting aspects in the central syndrome.

First of all, excitability is reduced as expected after the destruction of brain mass, which directly influences excitability dynamically. The reaction time becomes longer, and an increase in the intensity of the stimulus is necessary; in other words, the most immediate change is a greater demand for the two factors involved in nervous excitation: time and intensity. This change causes a series of remarkable phenomena, depending on excitability, that we already know from the exposition of dynamic action phenomena in previous pages. Along with a long reaction time, other phenomena arise such as iterative excitability and the facilitation phenomenon, both different manifestations of nervous summation. Thus, where there was only excitation by a single stimulus in a normal individual, there is now also iterative excitation by rhythmically repeated stimuli, whose action lies in the accumulation of residual effects that add up until they trigger the reaction of the organ in question. It is similar to the changes that occur in experimental animals when parts of the spinal cord are separated from the brain. An iterative excitability then appears for reflexes, which were obtained by single excitation in a normal situation. In our cases, iterative excitation allows significant savings in the intensity of the stimuli compared to a single stimulation.

As for the action of facilitation, it increases the excitability of the centers and thus compensates for a large part of the brain mass destroyed, thereby significantly reducing the various alterations of excitability and establishing a more favorable physiological level.

With regard to the intensity of the nervous excitability disorder, it depends entirely on the amount of brain mass excluded by the lesion. Thus, the disorder is important in the M case and much less pronounced in the T case. As for the deficit in elementary excitability (increased rheobase and chronaxie), the two types of summation, by iteration and facilitation, have a corrective effect on it.

A very important manifestation of the nervous excitability disorder is *asynchrony*, by which sensory functions are out of phase leading to the dynamic reduction described below.

2.2.3 Dynamic reduction

The excitability disorder causes a functional disaggregation or asynchrony. Depending on the physiological demands of sensory functions, an asynchrony arises that leads to a simplification or dynamic reduction of these functions.

As already mentioned, a sensory function that in a normal subject behaves practically as an all-or-nothing effect, i.e. it is shown in its totality or fails completely, is here disaggregated into partial functions with different degrees of excitability due to the pathological asynchrony, giving rise to a partial effect. The greater the disturbance in excitability, the more evident this partial effect becomes. This is the case, for example, with the inverted vision phenomenon; a strong stimulation generates normal vision, a moderate one produces tilted vision, and a weaker one causes inverted vision. The same applies to any other aspect of visual function, such as the visual field, which shows in case M a considerable concentric reduction by exclusion of the less sensitive peripheral areas, due to the excitability deficit.

By gradually excluding the more complex or less excitable activities when faced with the action of usual stimuli of medium intensity, it turns out that sensory functions get simplified, say dynamically reduced since they are lost in relation to the degree of physiological demand characteristic of each one of them. Since dynamic reduction is closely dependent on the degree of excitability deficit, the reduction in subject M is much greater than in subject T.

In short, dynamic reduction is a natural consequence of nervous asynchrony.

The syndrome opposite to the central syndrome is the *marginal* (or peripheral) *syndrome;* both are the two main forms of a brain disorder. The marginal syndrome corresponds to a lesion of the cortical projection areas, so it is well known in brain pathology, unlike the central syndrome. A lesion of the projection areas, which are point of origin of the long pathways, is almost equal to the destruction or interruption of these pathways, resulting in a *localized* disturbance in contrast to the general alteration of the central syndrome because of the dynamic repercussion of the centers. In the case of a marginal lesion, which occupies a peripheral position in relation to the central zones as its name indicates, there is no symmetry effect as we have seen in the central syndrome. On the contrary, it causes a localized effect that disturbs in a very circumscribed way contralateral functions, for whose activity the conductibility of the long pathways that go through the marginal zone is vital. Taking the visual field as an example, the marginal visual lesion causes a hemianopic alteration in which the homolateral halves on the side opposite the lesion are blind whereas the other two halves remain unscathed. Nor should we expect other alterations outside the visual domain, i.e. touch and hearing, for example, can remain totally free.

This would be a type of extreme marginal disturbance, and it can be said to be more of a nervous pathway lesion than a lesion of the cerebral cortex, the latter being regarded as a nervous center. Thus, the marginal syndrome shows a type of disturbance that is essentially asymmetrical. Between the pure central syndrome and the marginal syndrome there is a whole series of intermediate types, and it could be said that most brain lesions belong to these intermediate types. In fact, it could be stated that every cortical lesion that

does not exclusively affect the above-mentioned pathways, shares to a greater or lesser extent properties of the central syndrome. This will be studied later, and now the indication made about the types of brain disorder is enough, which helps to better characterize the central syndrome.

2.3 Brain dynamics issues raised by the central syndrome

The characteristics of the central syndrome constitute, above all, a true *experimentum crucis* to decide about the working of the brain, and what comes out leads directly to a dynamic conception of the brain. Thus, the brain lesions of the two cases studied here enlighten us about the mechanism of brain centers, completely ignored until now. This has brought about a profound change in the general ideas, which raises fundamental problems in the theory of the nervous system that can only be solved from the point of view of the dynamics of the brain. Next, we shall deal with three of the main issues: physiological level, sensory structures and brain system. We shall now restrict ourselves to indicate little more than their existence to highlight the most important theoretical consequences derived from the central syndrome. They will be the content of the third part of this work, devoted to the development of the general fundamentals of brain dynamics.

2.3.1 Physiological level

The excitability criterion is of primary interest for the physiological rationale of brain activity, hence, the first and most elementary issue to be considered in brain dynamics is the *physiological level,* which is an expression of nervous sensitivity to stimuli. The phenomena found here allow us to consider the cerebral cortex as a dynamic unit, that is, as a functional unit resulting from the activity of all the elements that compose it. In this ensemble, the amount of nervous mass of the centers determines the excitability level (physiological level); hence the exclusion of a portion of such mass causes a decrease in the functional level due to the subtraction of nervous energy that this entails. This is what happens, as we have already seen, in the central lesion of our two cases and also of the Schneider case.

There are precedents of this approach, some very old, for example in the doctrine of Loeb (1899) by considering in a very broad way the activity of the nervous system as a simple action of reinforcement of the natural sensitivity of the organism to the stimuli of the external world. In a more precise and concrete way there is a precedent in the experiments and explanations of the physiologist Lashley (1929) who continued the work of Flourens (1824), confirming and extending it. Lashley (1929), working with rats trained in the labyrinth´s intelligence tests, found a close relationship between intelligent behavior and the amount of brain mass destroyed. Consequently, he formulated the *mass action* law, considering that this brain mass is *equipotential*, i.e., of non-specific physiological activity and with the only effect of reinforcing or activating in a general way brain functions. More modern research on general physiology of the nervous system

is also going in the same direction. In particular, Lapicque M (1923), Lapicque L and Lapicque M (1928), Lapicque L (1934), and his many followers demonstrated the interaction between the nervous elements with each other by means of nervous *subordination* effects, which highlight a much more dynamic functional structure than that assumed by the static anatomical theory of pathways and centers. The end of the predominance of the anatomical view is evidenced in the constant progress of such dynamic conceptions in the physiology of the nervous system.

The activity of the central mass, resulting from the sum of actions of the nervous elements that compose it, can be replaced by various other nervous actions, as seen in the phenomenon of facilitation, thus making it possible to largely compensate for the destruction of the nervous mass. All these circumstances prove the non-specificity of the nervous influx, which acts simply by summation, increasing sensitivity to external stimuli.

2.3.2 Sensory structures

The study of sensory activity in brain pathology has so far been established, on the one hand, from an intellectualist psychology and, on the other hand, by trying to determine an anatomical localization in the cerebral cortex for each type of function, thus establishing, psychologically and anatomically, an endless series of "atomized" brain syndromes. Contrary to such theories, the facts found here evidence the functional unity of each sensory system, whose activity depends on the state of excitability of the system (receptor and nervous mass linked to it).

As for the sensory organization, it is carried out by means of the nervous mechanism of synchronization, i. e. excitability levelling of functional elements, thus obtaining more differentiated structures. This view, systematically adopted in the study of our cases, is an application of the theory of nervous isochronism of Lapicque (1937) to the human brain.

Also from a phenomenological point of view, it can be useful the dynamic conceptions of the Gestalists, such as Köhler (1930), Wertheimer (1912), Koffka (1919/ 1935), etc. It should be noted as a precedent, the indications of Stein (1928, 1930) in favor of basing all kinds of sensory activity, from the most elementary to the most complex, on the characteristics of nervous excitability.

In any case, it is clear that both *psychologism* and *anatomism* are insufficient guidelines that must give way to more physiological conceptions.

2.3.3 Brain system (brain organization)

The central syndrome has very important consequences for the functioning theory of the cerebral cortex, almost completely dominated until now by the theory of anatomical localization. Nonetheless, this conception has always had its weak points and, although in general it is dominant, it can be said that authors have been divided into localists and anti-localists from its beginning. Although the theory of anatomical localization seemed to be well founded in restricted areas (projection cortex), there were still large areas to be

explored and the physiological laws of the nervous mechanism were very little known. Thus, generalizations have been premature and highly questionable. Even before physiologists and pathologists established the theory of brain localization, Flourens (1824), whose conception could be very well rehabilitated here, had considered the cerebral cortex as a functional unity, based on his experiments in laboratory animals, in which a destruction of brain mass caused a reduction of all brain activity and not of a particular function. We have already indicated how Lashley (1929, 1933, 1937) has corroborated and specified such a conception. In a similar way to Flourens, the physiologist and nervous system pathologist Goltz (1881) soon opposed to the nascent theory of brain localization, considering that function goes beyond structure.

The non-specificity is a consequence of the physiological level we have discussed above, assuming that the central mass operates only by activating nervous excitability without any qualitative specificity. Thus, the rebuttal of cerebral localization is a direct consequence of the phenomena indicated on brain excitability.

However, the cerebral cortex does not have a completely homogeneous activity. For this reason, the cerebral cortex could be understood as a dynamic system, both because it constitutes a functional unit and because it changes its effects according to its different regions. The two extreme types of cerebral syndrome (central and marginal) limit a series of intermediate cases that prove the existence of a dynamic system layout.

We can say that the effects of a lesion on the brain system depend on the two following factors: the amount of brain mass excluded and the position of the lesion. Thus, the effect produced is in relationship with the *magnitude* and *position* of the lesion, fact that perfectly fulfills the conditions of a dynamic system. The magnitude, i.e., the amount of mass destroyed, determines the intensity of the excitability disorder and the degree of dynamic reduction suffered by sensory functions; the position of the lesion determines the type of distribution of the disorder, which is symmetric for the central syndrome and asymmetric for the marginal syndrome, counting also with the intermediate types such as a disorder of general distribution but with asymmetrical predominance.

This is how the problem of brain functioning can be addressed and the thorny issue of brain function localization theory solved. It is important to note that, except in the extreme marginal syndrome, which represents in fact a direct lesion of the nervous pathways, we should not speak of focal symptoms when referring to the cerebral cortex, contrary to conventional ideas. It is, of course, completely impossible when it concerns the central syndrome, since the theory of brain localization then fails completely. For this reason, it might be said that the dynamic conception developed here is the end of the controversy about localization of brain function.

This dynamic conception of the brain system is the most immediate and simplest way possible to understand the facts derived from experiments. We can concretely illustrate the theory by resorting, as in other occasions, to the behavior of the visual field in different types of brain injuries.

In the patients studied here, we have the type of lesion corresponding to a concentric reduction, that is, a disorder that can only be understood from a dynamic conception and that conforms perfectly to all indicated in the central syndrome.

For a marginal lesion, we have the hemianopic alteration, in which only the halves of the opposite side of the lesion are blind, and here, of course, the theory of localization is valid since we are dealing with an interruption or damage to the nervous pathways in the cerebral cortex (calcarine) where they end up. However, the symptoms may appear more complex in the first moments due to a certain nervous repercussion on the rest of the undamaged field, indicating that the dynamic mechanism is always present in all types of lesion.

Finally, we can still consider an intermediate type of disorder produced by lesions that, being in the projection areas or bordering areas, do not cause a direct alteration of the pathways. In these cases, if the patient is examined with due precision, it is usual to find a somewhat general distribution of the brain disorder but with asymmetric predominance. Most of the best known cortical syndromes or lesions undoubtedly belong to this type. In such a case the visual field shows the following characteristics: for each eye, the half opposite the lesion is blind except for the central zone, which can be restricted to the macular region; then the dividing line between the blind and healthy parts of the field of each eye is not a straight line but has a small concavity in the center, which corresponds to the most central zone (*Aussparung* of the macula according to German authors, whose point of view has been highly discussed). But the other half of the visual field, presumably intact, is not, since when it is properly examined, some degree of alteration can always be found. This alteration may range from a slight narrowing of color isopters to a concentric reduction affecting the most peripheral zones of the visual field of that half. Therefore, it turns out that both halves of the field are involved, albeit unequally, but according to the same mechanism. In the contralateral half, the functional reduction is so intense that vision is limited to the macular area (the most sensitive), whereas in the other half, the repercussion effect (action at a distance) is so weak that the alteration of the field manifests itself only in the most peripheral regions of the field, which are more difficult to excite, i.e. less sensitive.

Through these three examples, the action of the brain dynamic system we advocate is specified; the dynamic action applying to all cases. For the moment, it is sufficient with the indications made on fundamental issues derived from the central syndrome and its theoretical scope for a theory of the brain.

3 Dynamic analysis

3.1 Dynamic action and experimental analysis. Physiological levels

The special conditions of excitability make it possible to carry out experiments of great importance to address the study of hitherto unknown brain mechanisms. Experiments of a great demonstrative effect can be carried out on broad aspects of sensory activities, so that the factors involved in their formation can be discovered.

Basically, the exposition we shall make of brain dynamics is just the detailed study of the central syndrome. This special type of disorder offers very broad perspectives for research because, on the one hand, it allows any sensory system to be addressed in a single individual since all sensory systems are affected to the same degree, and on the other hand, because the special change in nervous excitability reveals the nervous mechanism that governs brain activity.

By supporting all research on a strict physiological basis, the study is developed free from psychological views and procedures that only obtain hypothetically interconnected data without reaching a direct and simple foundament of the phenomena. Even having to rely on subjective phenomena in the examined injured subjects, the analysis is ultimately well-established on the objective behavior of these subjects facing tests and experiments that, properly combined, allow a safe control of the brain disorder.

Along with the experimental analysis of the *qualitative* factors that determine an activity, there is a *quantitative* assessment of them, this being the only way to complete the information and to specify accurately the characteristics of the phenomena. Thus, in our two cases we try to express the different relationships of nervous excitability that intervene in the sensory functions by means of curves and graphs.

The dynamic nature of the fundamental disorder of nervous excitability determines the conditions of the experimental analysis to be carry out. This is not the place to explain in detail the causes of that fundamental alteration originated by the loss of central mass, but just to anticipate that it is characterized by a great *slowness in nervous reaction,* which gives rise to a special *asynchrony* of the nervous elements that integrate brain activity. A manifestation of that slowness is the alteration of the strength-duration curve of the elementary excitation, and a manifestation of the asynchrony is the fundamental experiment of sensory lag that causes a disaggregation of sensory functions into partial reactions.

On the other hand, effects of summation by iteration and facilitation, caused by the essential disorder of excitability, act by partially avoiding this slowness as well as much

of the asynchrony. In particular, the action of facilitation in replacing to some degree the loss of central mass, especially in the more profoundly altered M case, allows to generate new types of physiological levels, thus providing a wide range for experimentation. Thus, there are several factors to consider in the experiments, factors that we shall use systematically in the study of brain functions. Given the general characteristics indicated, the study can be called dynamic analysis.

3.1.1 Physiological levels

Figure 4 schematically shows the change in the strength-duration curve of any sensory receptor, due to the loss of central brain mass. The lower curve (the one closest to the axes) corresponds to the excitability of a normal individual, and the upper curve represents the activity of subject M, who has the greatest brain mass deficit. Between these two curves we have the curve corresponding to the facilitated state by maximum muscular effort in the M case, and those expressing the state of excitability of the T case. Slightly below the T-curve is the curve for the subject T under maximum facilitation which causes a very small descent, as can be seen. Thus, a number of excitability states or *physiological levels* result depending on the loss of brain mass, as well as facilitation which compensates to some extent for this loss. Facilitation by maximum muscular effort produces a large increase in excitability in the M case and a much smaller increase in the T case. Thus, it is observed that the more pronounced the disorder, the greater the effect that can be expected from facilitation. The same happens with iteration, whose saving action in stimulus intensity is much more pronounced in M than in T, as we will see later on. However, there is not a complete recovery to the normal level of excitability but, as we have seen, the maximum facilitating effect is still quite far from the normal level.

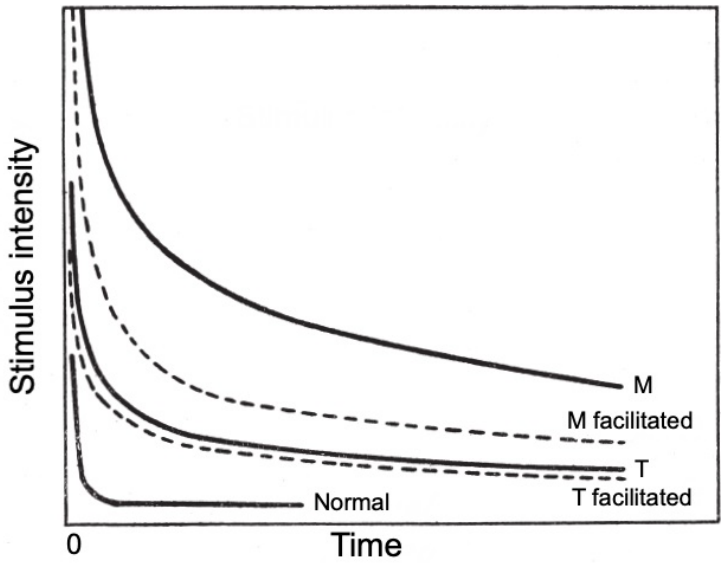


Figure 4. Schematic representation of the state of brain excitability according to the strength-duration (intensity-time) law in the M and T cases, without facilitation and under its maximum action. A stepwise series of different physiological levels can be appreciated.

The curves are characterized by two excitability factors: rheobase, which is the lowest value of intensity that produces a response, and chronaxie, which is the time corresponding to twice the rheobase. It should be noted the degree of curvature. In this series of curves, there is a gradation in the types of excitability in which the deficit is due to an approximately parallel increase in the two excitability parameters, time and intensity. Consequently, the curvature of the curve becomes less pronounced and therefore, the reaction speed is lower in the curves further away from the axes.

This series of excitability levels offers the possibility of investigating brain activity under very different conditions and, as we see, this is due not only to the existence of two subjects with a very different intensity of their lesion, but also to the combinations that can be made by means of facilitation, especially in the case of M. In Fig. 4, only the curves for the subjects in inactive state (without any facilitation) and under facilitation by maximum muscular contraction are indicated. However, other types of facilitation are still possible. By combining the effects of various facilitations, it is possible for the M case to become physiologically equivalent to the totally inactive T case. Although this equivalence may not be fully achieved, the physiological levels of both cases become very close to each other under these conditions. Thus, we have the possibility of converting, for example, the visual perception of subject M into that of subject T as long as the former is under the action of maximum muscular contraction and in binocular vision.

The very diverse types of facilitation (muscular contraction, sensory action, binocular vision, movements, etc.) give rise to very numerous types of physiological levels, whose exact knowledge is indispensable to establish with certainty the quantitative determinations in the experiments. In addition, facilitation is a very important means of extending and controlling all kinds of experiments, as we shall see later on.

3.2 Fundamental experiment of asynchrony

The previously mentioned nervous asynchrony has its most immediate manifestation in the *fundamental experiment* on sensory lag (or delay), of great importance in the analysis of sensory functions.

We have previously dealt with the excitability deficit due to a loss of brain mass. This deficit affects differently the nervous elements that are involved in brain activity and gives rise to a functional asynchrony that is easily revealed in the fundamental experiment. Nervous asynchrony, the degree of which depends on the excitability deficit, is also greatly reduced by facilitation in the same way that the level of excitability decreases in the strength-duration curve. But now the normalization is not complete either, although the pathological alteration is significantly reduced in the same way as in the curves shown in Fig. 4.

Using strength-duration excitability relationships, the fundamental experiment is presented under two types of graphs that are different expressions of the same asynchronous process: the asynchronous bundle of curves, each one corresponding to the

different (now independent) partial functions resulting from functional disaggregation by sensory lag; and the sensory recruitment curve of these disaggregated functions. These relationships are complemented in a very important way by the summative action of facilitation which, by correcting the disorder through central action, tends to synchronize the disaggregated functions.

Next, we shall briefly indicate the characteristics of the excitability relationships in the fundamental experiment.

3.2.1 Asynchronous bundle of disaggregated functions

Functional sensory lag spreads to all kinds of activities of any system: vision, touch, hearing, etc. As already indicated, it was first found in the phenomenon of inverted vision in which we already know the different effect of strong and weak stimuli; strong stimuli give normal or almost normal vision whereas weak stimuli, on the contrary, give inverted or strongly tilted vision. If in this phenomenon, which can now be considered a paradigm of sensory lag or asynchrony, we determine the strength-duration curves for the various functional levels (different orientations of the perceived visual image), we find a series of curves like those shown in Fig. 5.

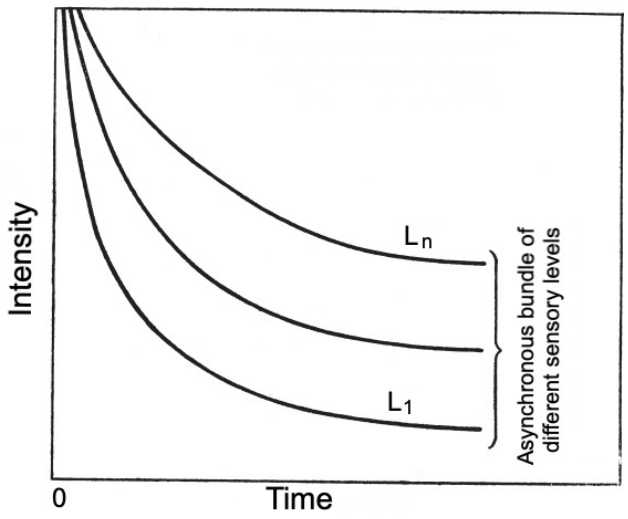


Figure 5. Schematic representation of the asynchronous bundle of curves corresponding to the fundamental experiment of asynchrony. The strength-duration (intensity-time) curves correspond to the disaggregated partial functions that would normally form a single curve (single sensory level).

What in a normal individual would give a single curve, now, due to asynchrony, becomes a bundle of curves placed far above the normal value; the greater the asynchrony, the wider the bundle. In this family of curves, the lowest curve corresponds to the most sensitive sensory function or level (highest excitability), which in our case is inverted or strongly tilted vision, and the uppermost curve corresponds to normal vision. Between these two curves there is a continuous series of curves corresponding to the different tilts

(intermediate functional levels). The schematic representation in Fig. 5 shows that the higher the curve, the lower the curvature (indicating a lower reaction speed); all the characteristics of the higher curve indicating a lower sensitivity.

In the asynchronous bundle of curves there is a series of sensory levels corresponding to the disaggregated partial functions, sensory level being understood as the rheobase threshold of the sensory manifestation. Due to the property of the strength-duration law, the curves of the asynchronous bundle tend to be closer together for strong stimulus intensities, and to separate from each other for small intensities or long times, showing then their respective rheobase levels.

The greater the excitability deficit, the more pronounced the asynchrony and therefore the greater the separation between the curves. Hence, this separation is much more pronounced in the M case than in the T case, and by means of facilitation it is possible, especially in the first case, to shorten such separation by a considerable degree. At the moment we are only considering, for the sake of simplicity, the general aspect of the asynchronous bundle without paying attention to the restrictions that each case imposes according to the intensity of the lesion and permeability to facilitation.

3.2.2 Recruitment of desynchronized levels

The other aspect of the fundamental experiment lies in the recruitment of the disaggregated (desynchronized) partial functions, and is more easily revealed than in the previous relationship about the asynchronous bundle.

In the recruitment experiment, the rheobase levels of the disaggregated functions can be reached by progressively intensifying the stimulus. That is, each sensory level is related with a certain stimulus intensity, without intervention of time, which is assumed to be unlimited because we are dealing with the rheobase threshold. In the case of visual image orientation, changes in the intensity of the stimulus easily occur when an object moves away from or towards one of these patients because the visual angle at which the object is seen changes as well as the intensity of the light reflected by the object. By increasing the intensity of the stimulus in this way, it is possible to go from the strongest inversion to the most normal upright orientation, the sensory level being determined by the degrees of rotation of the visual image.

This is an experiment of sensory growth as a function of the intensity of the stimulus, expressed by Fechner´s law (Fechner, 1860) which is also fulfilled in this pathological case of nervous asynchrony, as can be seen in Fig. 6 where the recruitment experiment is schematically represented. By taking the logarithm of the stimulus intensity at the abscissa, the sensory level grows directly proportional to the logarithm of the stimulus, although this relationship is only fulfilled exactly for the central part of the curve.

As can be easily understood, this recruitment curve is related to the curves that form the asynchronous bundle. Both types of experimental curves are aspects of the same phenomenon.

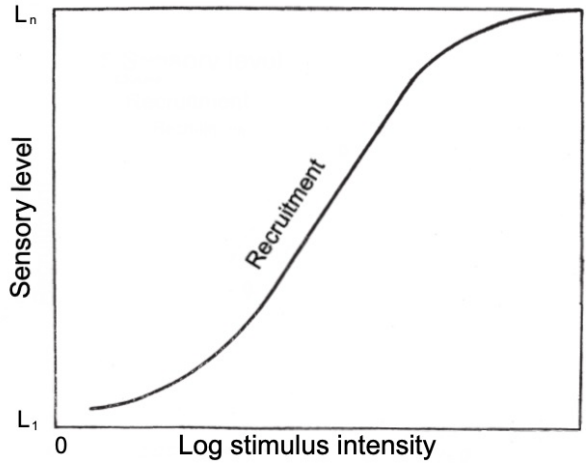


Figure 6. Recruitment of sensory levels as a function of the logarithm of stimulus intensity. The levels correspond to the rheobase thresholds of the desynchronized (disaggregated) functions shown in Fig. 5.

Another curve of the same type is obtained if, instead of relating the sensory level to the intensity of the stimulus acting for an indefinite time, the action time of the stimulus is the variable on the abscissa for a fixed medium intensity. In this case the recruitment is determined as a function of time, i.e. we obtain the development in time of the series of desynchronized functions, from the simplest (and fastest) to the most complex (and slowest). In this type of experiment, easy to carry out due to the great slowness in the reaction of the subjects, it is necessary to fix a medium intensity of the stimulus that cannot be lower than the rheobase threshold of the highest sensory level, otherwise only lower levels would be reached by the action of time, and an incomplete recruitment would be obtained. We can see in Fig. 5 the basis of the two types of recruitment exposed, that is, either by the action of increasing stimulus intensity or by the action of increasing time (a much slower process), a succession of sensory levels appears from the most anomalous function to the most normal one which appears last.

3.3 Synchronization by facilitation

The new type of experiment that completes the sensory analysis by making use of dynamic phenomena, is synchronization by means of facilitation; the latter acts in a summative way on the centers of the cerebral cortex, thus obtaining an action that is opposite and to a certain extent symmetrical to that of asynchrony.

Taking again the example of inverted vision, we consider the situation in which subject M, being in an inactive state (free of any facilitation), perceives the inverted (or almost inverted) image of an object due to its distance, size and illumination conditions. If then he makes a muscular contraction, a re-inversion is produced; the greater the muscular effort, the more pronounced the re-inversion. This type of facilitation can easily be measured if the subject holds a certain weight under conditions that will be detailed later on when the different experiments will be explained in detail.

Figure 7 represents the effect of synchronization by facilitation due to muscular effort, showing the growth of the sensory level as a function of the logarithm of muscular effort, this one measured by the weight held by the subject. The starting point is the state of maximum asynchrony, in our example it is the most inverted image, which corresponds to the lowest sensory level and also to a state of absolute inactivity of the subject; then the degrees of tilt of the visual image corresponding to each weight held are determined. If these degrees are indicated on the ordinate, and the logarithm of the held weight on the abscissa (the weight can be up to 80 kg to reach maximum synchronization), a sensory growth curve is obtained analogous to that obtained in the previous experiment of sensory recruitment by stimulus intensification. But whereas in the recruitment the increase of the stimulus intervenes, in the experiment with facilitation the sensory increase is due to the summative effect exerted on the nervous centers, synchronizing the sensory levels, that is, equalizing their excitabilities and forming again a unitary function.

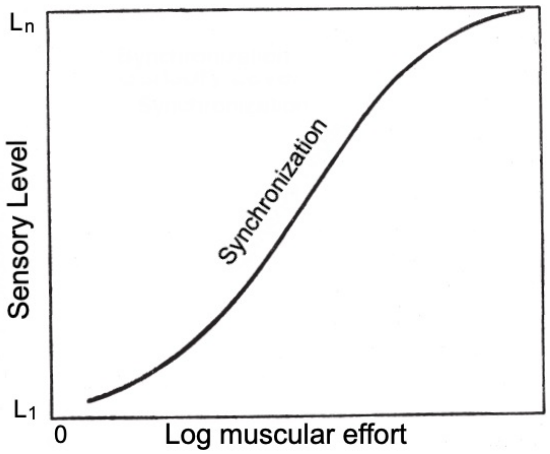


Figure 7. Diagram of the effect of synchronization by means of facilitation due to muscular effort for a fixed stimulus intensity, showing the growth of the sensory level by the progressive action of the applied effort.

However, even maximum facilitation does not achieve *ad integrum* recovery. Thus, if in the described experiment we start from the most strongly tilted or inverted position, it is not possible to achieve by means of maximum facilitation a complete re-inversion of the image, although one very close to it. Let us recall that the degree of permeability to facilitation is very diverse according to the degree of brain disturbance; it has a great effect in the M case and is much weaker in the T case, diminishing the effect notably as the excitability deficit decreases. In any case, the action of facilitation in both subjects is so great that in the conditions of ordinary life, it practically compensates for the defect of asynchrony. Only in the most accurate determinations of the experimental tests, it can be shown that a small asynchrony persists even with maximum facilitation.

In addition to the two ways of growth of sensory level, by intensification of the stimulus and by facilitation, there is a third type due to the existence of iteration. In this type of stimulation, a stimulus of constant intensity corresponding to the lowest sensory level is applied repeatedly, resulting in higher and higher sensory levels as the number of

stimuli increases. This is an iterative recruitment in which the accumulation of stimulus residues, which slowly fade due to the state of nervous excitability, causes the stimulation effect to progressively increase. The curve thus obtained is similar to that of recruitment by intensification of a single stimulus. Therefore, there are three different forms of sensory growth depending on the stimulation conditions: recruitment by intensification of a single stimulus, recruitment by iterative stimulation and synchronization by facilitation, with similar curves being obtained in all cases. We can say that sensory growth is proportional to the logarithm of nervous stimulation provided in one way or another.

Whereas iteration is in fact a special mode of stimulation, i.e. it is a sum of successive stimuli acting directly on the receptor in question, facilitation acts by modifying the fundamental excitability disorder (the excitability deficit and asynchrony). The change in the excitability level (or physiological level) by facilitation due to maximum muscular effort was already indicated in Fig. 4, bringing the strength-duration curve of the elementary response of the sensory receptor closer to the normal value. Facilitation works by reducing asynchrony in the fundamental experiment, and decreases iterative capability. The latter effect is due to the fact that the sensory system becomes faster, more sensitive and more synchronous; hence the saving obtained by iteration (in the intensity of the stimulus) with respect to a single stimulus and the degree of iterative recruitment are reduced. In summary, it is sufficient to emphasize that the fundamental experiment of asynchrony and the effect of synchronization by the progressive action of facilitation are experiments that close a complete cycle of tests in the dynamic analysis of sensory functions.

3.4 Sensory asynchrony

We have indicated the characteristics of the fundamental experiment and next we shall expose the meaning of the sensory lag (asynchrony) that arises in that type of experiment.

As already mentioned, the nervous origin of this type of phenomena lies in the asynchrony of the nervous elements that constitute the centers of the cerebral cortex. By virtue of asynchrony, not only the interval or gap of excitability existing in the normal individual between the diverse functions increases, but also other intervals or gaps are created where there was no separation, thus appearing new modalities of reaction to the stimuli. An example of the pathological increase of the normal interval (gap), due to asynchrony, is found in the photochromic interval of colors, which being minimal and even almost imperceptible for some of them in a normal individual, is extremely increased and clearly appreciable in most colors. The same occurs in other differential intervals such as those that exist naturally along the visual field meridian in which sensitivity decreases rapidly from the center to the periphery, or in other functions such as spatial discrimination as a function of stimulation intensity, etc. In all these cases, due to the increased interval, a higher intensity of stimulation is needed to overcome it, which means a loss of differential sensitivity.

Therefore, the most remarkable characteristic of asynchrony in the fundamental experiment is to generate new intervals (gaps) of excitability, thus causing the disaggregation of functional complexes that in a normal subject react "all at once", that is, according to the all-or-nothing effect. Due to the new intervals generated, the functional complexes are dissociated or fragmented into independent functions, giving rise to partial reactions or effects.

It can be admitted that, both for the normal intervals (normal differences in excitability) that become larger and for the new intervals generated, we are dealing with an abnormal increase in excitability differences, which within normality only some of them are evident whereas others are insignificant without establishing discontinuity. These new intervals, by fragmenting the unitary functional complexes, provide a kind of analysis of the sensory structures. In this analysis we must distinguish the phenomenic aspect of the sensory manifestations and the level of excitability (physiological characteristic) of each of them. In this way, the mechanism of formation of the sensory organization is revealed through the separation of the factors that compose it; these factors are ordered in terms of their excitability according to their degree of sensory differentiation. For example, the process of tactile spatial localization is disaggregated, first appearing the sensation of simple contact without any indication of the location of the stimulus, and then appears, as a last sensation, the function of precise localization, both processes being completely independent and separated from each other, which is completely impossible in a normal individual. Between the two processes there are several intermediate states corresponding to diffuse (irradiated) localization, similar to what happens in flat color vision (chromatic irradiation), as we will see later.

Particularly important for the analysis of functions are cases of asynchrony in which extremely unusual manifestations occur that have no precedent in normal sensory activity, such as the phenomenon of inverted vision and chromatopsia (colored vision), for example. From the dynamic analysis of the first example it follows that there are two different types of orientation of the visual image, one normal guided by the position and movements of the body, and another inverted on the retina which would decide the orientation. In the example of chromatopsia, in which affected subjects perceive white as pale green, the various colors that contribute to the formation of white do not come into action simultaneously because of their different excitabilities. In this way, it is possible to explore the brain mechanism in the formation of both white and other colors, thus determining which are primary and which are composite (mixed).

These examples among many others show the significance of asynchrony in the research of sensory structures and the useful applications of the dynamic analysis. The functional types that appear isolated or independent as a result of functional disaggregation are not always merely partial functions that in a normal situation are integrated into the total unified function, since it would then have to be admitted that inverted vision is included in normal right vision, and both types of vision mutually exclude each other. More accurately, all these facts must be understood on the one hand as expression of the factors involved, and on the other hand as new physiological predispositions derived from the fragmentation, representing simpler and less differentiated manifestations.

Thus, in the fundamental experiment of sensory asynchrony, we not only witness an analysis of the sensory structures but also a development and differentiation of sensory activity that goes through very d stages in parallel with their own excitability level. What is shown along the new intervals is a series of functions that by transit from one to another, an increasing differentiation and sensory organization is developed.

3.5 Dynamic reduction

A consequence of asynchrony is dynamic reduction, which means a functional simplification arising from the nervous asynchrony disorder. The latter, by producing an increase in the differential interval and even creating new ones, causes a large part of the sensory activities to be excluded under stimuli of ordinary intensity, resulting in a reduced function, but reduced according to a ranking of nervous excitabilities; this is why the process can be called dynamic reduction.

Complex functions with a high level of excitability, i.e. requiring high intensity and long duration of the stimulus, rapidly increase their excitation threshold and are replaced, in the face of ordinary stimuli, by other simpler and more easily excitable functions. The greater the asynchrony that causes the separation between the various functional levels, the more intense the reduction. All these functional levels suffer from an excitability deficit, but in greater proportion the higher ones, corresponding to more complex and differentiated functions.

It can be stated that the dynamic reduction, generated according to the exposed mechanism, is the characteristic type of disturbance of brain activity for any type of anatomical lesion and, in general, for any type of alteration in the nervous system. It is a well-known fact in nervous pathology that in the impairment of a sensory system, mainly the most complex and differentiated functions are altered, and this occurs quite independently of the anatomical location of the lesion. However, such a significant fact has not acquired all the importance it deserves on a theoretical level, and with the exception of authors such as Stein and Weizsäcker (1927, 1928), it has hardly been taken into consideration, since it is in clear conflict with the theory of anatomical localization. The mentioned authors advocate a functional unity for a given system, and reject the specificity of pathways and centers of the anatomical theory of nervous functioning. According to them, the excitability of the system is altered and its functional character varies. However, they do not sufficiently specify this change of the functional character, but we can say on the basis of our experiments that this change is entirely due to nervous asynchrony, which raises differently the excitability thresholds of the brain functions, thus causing a dynamic reduction. For example, concentric reduction of the visual field is easily explained from the point of view of dynamic reduction; by contrast, it represents an insurmountable obstacle to any attempt to explain it according to the theory of anatomical localization in the brain. This dynamic reduction of the visual field is not an isolated case but the general rule for all kinds of nervous functions.

In such simplification or functional reduction, there is no inextricable chaos of symptoms as would be the case if we had to strictly abide by the theory of anatomical specification. The lesion, by provoking the excitability deficit that gives rise to the above-mentioned asynchrony, establishes a type of nervous organization at a different excitability scale from the normal one but according to the same general laws. Therefore, there is no radical separation between a normal and a pathological state, and from a broad point of view it is possible to determine the quantitative variations that establish a rational transit from one state to another.

A notable feature of dynamic reduction in the cases of central syndrome studied here is the reversibility of this reduction due to the rather moderate inequality in the excitability of functions; this allows recruitment of the more delayed functions by sufficiently increasing the intensity of the stimulus. In this way, even in the M case using the worse eye, it is possible to gain vision in the most peripheral part of the visual field, without any facilitation and only by adequate increase of the luminous stimulus, and thus a normal oriented image can also be obtained. It is clear that dynamic reduction depends on the excitability deficit, that is, on the degree of asynchrony; thus, in our two cases we find very different degrees of dynamic reduction, which in addition can be modified by facilitation. However, in a very pronounced nervous disorder, asynchrony would distance the levels considerably from each other and reversibility disappears.

The indicated reversibility presents a type of behavior very similar to that of Pflüger's strong and weak reflexes (Pflüger, 1859, 1877), in which a weak stimulation produces a partial or restricted reaction, and a strong one causes an extended and general reaction. In all cases, the relationships of excitability and asynchrony are fulfilled in the same way.

3.6 Sensory structures

If asynchrony leads to a functional disaggregation and a simplification or de-differentiation of sensory structures, its opposite effect consists in the synchronization of the constitutive factors and in sensory differentiation. Therefore, the degree of differentiation runs parallel to the degree of synchronization.

The structures of a given sensory system respond to a unitary global activity in which it is not possible to separate autonomous processes. For example, functional vision impairment occurs in brightness, colors, field of vision, visual acuity, image orientation, and recognition and understanding of shapes and schematic drawings. The alteration affects the whole system without exception and each function suffers according to its particular conditions of physiological demand. At the beginning of these studies, following the criterion of nosological syndromes of the usual brain pathology, it seemed that we were dealing with a series of independent disorders, but a more precise quantitative examination demonstrating a general disorder, gave way to the idea of a functional unity, thus ruling out any attempt to establish nosological units and anatomical localizations.

Unlike the *anatomical psychology* exercised by classical authors of brain pathology, we shall now study all kinds of sensory activities on a strictly physiological basis, from the point of view of nervous excitability. For each sensory system, discrimination and functional organization depend directly on its dynamic conditions, each system constituting a compact activity in which it is impossible to point out processes independent from the general activity. This conception is contrary to the rigid separation of brain activities into independent faculties such as sensation, perception, recognition, etc., used by classical psychology and adapted by brain pathology to clinical nosology. Likewise, the exclusion of the principle of brain localization means the rebuttal of the usual nosology that follows the criterion that a disturbance corresponds to a focal symptom.

Instead of classifying brain disorders according to a series of entities or nosological syndromes whose number grows endlessly (they are only partial aspects of a more extensive and general disorder that goes unnoticed), this issue would be greatly simplified by taking into account the *degree of alteration of the sensory system*, i. e. the level of differentiation and organization to which it is reduced as a result of the lesion. Such a functional level is expressed both by sensory manifestations and by nervous excitability relationships. In sensory differentiation, we witness a progressive and unitary qualitative-structural development, that is, a multiplication of qualities (e.g., development of the chromatic series in innumerable hues) and an increase in the spatio-temporal relationships that give rise to forms and structures such as localization, figure, motion, etc. This long series is produced by gradual transitions in which certain functions are transformed into others without any discontinuity, and there is a continuous transition from the most primitive manifestations of sensation to the most complex intellectual activities.

However, exposition needs force us to establish divisions that have only a circumstantial value and do not compromise the unitary functional principle that has been advocated. Therefore, in the following part, the following classification of sensory functions will be used for all the sensory systems studied (visual, tactile and auditory): *qualities*, *forms* and *schemas*. Qualities correspond to specific manifestations of each system (luminosity and colors; pressure, pain and temperature; noises, sounds, etc.); forms refer to spatial and temporal structures of all kinds (localization, shapes, motion, rhythm, etc.), and finally, schemas represent a special type of structures directly derived from the last group, and constitute the basis of superior or intelligent behavior.

Sensory dynamics. Visual functions

Once the general orientation of these investigations has been established in the preceding pages, we shall proceed to study more specific issues concerning the sensory dynamics of the three most important sensory systems: visual, tactile and auditory.

This study consists essentially of investigating the organization of sensory activities, which are presented under two fundamental aspects. Firstly, the phenomenological aspect, which is the sensory expression itself, which we address either by the patient's subjective manifestations or objectively by controlling his behavior in the face of very diverse tests. The second aspect is the determination of the excitability level and other physiological characteristics corresponding to each sensory manifestation. Thus, both *qualitative* and *quantitative* manifestations of excitability are addressed. The latter is of the utmost importance for brain dynamics.

The working method consists of the application of the dynamic analysis already described. Therefore, it is mainly a matter of determining the conditions for the fundamental experiment of asynchrony and dynamic reduction in all types of sensory functions. In this way it is shown that sensory organization has its physiological basis in the degree of nervous synchronization of the functional elements of the cerebral cortex.

The investigation described below is entirely original, and whenever there is a precedent it will be duly indicated. In the exposition we shall limit ourselves to the essential facts, excluding details and discussions that would demand a much greater extension.

The sensory activity of the visual system has always provided the greatest number of problems to research, given the complexity and variety of its functions and its easier accessibility compared to other sensory receptors. Here we have a wide field of experimentation that presents aspects as diverse as: general excitability conditions, color vision, structure of the visual field, forms, motion, mechanism of the visual image orientation, etc., and finally, the most complex activity of schema function that corresponds to the so-called visual recognition.

In all these topics we present new facts and experiments. As far as possible, the experimental results are represented by graphs and curves that show in a simple and immediate way the character of the phenomena. This is performed in the most complete way, especially in the experiments on visual image orientation. Of the two patients studied here, subject M is the most relevant, and subject T is more like a complement.

General excitability

4 Electrical excitability

4.1 Strength-duration curve (Hoorweg's law)

Of the two main ways of determining the state of elementary excitability in the visual system, electrical stimulation and luminous stimulation, we shall first deal with the former, which provides more important and accurate data.

Excitability behavior for the most elementary response function in both types of stimulation is the starting point for the study of more complex and differentiated functions.

In the subjects we study, we can get a very precise idea of the physiological state of the visual system by electrically stimulating the retina. The simplest and most elementary of all manifestations is the strength-duration curve, which we shall examine first. Next, we shall study the very special phenomena concerning facilitation and iteration. This set of quantitative determinations shows with great accuracy the degree of nervous excitability disturbance in the central syndrome.

Of the two procedures to determine the strength-duration curve, the one of Weiss (1901) by means of a ballistic rheotome to obtain a direct measurement of time, and the other, of Hoorweg (1892) by capacitor discharge, we utilize the latter in all our tests, employing the usual device of Bourguignon (1923) for chronaxie determinations. As is well known, time is measured in this method by the discharge duration of the capacitor used; the greater the capacitance of the capacitor, the longer the discharge duration. Thus, the strength-duration curve is replaced by that of voltage-capacitance. The active electrode or cathode is applied to the upper eyelid and the anode to any limb. The active electrode, which ends in a small sphere of 5 mm in diameter, should be applied gently on the upper eyelid to avoid the production of photomas that could hinder the perception of minimum luminosity, which is used as a test when the electric current is acting. In order to have a higher voltage, the final resistance of the circuit is reduced to 3.000 ohms instead of the usual 10.000 or 20.000 ohms, so we will refer to the capacitance in microfarads, without converting them into time units. This is not a disadvantage, and instead measurements can be made that would otherwise be very restricted, given the excitability conditions in the subjects studied.

Instead of restricting ourselves to finding only the rheobase and chronaxie capacitance, a series of values are determined to construct the entire excitability curve,

thus showing in the most complete way the state of sensitivity of the sensory system. Given the characteristics of nervous excitability in our subjects, a series of precautions must be taken, without which the result obtained would be completely wrong in terms of the strength-duration law. The three main aspects to be watched are described as follows.

First of all, it is necessary to avoid any possible facilitation phenomenon, which would increase visual excitability and thus hide its true state. To this end, the tests must be performed when the subject is in complete muscular relaxation and free from all activity, i.e. in an inactive state. This condition must be taken into account especially in subject M, who has a great capacity for facilitation.

Secondly, given the extraordinary latent addition (or iterative excitability) capability, each determination using a single stimulus must be suitably distanced from other excitations. Otherwise, the stimulus residues, which fade very slowly, are easily summed and a much lower value of the applied stimulus intensity than that corresponding to a single excitation would be obtained. The stimulations should be at least 30 seconds apart, and sometimes even more, depending on the subjects. Lapicque (1926) has already warned about the considerable errors that can be made in determining rheobase in tissues of slow excitation, as is the case of these subjects. In them it is not indifferent whether the determination is made by increasing the voltage or decreasing it, obtaining in the second case much lower values and thus obtaining a measure not of the characteristic excitability of the prepared state but of the transitory excitability due to a previous operation.

Finally, we must take into account the easy fatigue of these subjects, which raises the necessary stimulation values, constituting another source of errors. For all these reasons, it is understood that electrical measurements require great patience since each point of the curve must be determined by successive approximations.

Table 1. Case M: Rheobase in volts (V) with direct current, and chronaxie in µF.

	Case M, right eye	*Case M, left eye*	*Case T, right eye*
Inactive	Rheobase: 14 V Chronaxie: 3.5 µF	Rheobase: 15.2 V Chronaxie: 3.7 µF	Rheobase: 7.8 V Chronaxie: 1.4 µF
Facilitated by muscular effort	Rheobase: 9.5 V Chronaxie:2.7– 2.9 µF	Rheobase: 10 V Chronaxie: 3–2.9 µF	Rheobase: 7.2 V Chronaxie: 1.2 µF

Beginning with case M, Table 1 shows the values found for both eyes in the excitation by means of capacitors. The sensitivities of both eyes are quite similar although somewhat higher in the right eye. The strength-duration excitability curves for the right eye of the M case, both in inactive state and under maximum facilitation by strong muscular effort, are shown in Fig. 8. The upper curve corresponds to the inactive state (complete muscular relaxation); the middle one corresponds to the state of facilitation by maximum muscular effort, the subject contracting all the musculature with maximum

strength every time an electrical determination is made; and the lowest curve represents the excitability of a normal subject under the same general experimental conditions.

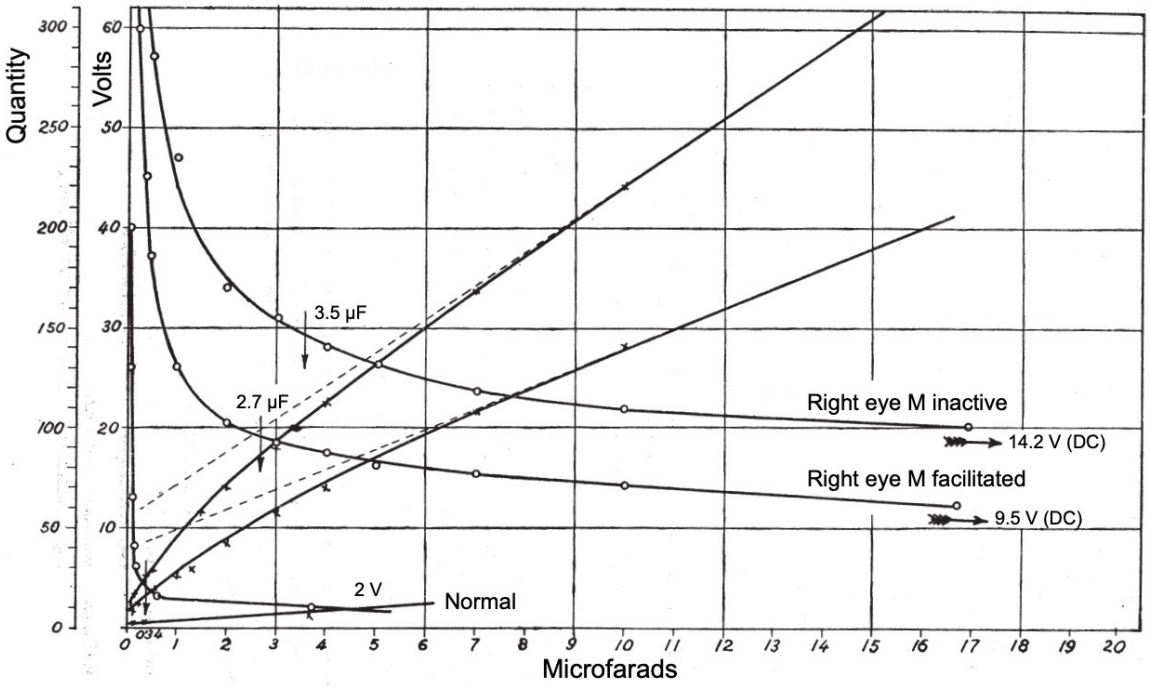


Figure 8. Strength-duration curves according to the Hoorweg' law (volts vs. microfarads) for the right eye of subject M. Upper curve: inactive state. Middle curve: under facilitation by muscular effort. Lowest curve: normal subject. Note the different rheobase values in direct current (DC), and the different chronaxie capacitances (indicated by vertical arrows), as well as the different bend in the strength-duration curves and in the electricity quantity lines (left scale).

The results obtained show the huge difference between the values of the inactive state of subject M and those of the normal subject. In the M case, the two excitability factors, strength and duration of the stimulus, increase considerably and in a more or less parallel manner, since chronaxie is increased by about ten times and slightly less rheobase. The pathological curve also shows a much smaller curvature than that of the normal subject, which corresponds to a lower speed of nervous reaction. This can also be seen in the electricity quantity lines starting near the coordinate origin and crossing the figure. Whereas it is practically a straight line in the normal subject, it has a strong curvature towards the origin in the pathological case.

As for the facilitation curve, i. e. the curve of the excitability state reached under maximum contraction of all the voluntary musculature, it is situated between the curve of the inactive state and that of the normal subject, although much closer to the former. Therefore, facilitation has the effect of bringing the pathological curve of the inactive state closer to the normal one, reducing rheobase and chronaxie values, and consequently increasing the reaction speed. Therefore, the curve has a more pronounced curvature than that of the inactive state, but still shows a very slow reaction speed compared to that of a normal subject.

It is important to indicate that the facilitation permeability, and therefore the rapprochement to the normal values of excitability, presents a limit that is reached relatively soon, the values of the facilitated state still being very distant from the normal values.

Concerning the other subject, the T case, the values of excitability in his right eye, which in this subject is somewhat less excitable than the left eye unlike the previous subject, are shown also in Table 1. The values are considerably lower than those of the previous case, since the parallel increase of the two excitability parameters is approximately four times the normal value, as compared to about ten times for the inactive state of case M. The T case also presents the facilitation phenomenon, but much less pronounced because of his less severe disorder. The facilitation effect is manifested sufficiently so that it can be shown in the electrical excitation tests (Fig. 9); thus, the rheobase voltage drops a little more than half a volt by the action of maximum muscular contraction; this drop is maintained throughout the curve, and being so small, no change in the chronaxie value is indicated in Fig. 9.

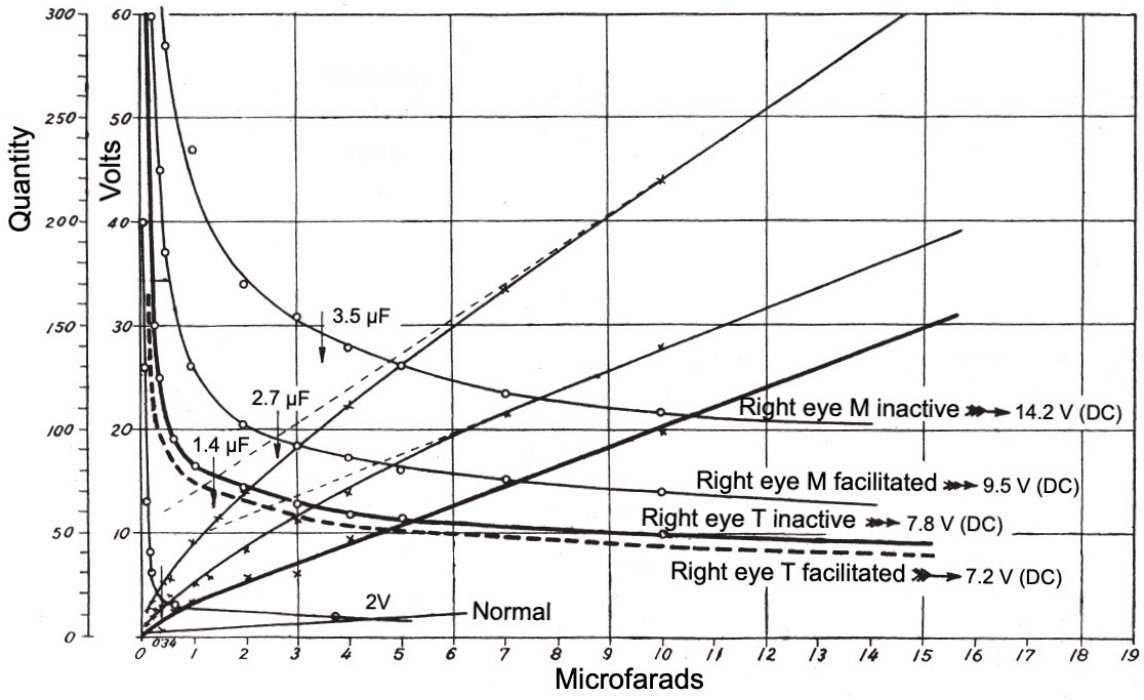


Figure 9. Strength-duration curves according to the Hoorweg' law, as in Fig. 8 but with the T case added: right eye of T inactive (thick solid line); right eye of T under facilitation (thick dashed line) which shows a small reduction of the disturbance. Note the different values and the different curvatures, in particular in the electricity quantity lines (left scale); in the T case, only a small curvature close to the origin.

In this Fig. 9 we can see the state of excitability of subject T compared to that of subject M and that of a normal subject, as well as the very different effect of facilitation by muscular effort in the two studied cases. The closer the strength-duration curve to the normal position, the lower the permeability to facilitation. The small descent of the curve for subject T under facilitation is obtained both by intense muscular contraction and by

very active movements of the head and extremities, and when both types of facilitation are combined, a much more evident descent is obtained. Much more care is needed in subject T than in M to highlight the small difference between the inactive state and the facilitated state in electrical excitation tests. This difference appears much more easily in other experiments, in particular in the phenomenon of inverted vision, which appears only as tilted vision in subject T, and by means of facilitation the tilt varies very clearly.

In the T case, the excitability curve has a more pronounced bend hence the excitability state is quite fast compared to the two states of the M case, especially the inactive state, although when compared to the normal subject, T still offers a rather considerable slow reaction time since the chronaxie is about four times higher. The different curvatures of the electricity quantity lines corresponding to the respective strength--duration curves also characterize the different reaction speed in the cases studied. The greater the curvature of the quantity line, the lower the speed of reaction.

In the excitability disorder we therefore find a functional decrease since there is an increase in the excitability parameters, rheobase and chronaxie, and therefore lower curvature of the voltage-capacitance (strength-duration) curve. Such a decrease is dependent on the amount of brain mass destroyed by the lesion, and constitutes a loss of nervous action of central subordination (Lapicque M 1923; Lapicque L and Lapicque M 1928; Lapicque L 1934). Thus, there are different degrees of excitability disorder which, including the states under facilitation, can be sorted by decreasing disorder as shown in Table 2.

Table 2. Different degrees of excitability disorder.

	Rheobase (V)	*Chronaxie (μF)*
Case M, inactive state	14.2	3.5
Case M, under facilitation	9.5	2.7
Case T, inactive state	7.8	1.4
Case T, under facilitation	7.2	1.2
Normal	2	0.34

This type of alteration of excitability (increase in the required intensity and duration of the stimulus) is also produced both by the action of curare and by cooling, since both agents slow down functional speed. Thus, we find a similarity between the behavior of the excitation curves in our cases and in the case of curare poisoning in rapid response tissues such as striated muscle.

In Fig. 10, adapted from that of Lapicque (1938), it can be seen how curare poisoning modifies muscle excitability by increasing rheobase and shifting chronaxie to long times, the curve showing a much smaller curvature, which makes the quantity line concave downwards. Such a decrease in the speed of reaction is what we find in the central syndrome, and the greater the excluded brain mass the more pronounced the alteration. We also find that facilitation can to some extent compensate for this deficit depending on the case, thus showing a certain equivalence with the action of the lost nervous tissue mass.

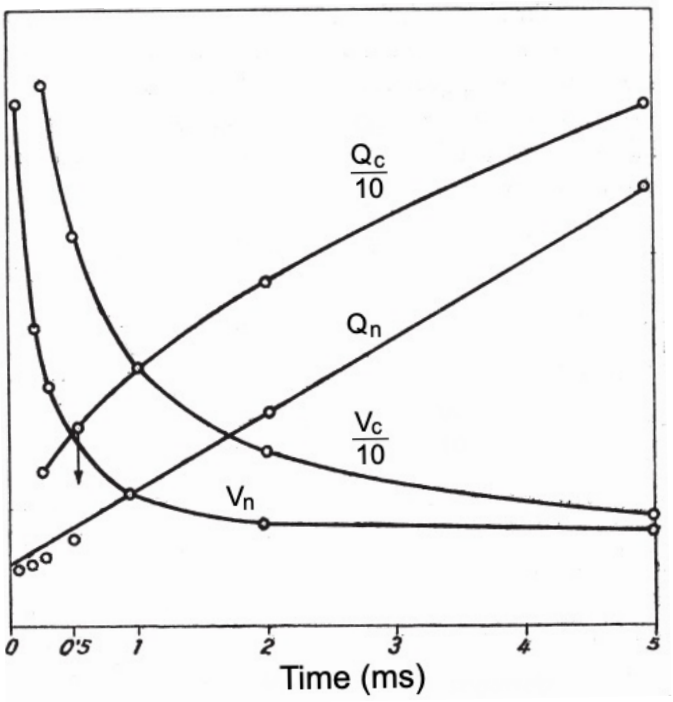


Figure 10. Strength-duration curves for the gastrocnemius striated muscle of a normal frog, and poisoned by curare, according to Lapicque (1938). V_n and Q_n: voltage and quantity curves respectively in the normal state of the muscle. The quantity curve is practically a straight line. V_c and Q_c: voltage and quantity curves respectively for the same muscle after a strong dose of curare. They are divided by 10 to bring the curves closer to the normal curves. The quantity curve Q_c is clearly bent downwards.

4.2 The facilitation phenomenon

From the two curves of subject M in Fig. 9, inactive and under facilitation, it can be seen the saving in the intensity of the stimulus for the transition from the inactive to the facilitated state. Thus, the lower the intensity (voltage), the greater the saving in the duration (in μF) of the stimulus. This saving is very small at high intensities and indefinitely large at low intensities. Facilitation has an extremely different effect in the two cases here studied, and it can be deduced from the curves in the figure indicated that the permeability to this type of summation decreases rapidly when the excitability deficit is lower, that is, when the brain system shows a more favorable physiological level. Considering the reduction in the rheobase threshold by means of facilitation, it can be said that the reduction of the disturbance in the M case is about one third or a little more, and in the T case one tenth or less. However, the values indicated on the previous pages about the facilitated state by maximum muscular effort do not completely show all the permeability to this type of nervous summation, since other types of facilitation are still possible (binocular effect, for example). But in electrical excitation tests, it is not easy to use other types of facilitation different from muscular contraction. We shall now refer

only to this type of facilitation, leaving the study of the simultaneous action of various types of facilitation for other experiments.

Between the two curves of case M (see Fig. 9), that of the inactive state and that of the facilitated state by maximum muscular effort, there would be a series of curves corresponding to different degrees of facilitation (different degrees of muscular effort), which would represent other excitability states of the brain system or, more specifically, of the visual system. A question of great interest can be raised; namely how much excitability is increased by increasing the facilitating stimulus. This question is of fundamental importance in order to know the action of nervous influx on the activity of the centers. Since facilitation can be equated to the action of the nervous mass destroyed by the lesion, the functional action of that nervous mass could be determined indirectly.

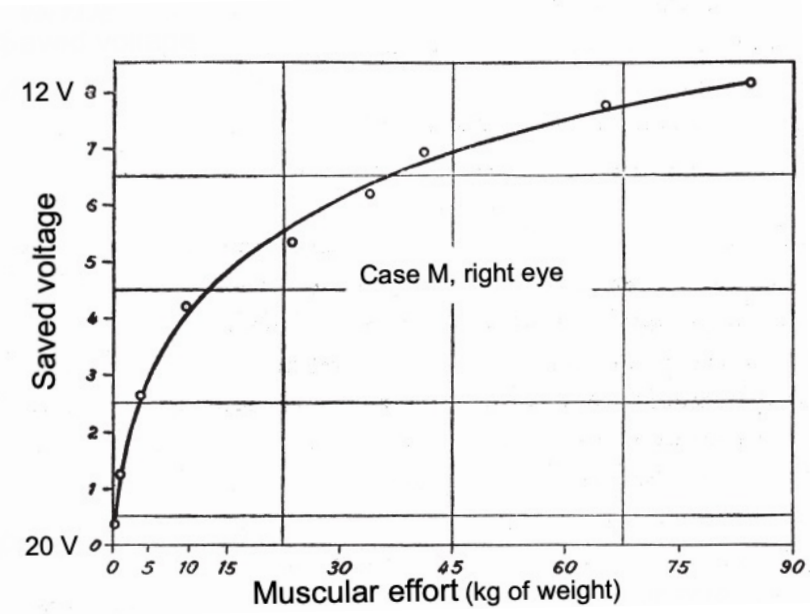


Figure 11. Voltage saved as a function of muscular effort measured in kg of weight held by subject M. Test on the right eye for the transition from the electrical excitability curve of the inactive state to that of the facilitated state, for a fixed stimulus duration (17 µF).

To this end, the voltage saved is determined as a function of the muscular effort made by the subject when changing from the inactive state curve to that of facilitation by maximum muscular effort. The effort made by the subject can be measured quite accurately by having the subject hold known weights. In the standing position, the subject holds a sturdy handle in each hand from which weights of 5 and 10 kg are suspended until a total of about 80 kg is reached, an extremely large amount although necessary for maximum facilitation. The weights in both hands must be properly balanced so that the effort consists only of the action of holding the weights. In addition, care is taken to maintain regularity in all weight tests, the weights always held at the same height. Under these conditions it is not difficult to perform electrical measurements of the retina with a good degree of precision in order to obtain usable results such as those indicated in the curve of Fig. 11, which shows the voltage saved as a function of muscular effort measured in kg of weight held. In this experiment, a slightly higher voltage than that of the rheobase

was taken as the starting point, using the discharge of a capacitor of about 17 microfarads (fixed stimulus duration). Analogously, for a constant stimulus intensity (voltage), the time saved in the duration (capacitance) of the stimulus could be determined as a function of muscular effort, when passing from one curve to the other.

It can be seen in Fig. 11 that there is a rapid growth in saved voltage for very small muscular efforts, and a slower growth in saved voltage as the muscular effort is intensified. It is important to point out that relatively very small muscular efforts in comparison with the total effort that can be made, produce a very considerable economy in the voltage, that is, an increase of excitability (or sensitivity) to stimuli, which explains that in the subject M, small muscular contractions or other muscular actions in his ordinary life greatly facilitate his sensory activity.

Thus, by the muscular action exerted in different positions or activities of the body, the voltage corresponding to a fixed capacitance can show an important reduction in the stimulation of the retina, as can be seen in the results shown in Table 3. We find that the greater the muscular innervation used, the greater the increase in excitability.

Table 3. Voltage required in electrical stimulation of the retina, for a given capacitance, in the right eye of subject M in different positions and activities.

Subject M	*Voltage (V)*
Lying down	19
Standing	18.4
Walking briskly	16.2
Crouching down with incomplete leg flexion	15 - 14.5

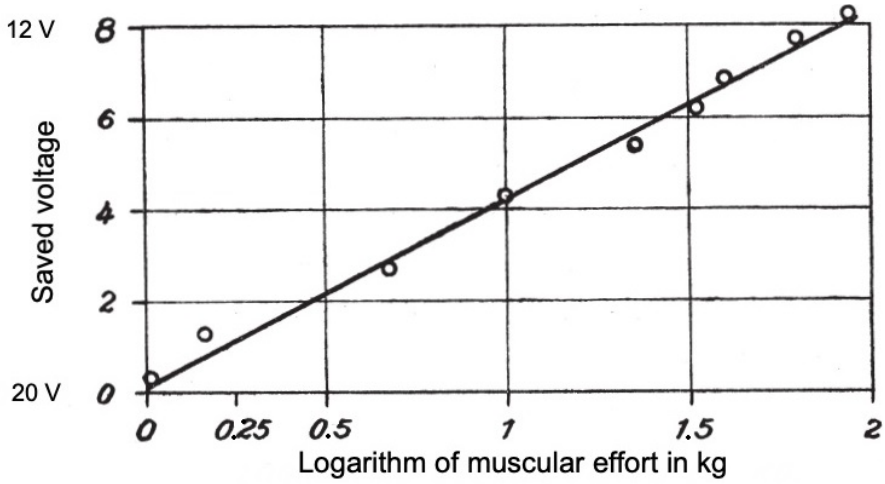


Figure 12. The same experiment as in Fig. 11 but taking the logarithm of the weights held by subject M. The voltage saved (increase in excitability) grows proportionally to the logarithm of muscular effort.

Coming back to the curve of the mentioned experiment (see Fig. 11), if the logarithm of the weights is taken, the points fit well a straight line, as can be seen in Fig. 12. It can

then be stated that the voltage saved, i.e. the increase in excitability (or the increase in the physiological level) is proportional to the logarithm of the muscular effort applied. The nervous energy provided by the proprioceptive receptors of the muscles to the cerebral cortex, modifies the state of excitability by the influx it exerts on the nervous element that determines the elementary response of the visual system (such as the minimal sensation of luminosity evoked by electrical stimulation). This law on the action of facilitation is of great importance for the dynamics of the brain, because if the nervous influx of facilitation can be equated to the influx of nervous centers, the latter could be measured indirectly and thus the level of nervous activity could be determined as a function of the amount of nervous mass. But these are general issues that we shall address later. For the time being, it suffices to mention that this law on the action of facilitation will be also found in the case of other receptors (tactile, auditory, etc.) and also for other types of facilitation different from muscular effort.

The facilitation action we have now studied does not refer to the synchronization experiment exposed when dealing with the dynamic analysis, since now the sensory response is always the same (minimum sensation of luminosity), and facilitation is studied in its capacity to reduce the excitability threshold, i. e. to increase the sensitivity of the system. It is clear that this entails a general action on the entire visual system, and therefore the synchronization of different abnormally asynchronous sensory functions, which become synchronized by different increase in their sensitivities. But all this corresponds to another type of experiments that we shall deal with later.

We have already indicated that facilitation is not an exclusive effect of muscular effort; similarly, any other type of nervous stimulation produces summation effect. Auditory or tactile stimuli can enhance visual activity, although no matter how intense such stimuli are, their effects are still far below the effect of a strong muscular contraction. Examples of summation effects by intersensory facilitation for the visual receptor in the M case, are shown in Table 4.

Table 4. Reduction of the voltage required in electrical stimulation of the retina in the right eye of subject M, by means of different intersensory facilitations.

Whistling strongly next to his ear: from 19 V to 17 – 16.5 V
Weight of 20 kg held by hand: from 19 V to 15 V
Pressing down on his hand until causing pain: from 19 V to 14 V and even less

Finally, we make some considerations about the characteristics of the facilitation phenomenon. Facilitation increases excitability, lowering the Hoorweg curve (Hoorweg, 1892) in which both rheobase and chronaxie diminish, modifying the curvature of the curve and coming closer to the normal curve. The transition from the inactive state of excitability to the facilitated state is carried out, as said, in the proximity of the rheobasic level, and follows a logarithmic relation (saved voltage proportional to the logarithm of the applied muscular effort). Facilitation exerts a central action whereby the fundamental disorder (slowness or excitability deficit and asynchrony) is reduced from the root. The excitability deficit has already been seen in the Hoorweg curve, and more details will be

given when studying iteration. The reduction of asynchrony, which we shall deal with later, is obtained by lowering the dissociated threshold values and unifying them by means of facilitation. Synchronization can then be represented by a curve similar to that of the transit from the inactive state to the facilitated state, studied in this section.

From a strictly physiological point of view, facilitation corresponds to an action of nervous *subordination* by means of the influx of peripheral excitations on nervous centers, partly compensating for the loss of action that the centers normally carry out on the nervous regions that are dynamically subordinated to them. Thus, the exclusion of some nervous centers produces a decrease in excitability, i.e. loss of *central subordination* as was found by Lapicque M. (1923) in laboratory experiments; and now facilitation is offered as a reciprocal effect, i.e. external influx on the centers. These phenomena are studied in humans for the first time, without any precedent as far as we know.

4.3 Iterative excitability or latent addition

The fundamental disorder, with its considerable deficit of excitability, gives way to nervous summations by facilitation (indirect summation) and by iteration (direct summation), the latter consisting of stimulation by rhythmically repeated stimuli with the peculiarity that a single stimulus does not produce any effect but a series of them does. The parameters involved in this type of stimulation are: intensity of the stimulus, i; duration of a single stimulus, t; time interval between two stimuli, ε; and number of stimuli, n; the intensity being a function of t, ε and n, that is, $i = f(t, \varepsilon, n)$, according to Lapicque (1925). This author established two important relationships, the intensity-number relationship (*law of numbers*) and the intensity-interval relationship (*law of intervals*), for given values of the other two variables in each relationship.

As for the technique to determine in our cases the laws of iterative excitability, we use the Lapicque iteration cylinder (Lapicque 1925), which we made build for this purpose. The cylinder consists of a roller that when rotating activates a system of tabs that produce an automatic charge and discharge of the capacitors according to a widely adjustable rhythm in which the time interval and the number of stimuli can be set. The intensity and duration of the stimulus are regulated according to the usual procedure of varying voltage and capacitance of the capacitors, as for the Hoorweg curve. The iterative cylinder is connected or disconnected to the circuit of the capacitors according to the needs of the experiments, thus having one setup for single stimulation and another for iterative stimulation.

Firstly, it should be noted that the normal human retina responds to a single excitation without showing the slightest tendency to latent addition or iteration; however, in the two patients we have studied, the iteration is extremely pronounced and to a different degree depending on the intensity of their lesions. It happens that a non-iterative organ becomes, due to the brain lesion, an iterative organ, although not exclusively since it also responds to a single excitation. The basis of the iteration lies in the sum of residuals of successive

stimuli which fade away more slowly as the iteration capability of the organ increases. The accumulation of residuals thus increases progressively until a new stimulus triggers a response from the organ examined.

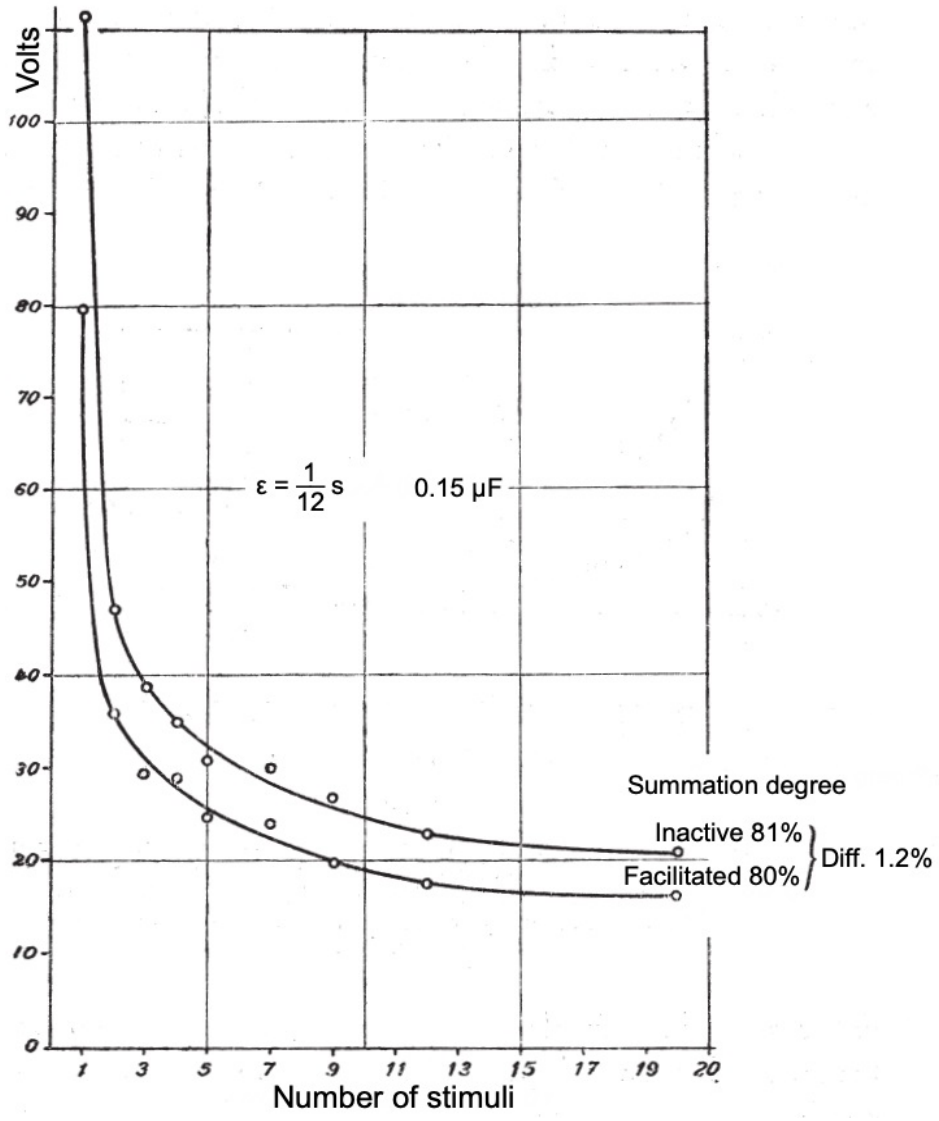


Figure 13. Law of numbers in patient M. Upper curve: inactive state. Lower curve: with facilitation by strong muscular effort. Time interval ε = 1/12 seconds, capacitance 0.15 μF. Corresponding degrees of summation and the difference in percentage are indicated.

Summation depends largely on the time of action of the stimulus, as well as other conditions on time interval and number of stimuli. The shorter the duration of the stimulus, in relation to the nature of the organ being examined, the greater the summation. As for the time interval, the smaller it is, the greater the degree of summation, i.e. the greater the difference between the required intensity of a single stimulus and that of a stimulus from a series of them.

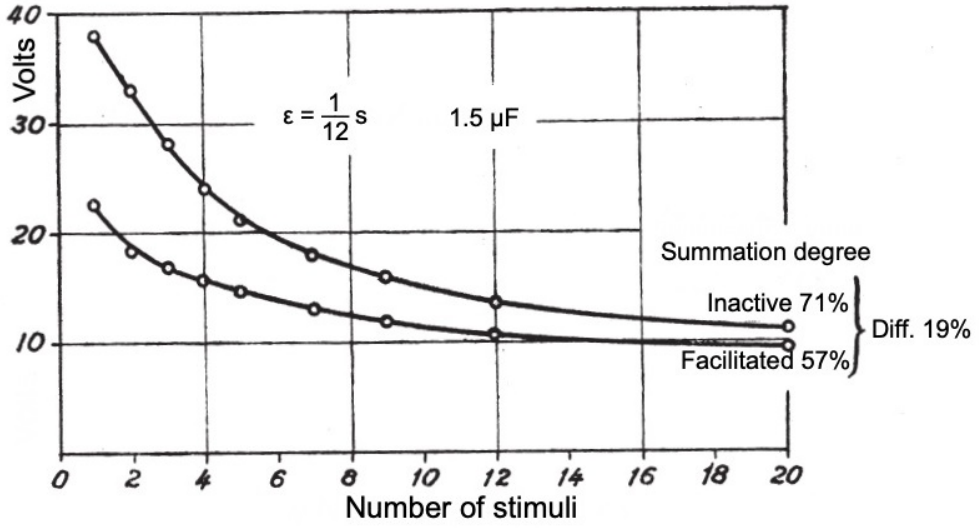


Figure 14. Law of numbers in patient M under the same conditions as the previous Fig.13, but modifying the capacitance, now ten times greater. Note the different slope of the two curves, inactive and facilitated, and the degree of the corresponding summation.

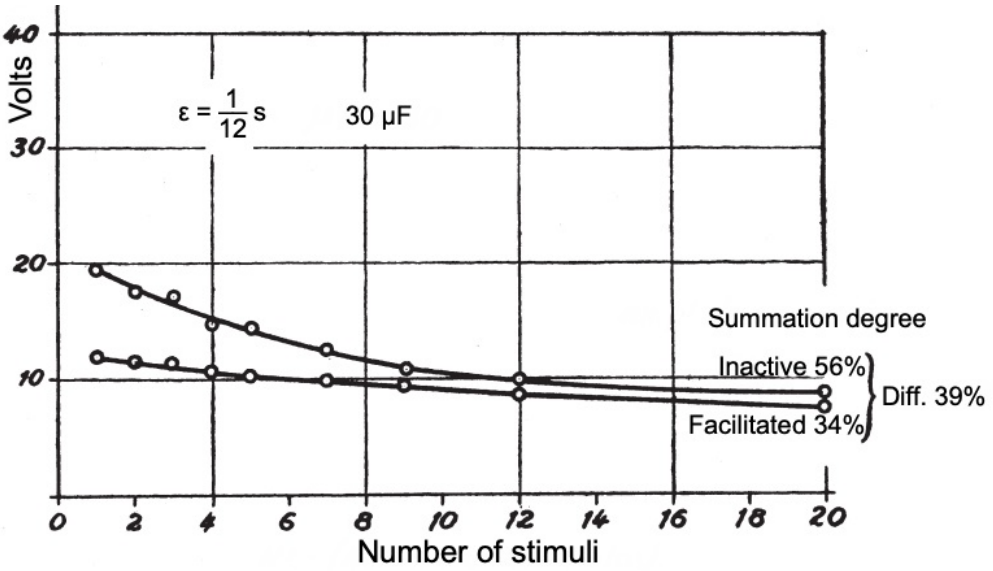


Figure 15. Law of numbers in patiente M using an extremely large capacitance. The difference in iteration between the two curves is maximum.

To find the law of numbers in the case M, the intensity (voltage) of the stimulation is determined as a function of the number of stimuli used, for constant values of stimulus duration (capacitance of the capacitor) and time interval between two stimuli (1/12 second). Figure 13 shows the results of the iteration in case M, for the inactive and the facilitated state. Note the decrease in voltage required as a function of number of stimuli. Using a very small action time (very small capacitance), a high degree of summation is obtained in this case. *Degree of summation* is the difference between the voltage of a single stimulus that produces sensation and the lowest voltage when a series of stimuli

produces sensation, expressed as a percentage. Using stimuli whose duration corresponds to a capacity of 0.15 µF, very high degrees of summation are obtained, both without and with facilitation; the respective values obtained of 81% and 80% indicate that the system is strongly iterative. The proportional difference of summation between both curves is insignificant, 1.2 %. This is because we are dealing with a capacitance for which the two Hoorweg curves, inactive and facilitated, are almost together. This small difference in iteration between the inactive and facilitated states is considerably enlarged by using higher capacitances, 1.5 µF and 30 µF, as shown in Figs. 14 and 15 respectively. By prolonging the duration of the stimulus, the degree of iteration decreases, but the difference in iteration between the inactive state and the facilitated state increases since the Hoorweg curves for these states are quite separate for the capacitance used. In short, it can be said that iteration is very significant and that facilitation reduces it, especially in the range of excitation where facilitation acts most strongly. Note the different slope of the iteration curves without facilitation and with it in Figs. 14 and 15, especially in Fig. 15 where the difference in iteration between the two curves is maximum.

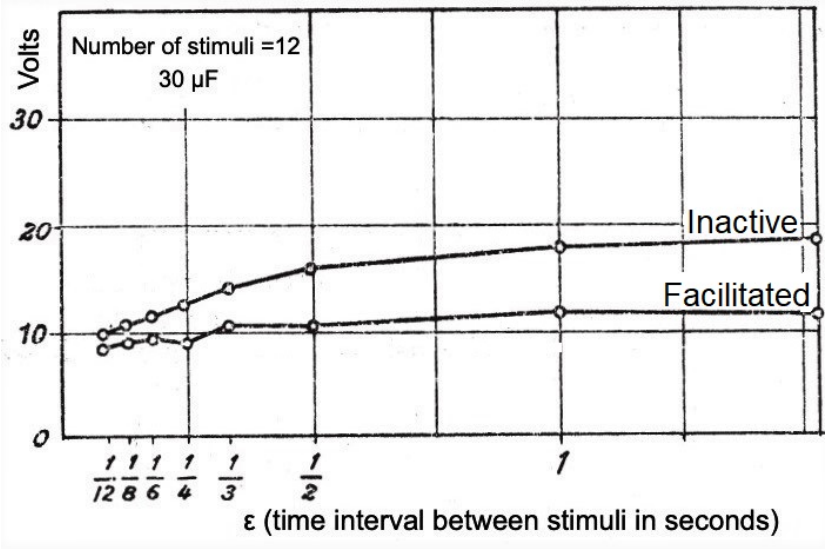


Figure 16. Law of intervals in patient M for the inactive and the facilitated state, using a capacity of 30 µF and 12 stimuli in each determination. The curve of the facilitated state is much more horizontal than that of the inactive state.

Here we also find two different types of degree of summation, greater degree for the inactive state than for the one facilitated by muscular effort. As with strength-duration curves, facilitation reduces the excitability deficit in the iteration curves, thus making the system less iterative. But since facilitation is not able to completely restore the function to normality, a certain capability for iteration is still shown.

The *summation time*, obtained by the product of the number of stimuli employed by the ε interval when the voltage is no longer reduced, is approximately 1.5 seconds for subject M in the inactive state, and perhaps somewhat less in the facilitated state. This is the time in which the latent addition of stimuli can be made, saving maximum voltage.

The other important relationship of iterative excitability is the law of intervals, in which stimulus intensity is a function of the ε interval, with the number of stimuli and their duration being constant. We use 12 stimuli, and a time interval between two stimuli from 1/12 second to more than one second. For the law of intervals, it is now sufficient to indicate the results shown in Fig. 16, for which a capacitance of 30 μF was used, as in the last case of the law of numbers. It can be seen how the voltage decreases as the time interval gets shorter. The more iterative a nervous organ is, the steeper the slope of the curve and the greater the economy in voltage with very short time intervals. Fig. 16 shows rather horizontal curves due to the long duration of the stimulus used. The same considerations made about Fig. 15 (third example of the law of numbers) are valid now since the phenomena are the same under another aspect; with facilitation, the curve is more horizontal than in the inactive state. By using smaller capacitances, results analogous to those mentioned above for the law of numbers are obtained.

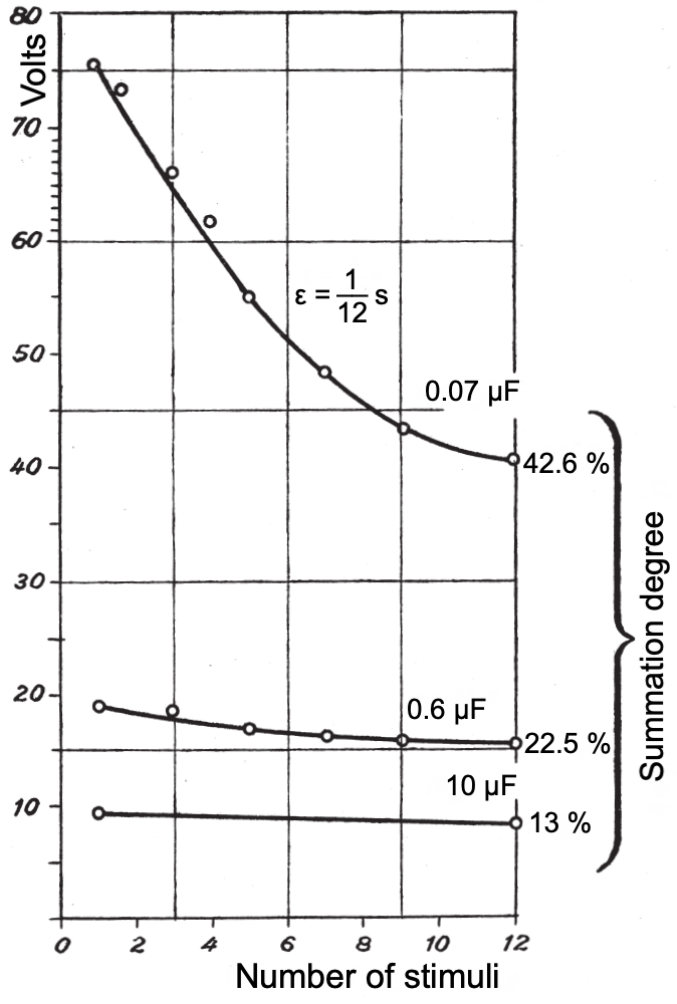


Figure 17. Law of numbers in the T case for three capacitance values (stimulus duration). The value of the interval ε is the same as in the M case. Note the slope of the curves and the corresponding summation degree values.

As for the T case, it is shown that it has a not inconsiderable iteration capability, although much less pronounced than in the M case, as can be expected from the different behavior of their Hoorweg curves. Iteration in the T case is very easy to study and can be determined with much less difficulty and more certainty than the phenomenon of facilitation by muscular effort in this subject. This is because iteration lowers the excitation threshold much more than facilitation by maximum muscular effort, especially if very short stimuli are used in iteration.

Figure 17 shows the behavior of the law of numbers in the T case for three different values of stimulus duration (in μF), and an interval of 1/12 second as in the M case. Under these conditions, the degree of summation varies from 42.6 % for a very small capacitance, to 13 % for a very large capacitance in relation to the characteristics of the excitation time in subject T.

A much lower summation degree is obtained than in the case of M, even when M is under facilitation; thus, T is a much less iterative system. However, whereas in the M case, using an interval of 1/48 second in the law of numbers, no greater summation capability is achieved, in the T case such capability clearly increases when the interval is significantly reduced, as shown in Table 5.

Table 5. Iteration capability in the T case.

Capacitance (μF)	*Voltage needed with a single stimulus*	*Voltage needed with unlimited number of stimuli with interval* $\varepsilon = 1/12$ s	*Voltage needed with indefinite number of stimuli with interval* $\varepsilon = 1/48$ s
10	9.6 V	8.3 V	7.6 V
2	15 V	12 V	9 V
0.6	19.5 V	14 V	11 V
0.07	75 V	39 V	23 V

Thus, using a capacitance of 0.07 μF, the degree of summation can be increased from 42.6 % using a time interval of 1/12 s to 66 % using a time interval of 1/48 s, and analogously for the other capacitance values shown in Table 5. But even so, the degree of summation remains well below than that of M, even though M is under facilitation by maximum muscular effort.

Regarding the law of intervals, subject T presents a behavior similar to that shown in the law of numbers, so we shall not insist on it. As for the summation time in subject T, it is a little less than one second.

In summary, if we try to characterize as a whole the emergence of iterative excitability of an organ such as the retina, which does not have it at all in a normal situation, we can say that the greater the deficit in nervous excitability (the slower the system, i. e. the greater its chronaxie), the more pronounced the capability for iteration. Considering the three main types of excitability, two from the M case (in the inactive state and under facilitation by maximum muscular effort) and from the T case, different

capabilities for iteration are obtained, corresponding respectively to the three strength-duration excitability curves with a single stimulus (Fig. 9).

Comparing the effect of facilitation with that of iteration, the latter lowers the threshold of the inactive state somewhat more than facilitation by maximum muscular contraction. As said, facilitation, by its central summation action, reduces the degree of summation by iteration since it makes the system to react faster. Once facilitation by muscular effort has acted, iteration lowers the threshold voltage very little, and vice versa, once a series of stimuli have acted up to reach the lower voltage limit, facilitation lowers the threshold weakly. Both types of summation can act simultaneously but the final effect is not the arithmetic sum of the action of the two types separately but much less, since facilitation modifies iteration capability.

The permeability to these two types of summation, iteration and facilitation, the first being direct and the second indirect, depends entirely on the slowness in the reaction of the brain system, as has just been said for iteration, and in previous pages for facilitation, although their respective modes of action respond to very different mechanisms. As for iteration, we must point out that its occurrence is a consequence of the loss of reaction speed in nervous organs or in any other tissue. Striated muscle tissue, which normally does not show any iteration, when it becomes slower due to incomplete curare intoxication (see Fig. 10), offers a very important iteration. The slower the reaction of an organ, the slower the disappearance of the electrical modification produced by a stimulus. This results in the accumulation of the residual action of closely spaced stimuli, an accumulation that grows up to a certain limit.

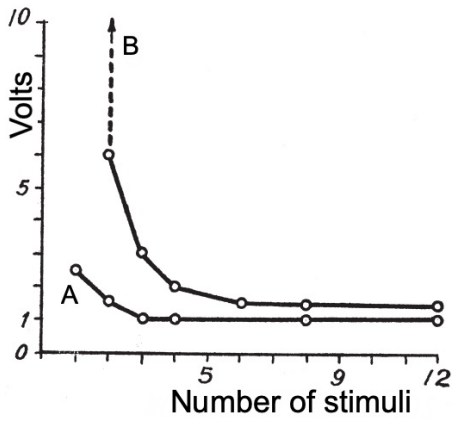


Figure 18. Law of numbers in the hemispherectomized frog (A) and in the spinal frog (B) for the reflex of crossed shortening of the lower extremity. Excitation of the open sciatic nerve using rhythmic capacitor discharges. Constant capacitance of the order of the chronaxie capacitance of the sensitive fibers of the sciatic nerve. Time interval between stimulus: 1/12 s. (Schriever 1933).

We know that in our two cases, the excitability deficit must be put in relation to the loss of brain mass, and in this respect, the experiments of Schriever (1933) (see Figs. 18 and 19) are very illustrative. The crossed reflex of shortening of the frog's lower limb, which normally responds to a single stimulus and does not present any iteration, becomes an iterative reflex due to the removal of the central nervous mass. The more extensive the

removal of nervous centers, the more pronounced the iterative capability. Thus, if only the cerebral hemispheres are removed, it is still possible to trigger the reflex with a single excitation, as seen in curve A of the law of numbers, although iteration allows it to be obtained with lower voltage. However, if there is an extraction just below the midbrain, i.e. if a spinal frog is prepared, the reflex becomes totally iterative, and the B curves of the laws of numbers and intervals not only have steeper slopes, but also show that a single excitation is not capable by itself of triggering the reflex, no matter how much the stimulation voltage increases.

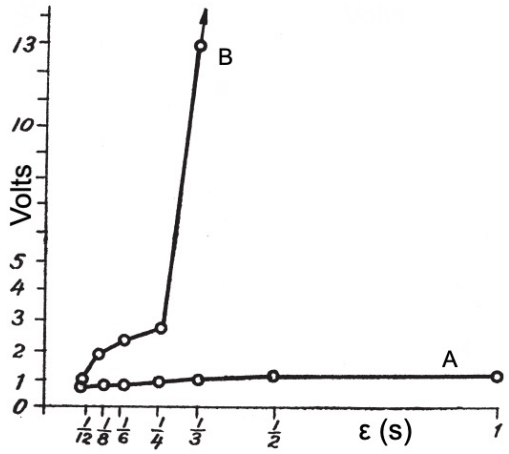


Figure 19. Law of intervals in the frog in accordance with the indications in previous Fig. 18. Constant number of stimuli:12. (Schriever 1933).

In subjects M and T two different degrees of iteration are also obtained in accordance with the amount of brain mass destroyed by the lesion. In addition, another intermediate type of curve is obtained due to the facilitation action in subject M, showing how facilitation can replace the action of the centers. This latter effect offers a wide field for experimentation, reinforcing the dynamic thesis about the nervous system.

4.4 Conclusions on electrical excitability

In general, it can be said that we are dealing with systems with different excitability degree, i.e. different physiological activity depending both on the amount of nervous mass destroyed by the brain injury and on the action of facilitation. In relation to the physiological level, the results of electrical stimulation of the retina can be grouped as shown in Table 6.

In such systems, the excitability deficit caused by the loss of brain mass is compensated in part by types of summation such as latent addition and facilitation, which do not exist at all in a normal subject and are caused by the slowness in nervous reaction. The greater the excitability deficit, the more pronounced the iteration capability. Iteration acts directly by producing a special mode of stimulation that allows saving stimulus

intensity, and facilitation acts indirectly by increasing the excitability of the nervous centers, i.e. by increasing the reaction speed and making efficient the sub-rheobase stimulus corresponding to the inactive state. At the same time, by increasing the speed of the system, the capacity for latent addition is reduced. There is therefore a margin of permeability to summation which can be filled either by iteration (direct addition of stimuli) or by facilitation (indirect summation on the nervous centers).

Table 6. Results of electrical stimulation of the retina in the M and T cases.

Case M *(severe brain lesion)*		***Case T*** *(mild brain lesion)*
Inactive	*Facilitated by maximum muscular effort*	*Inactive*
Very slow reaction system. Rheobase & chronaxie ~ 10 times larger than normal.	Faster system. Rheobase & chronaxie ~ 4 times larger than normal.	Even faster system. Rheobase & chronaxie 4 times larger than normal.
Very iterative system. 71% summation degree using half chronaxie capacitance.	Less iterative system. 57% summation degree using half chronaxie capacitance of the inactive state.	Much less iterative system. 22.5% summation degree using half chronaxie capacitance.
System very permeable to facilitation, saves 1/3 of rheobase voltage.	Does not admit more facilitation by muscular effort, but it does of any other type.	System very little permeable to facilitation, saves ~ 1/10 of rheobase voltage, by muscular effort and movement.

As for facilitation, in the case of visual excitability, there is a reinforcement of the stimulus on the retina by means of another extra visual stimulus, thus, there is an indirect summation of both stimuli through the nervous centers. If the margin of permeability to summation is almost filled with iteration, then little action is left for facilitation, and vice versa, since the system can be saturated by either type of summation. Therefore, their effects can be considered similar, although the mechanism is not the same.

In conclusion, it can be said that if brain excitability decreases due to loss of brain mass, this deficit is partially compensated by the forms of summation we have discussed. The central mass normally acts by reinforcing or increasing by summation the excitability of the various receptors and sensory systems, that is, by exerting an action of central subordination over them, whereas in the case of summation by facilitation and iteration, it is an inverse case, of peripheral subordination over the nervous centers, i.e. an action of stimuli over the centers. Therefore, for all cases it could be said that excitability

depends on nervous summation. We have already seen in the curves about facilitation by muscular effort (Figs. 11 and 12) how excitability changes by the action of summation. If such a facilitation is similar to the action of summation normally exerted by the nervous centers, then the dependence of excitability on the logarithm of muscular effort (Fig. 12) can be also extended to the action exerted by the amount of brain mass. This means stating a law of primary importance for the dynamics of the brain, as it will be seen in subsequent work.

5 Light excitability

5.1 Excitation with light stimulus

In the same way we have studied excitability as a function of time using electrical stimulation, we can now do so by means of appropriate light stimulation. For this purpose, we use a white test 1 cm in diameter, diversely illuminated, and a simple tachistoscopic shutter mechanism that regulates the exposure time of the stimulus. Due to various circumstances, the results cannot be as accurate as those obtained in electrical stimulation, but they are good enough to determine the state of excitability in the two patients we are studying.

Fig. 20 shows the curves of the minimum light excitation necessary to produce sensation in central vision in subject M in his two extreme states, inactive and under facilitation by maximum muscular effort, and in subject T, together with the curve for a normal subject used as reference. For easier representation of these curves, the luminous intensity values are expressed logarithmically. Several properties can be appreciated in the different curves: different reaction speed according to their concavity, rheobase thresholds that place the curves at different heights, and different useful time (duration of the stimulus of minimum intensity that produces sensation).

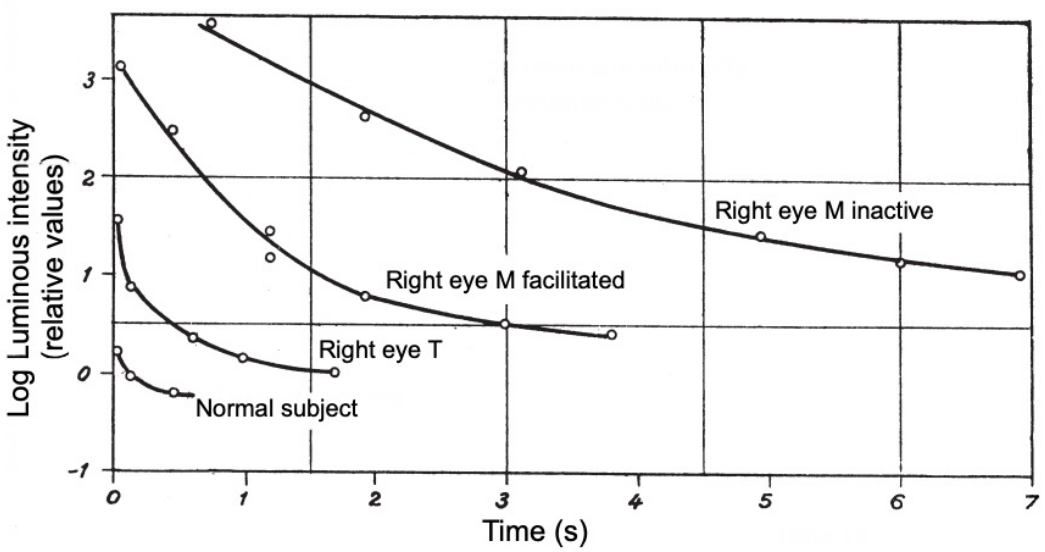


Figure 20. Light excitability curves in central vision of the right eye of: M in the inactive state, M under facilitation by maximum muscular effort, and T in the inactive state; compared to the lowest curve for a normal subject.

Due to the imprecise determination of the rheobase threshold by its normal lability, which is even more pronounced in these pathological cases, and due to the simplicity of the measuring instruments, we can only expect approximate values, yet good enough to distinguish the types of excitability of the cases we are studying. Thus, the useful time for the right eye of subject M in the inactive state can be assessed in approximately six seconds, i.e. the lowest intensity light stimulus that can be detected by the subject requires action for about six seconds. For the same eye but applying facilitation by maximum muscular effort, besides being possible to reduce the intensity of the stimulus to about half, a useful time of about three or four seconds is sufficient.

In the case of T, the rheobase threshold and the useful time are further reduced, this being about 1.6 seconds; and finally, for the normal subject a useful time of about half a second is reached and with a much lower intensity threshold than in the previous cases. These different useful time values show the corresponding reaction speed for each case; the values keep approximately the same proportions as in the chronaxie capacitances in electrical excitation using capacitors. In the curves of case M, a considerable saving in time and intensity is observed in the facilitated state with respect to the inactive state, as in the tests of electrical excitation.

It should be noted that the duration of the stimulus is usually several times longer than that of electrical stimulation. This increase is attributable to the intermediate photochemical process which, when it is developed, triggers the nervous excitation, therefore, this is a more indirect mode of stimulation than that of the electrical stimulation.

As with electrical stimulation, care must be taken to avoid iteration of stimuli, facilitation and fatigue, in order to find the true state as regards light excitability in these subjects, especially in subject M in the inactive state. The importance of facilitation has already been seen in Fig. 20. As for fatigue, it easily arises as soon as experiments are prolonged for a certain period of time, leading to an increase in intensity and time values. As for iteration, the same relationships exist as in the case of electrical stimulation, although in the type of stimulation we are dealing with now they are difficult to evaluate. It is observed that single isolated stimuli that do not produce sensation can trigger it by reiteration of the same stimulus. The difference in intensity required between a single stimulus and several is greater the shorter the duration of the stimulus and the shorter the time interval between them.

The sensation of luminosity develops along different phases whose duration depends on the speed of nervous reaction. In this evolution we distinguish three stages: latency or time elapsed from the initiation of the stimulus until the emergence of sensation, the stage of establishment of the sensation during which it remains stable after having gone through certain fluctuations and, finally, the stage of progressive fading until the sensation disappears. When nervous processes are very slow due to a deficit of excitability, as in the M case, this development of sensation occurs with remarkable slowness; thus latency is very long as well as the fading phase which can reach up to four seconds, especially for weak stimuli.

This slowness in reaction makes both subject T and subject M not perceive rapid medium-intensity light stimuli as light sparks, even when subject M is under facilitation. This subject in the inactive state does not detect even intense electric light from a distance

of a couple of meters and duration less than half a second. The reaction is so slow that the duration of the light stimulus can easily be measured with a stopwatch for most of the excitability curve (see Fig. 20), and equally for any other type of test with colors, shapes, visual image orientation, etc., using the appropriate stimulus.

In the genesis of sensation, both stimulus and receptor intervene. In our subjects, excitation with a single stimulus and iterative excitation must be considered, and in both cases with extremely increased time and intensity, as well as savings in iterative excitation. The receptor conditions the sensation according to the degree of adaptation to the stimulus and according to the sensitivity of the region of the receptor (foveal or peripheral vision). These circumstances are also present in normal individuals but are extremely pronounced in our cases. There are also special circumstances such as phenomena of summation by facilitation which can be of an extra visual type, such as muscular effect, acoustic or tactile stimulation, etc., and of a visual type, such as the effect of one eye on the other eye that is stimulated, binocular vision, light on the eye that is being examined, etc. In short, actions that change the central state.

All these circumstances must be carefully considered in order to properly establish the various tests and to maintain uniformity and constancy in the course of them. Since many factors intervene in the result of the sensation in our subjects, it is possible to perform a very varied experimental analysis which shows the complexity of nervous activity governing sensory function.

To highlight the conditions that intervene in visual sensation (analogous for other receptors) we can group them as shown in Table 7.

Table 7. Stimulus and receptor conditions in visual sensation.

Visual sensation		
Stimulus	*Receptor*	
	Own state	*Summations (central state)*
Single excitation: Intensity and time Iterative excitation: Time interval, etc.	Region of the visual field Light adaptation	Extra visual facilitation: muscular effort, etc. Bi effect: binocular, light over the eye not being examined. Light over the examined eye.

For the curves in Fig. 20, relatively simple conditions have been considered, which are the ones we shall use in general. These are: vision only in the right eye with the other eye closed, application of facilitation by maximum muscular contraction in subject M, or keeping him in an inactive state, although sitting in front of the measuring instruments always implies a small facilitation that can be considered negligible. In addition, unless otherwise indicated, the tests refer to central or macular vision and to the state of

adaptation to darkness, i.e. of maximum sensitivity. The values thus obtained differ significantly from those obtained in binocular vision due to the facilitating effect of one eye on the other. In the latter case, the rheobase threshold can be lowered almost as much as in monocular vision combined with facilitation by maximum muscular effort. In order to obtain a binocular summation effect it is not necessary that both eyes receive the same stimulus (which is the case of maximum summation effect), but a rather similar effect is obtained when one eye receives the stimulus and the other eye (impeded by a screen to see the stimulus) receives a certain amount of light.

These types of conditions will only be systematically analyzed when dealing with inverted vision phenomena, which are very appropriate for all kinds of determinations. For other phenomena, we shall follow the conditions already exposed: vision in the right eye of subject M, in central vision, adapted to a certain darkness, in an inactive state, or under facilitation by maximum muscular contraction. The same applies to subject T, who will be useful mainly as a complement in the experiments.

5.2 Light and dark adaptation

The threshold for perceiving a sensation of light depends on the previous state of adaptation of the visual receptor, i.e. on its degree of sensitivity or capacity to adapt to changes in illumination. In patients M and T and many others with occipital lesions that we have studied, adaptation to darkness is notably diminished. This alteration consists mainly of a great slowness in adaptation and a decrease in the degree of adaptation. Here we shall only expose the tests of subject M, in whom the alteration in adaptation is maximum. This disorder must be related to the deficit of excitability of the centers, i. e. to the slowness in nervous reaction, and not to an alteration of peripheral photochemical mechanisms which must be admitted as intact.

The procedure for studying the adaptation is as follows. First, intense dazzle with a white light lamp of 250 foot candles for two minutes and then determination with the photometer of the minimum intensities of light that can be perceived along half an hour during which the subject remains in darkness and adapts to the minimum intensities. A normal individual under the same experimental conditions serves as a comparison. The measurements are made at first every one or two minutes due to the initial speed of adaptation, and after ten minutes, they are made more separated in time because the change is slower. In these determinations, although central vision is used, the visual field is not limited exclusively to the macular region but slightly exceeds the periphery due to the aperture of the photometer eyepiece. These circumstances are not indifferent since the adaptation curve depends on several factors such as the wavelength of the light, the region and size of the visual field examined, the duration of the dazzle, etc.

Figure 21 shows the dark adaptation curves obtained in subject M in the inactive state and in the normal subject, according to the experimental conditions mentioned above. Between both curves there would be another one corresponding to the facilitated state which has not been drawn for the sake of simplicity. It is observed that the process

of adaptation to darkness lasts a long time and although we have taken the values up to thirty minutes, the process is prolonged for about forty-five minutes although by then the decline is almost insignificant.

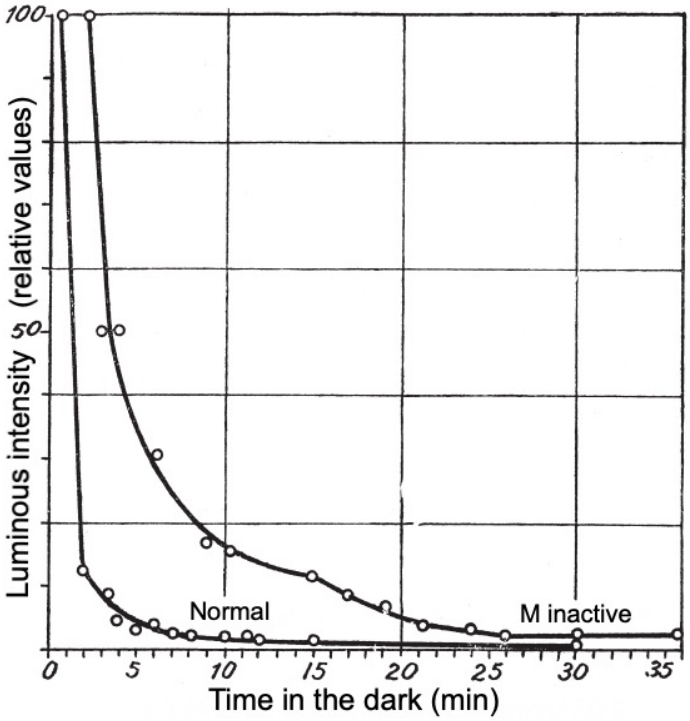


Figure 21. Dark adaptation of the right eye of subject M in the inactive state, and of a normal subject, both dazzled under the same conditions. In the case of M, observe the much slower adaptation and the change of behavior after fifteen minutes.

The process of dark adaptation in the normal subject takes place very quickly in the first two or three minutes, and after five minutes the decrease of the threshold is very considerable. By contrast, adaptation is much slower in subject M, and he takes about fifteen minutes to reach a threshold like that of the normal subject after two minutes. The elevation of the curve in the M case over that of the normal subject, and the smaller curvature, indicate a decrease in the degree and speed of adaptation. The pathological curve also shows a small change in its behavior, around 15 minutes, which means a small variation in the adaptation speed. This change is not seen in the normal curve in this type of representation but it can be appreciated with a logarithmic scale of the intensity as we shall see later.

Another way of expressing dark adaptation is by displaying sensitivity (inverse of luminous intensity threshold) as a function of time in the dark. In this way, the curves of Fig. 21 become those of Fig. 22 showing how sensitivity increases.

Sensitivity increases rapidly in the normal subject and very slowly in subject M. The pathological curve has a smaller slope, which means a slower evolution of the nervous process. The final value reached by the curves indicates the maximum sensitivity. In the

M case, this value (about a quarter of the normal value) is only approximate due to the poor detection of the photometer in very low light.

A third way of expressing dark adaptation measurements is shown in Fig. 23, where the intensity values in Fig. 21 are represented logarithmically. Thus, the phases with different adaptation regimes in both the pathological and the normal curve are easily highlighted.

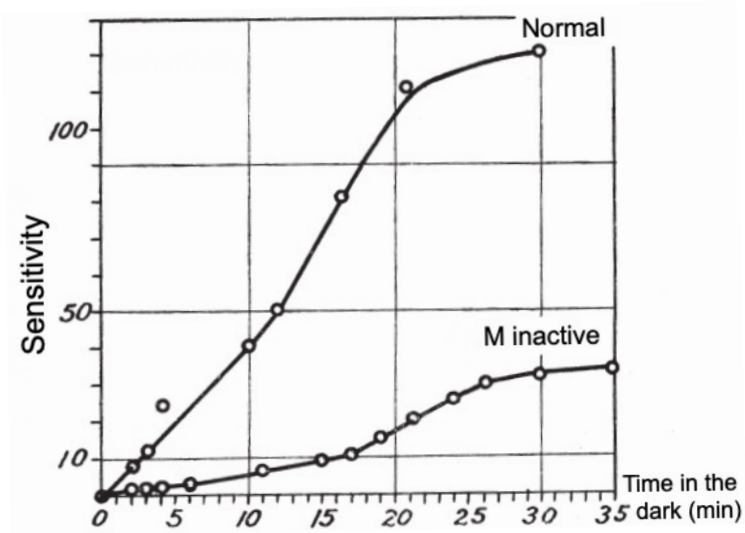


Figure 22. Luminous sensitivity (inverse of luminous threshold) in dark adaptation, for the right eye of a normal subject and of subject M in an inactive state. Note the different slop of the curves and the different values at large times.

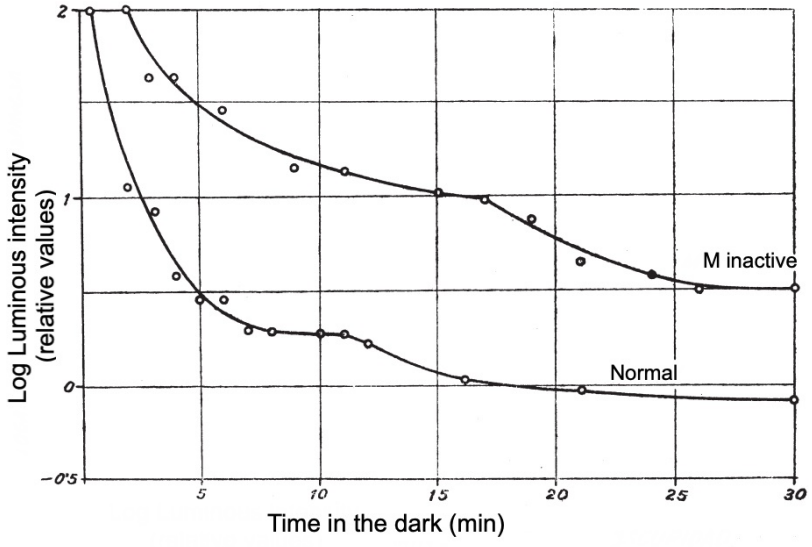


Figure 23. Dark adaptation of the right eye of subject M in the inactive state, and of a normal subject, as in Fig. 21 but taking the logarithm of luminous intensity. The adaptation process is divided into two phases with different adaptation regimes.

In Fig. 21, only a small change in the evolution of the curve can be seen for the M case, whereas in the logarithmic representation (Fig. 23), the two curves clearly show two

regions with different adaptation regimes. In the normal subject, there is a rapid adaptation phase during the first ten minutes and then a slower adaptation phase. In the M case, the change in behavior occurs later, around seventeen minutes, and adaptation in the second phase is faster than in the normal subject.

In the duplicity theory of vision, of cones and rods, this change in the curves is explained as the result of two systems that react differently. The first part of the curve corresponds to cones, which adapt quickly, and this part ends promptly; the second part corresponds to rods, which react slowly and act in low light. When only central vision is used and therefore only cones are involved, dark adaptation is limited to the first part of the curve. In our test, the visual field is very reduced and therefore few elements of the periphery, rods, come into play, this being the cause that the transition from the first part of the curve to the next one is not too pronounced.

The slowness of adaptation in subject M must be explained by the deficit of nervous excitability of the centers and not by the photochemical mechanism of the retina.

Due to their slow adaptation, M and T patients have difficulties in ordinary life to adapt to sudden changes in luminosity. Such a delay makes them appear to be affected by night blindness (nyctalopia), that is, difficulty in adapting to twilight vision, but the curves show that the adaptation is rather good although with much less speed than in the normal individual, so it is a moderate nyctalopia. We have also observed this adaptation deficit in many patients with brain lesions in the visual domain. They show difficulty in the transition from strong to weak illumination, but they are not blind in the dark in a lasting way as is the case in genuine nyctalopia. However, it should be noted that the two subjects we are studying as well as others with an occipital lesion (or near that region) and visual alterations of variable intensity, present, in addition to this slowness and some deficit in the degree of dark adaptation, the peculiarity of feeling better under weak illumination than under strong illumination because of the easy fatigue they experience under the latter. Many of these brain-injured people tend to avoid sunlight because it causes them eye fatigue too soon, and instead they feel very well during the twilight hours. Thus, they show a certain tendency to day blindness (hemeralopia), i.e., better visibility in dim light than in strong light. In all these cases, such disorders have a brain origin and are produced by an excitability deficit, a cause not mentioned so far. However, alterations in adaptation to darkness in purely nervous injuries are not new. In fact, Best (1919) and Igresheimer (1918, 1919) report a slow adaptation in some cases of hemianopic defect, and especially, in the research of Kaltwasser (1920) on subjects with occipital injuries, a diminished adaptation is also indicated.

5.3 Intermittent stimulation. Flicker-fusion frequency

We shall complete the study of light stimulation by determining the behavior of subject M under intermittent stimulation in order to find the visual critical flicker-fusion frequency. For these tests, we use a rotating disc of adjustable speed, activated by a synchronous electric motor; the speed is indicated by a revolution counter. The disc

consists of two equal semicircles, one white, one black, and when it rotates, an alternating sensation of light and darkness is produced. When the speed is increased, a weak oscillation appears corresponding to the flutter point, and when the speed is increased even more, a uniform sensation is established (luminous surface without the least oscillation), which corresponds to the flicker-fusion point.

This critical point depends on the alternating frequency of the phases (white and black) and on the luminous intensity on the disc. The relationship between frequency and luminous intensity is governed by the law of Ferry (1892) and Porter (1902) according to which critical flicker-fusion frequency is proportional to the logarithm of luminous intensity. As in the previous case of dark adaptation, the results depend on the region and size of the stimulated retinal field, and on the nature of the radiation used. The different curves thus obtained are also interpreted according to the duplicity theory of vision, that is, by different action of central cones and peripheral rods.

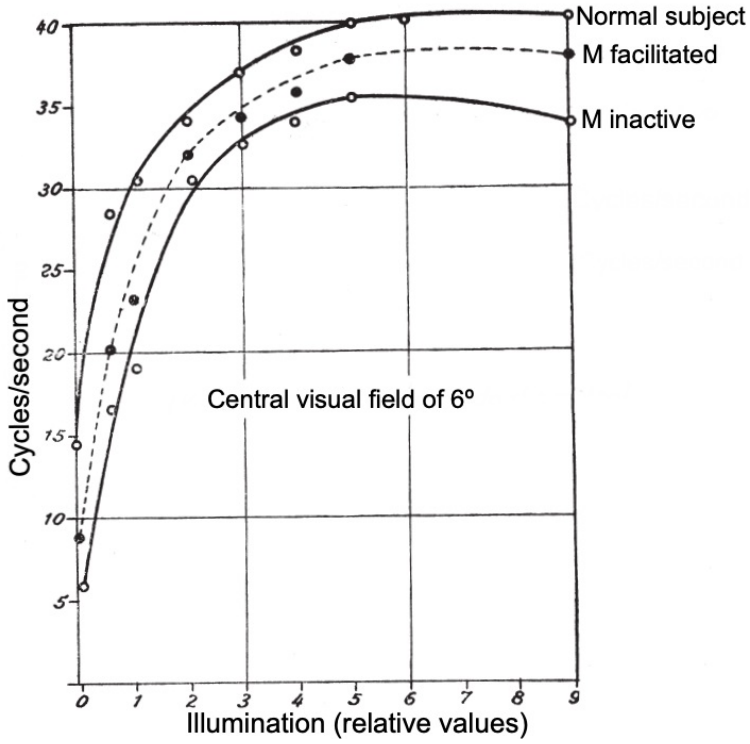


Figure 24. Critical flicker-fusion frequency as a function of illumination, for the right eye of subject M in the inactive state and under facilitation by maximum muscular contraction, and for a normal subject. White light and central visual field of 6 degrees in diameter.

In the tests performed in subject M and in a normal subject for comparison, white light and a central visual field of 6 degrees in diameter are used. The curves obtained for critical flicker-fusion frequency as a function of illumination intensity are shown in Fig. 24. The lower curve corresponds to subject M in the inactive state, the middle curve to M under facilitation, and the upper curve to the normal subject. The ordinate indicates rotation frequency in cycles per second, and the abscissa indicates the luminous intensity on the disk so that the critical flicker-fusion point is obtained. At this point, the weak

oscillation corresponding to the flutter point disappears and a constant uniform surface appears whose brightness is equivalent to the average brightness of stimulation, according to the law of Talbot (1834). Intermittent stimulation, by involving time, allows the kinetic properties of the visual system to be known. The two halves of the disc, white and black, correspond respectively to a succession of light stimulus and absence of stimulus, both phases being of equal duration as the semicircles are identical. Therefore, the intermittent stimulus informs us about the degree of persistence of the stimulus, that is, about the speed of visual reaction, which constitutes the basis of the flutter point and the critical flicker-fusion point. The two curves of the M case are below the normal curve due to his slow reaction time. Given the alteration in excitability, the evolution process of the sensation of luminosity is very slow, leading to an increase in the persistence of sensation and an easy fusion of intermittent stimuli. For a given value of luminous intensity, the fusion is obtained in subject M with a much lower frequency than in the normal subject, and for a given frequency, subject M needs more luminous intensity than the normal subject since the persistence in M decreases with the intensification of the stimulus. The curve corresponding to the facilitated state is in between the normal curve at the top and the curve of the inactive state, below. Thus, facilitation either saves luminous intensity or allows for an increase in the flicker-fusion frequency because it makes the visual system to react faster, since as excitability increases, the persistence of the sensation decreases.

If in the graphic representation we take the logarithm of the luminous intensity, we obtain an almost straight line which is an expression of the law of Porter (1902): critical flicker-fusion frequency is proportional to the logarithm of the luminous intensity. But here we give the intensity values arithmetically because it is more appropriate to the values found experimentally, since at the bottom of the curve there are very few values - because of the device used - to obtain logarithmically a straight line with sufficient accuracy.

Let us note that for a given luminous intensity, when subject M reaches the flicker-fusion point, the normal subject sees a fluttering or a rather strong oscillation of luminosity. But intermittent stimulation, by producing a latent addition of stimuli in M, reduces his excitability deficit significantly and therefore shortens the separation between his curves and that of the normal subject. If there are only two successive light stimuli, subject M does not perceive any interruption or visual oscillation even if the interval reaches two seconds. If the light from a low intensity electric bulb is suddenly interrupted for one second or less, subject M does not perceive any oscillation of luminosity, even under facilitation, and the same happens to subject T (although to a lesser extent), thus both subjects show a great difference in behavior with respect to the normal subject. Thus, they present a great persistence as a consequence of a slow nervous reaction.

Therefore, visual sensitivity to time discrimination of stimuli is evidenced by the flicker-fusion frequency. There is a deficit in the appreciation of short time intervals since consecutive sensations merge easily whereas the normal subject still has a wide margin to perceive the separation between stimuli. The pathological curves of the M case are similar to the curves of a normal case in peripheral regions of the visual field which show a lower flicker-fusion frequency than in central regions.

As it has been mentioned, the alteration in the flicker-fusion is due to an increase in the persistence caused by the excitability deficit; therefore, it has a central nervous origin, as does the alteration in dark adaptation and also the alteration of the strength-duration curve in light stimulation, and should not be considered as alterations of the photochemical mechanism of the receptor. In this regard, it should be noted that in contrast to the photochemical theory of these phenomena developed by Hecht and Shlaer (1935), other authors are in favor of a purely central nervous mechanism. Thus, according to Brecher (1932), the flutter rhythm perceived as a vibration, and which is independent of intensity, is 18 c/s and has the same value for the different sensory domains (visual, tactile, acoustic), depending exclusively on the characteristic refractory period of the nervous centers. In the same direction is the research of Bartley and Bishop (1933) in the rabbit. He establishes, from experiments on the retarded retinal potentials when the eye is intermittently illuminated, a basis for a clearer explanation involving mainly the nervous centers.

With the exposition of all these tests about light excitability (strength-duration, dark adaptation and fusion frequency) we conclude the study on general behavior under adequate stimulation to generate sensation, and we shall now undertake the study of the various visual functions.

Color vision

6 Alteration of the chromatic spectrum

6.1 Color vision research

The chromatic disorder that we study here presents very profound alterations, especially in subject M, on whom we shall focus most of this study, the other subject T being a complement in certain issues.

In addition to color disorder per se, i.e. all that has to do with the important issue of color vision and color blindness, there are other types of disorders such as flat color vision and color agnosia (difficulty in understanding the names of colors), which will be discussed later in other chapters along with disorders of a similar nature.

Since we shall refer directly to the discriminatory activity of brain centers in the face of light, the present study on color function is different from other studies on color vision performed in subjects with retinal abnormalities but with normal nervous centers. Here, the study of color vision is based on the relationships of nervous excitability by means of the dynamic analysis we have applied in previous experiments.

Given the complexity of color alteration, certain methodological precautions must be taken since, in addition to the strictly sensory disorder, there are also perceptual disorders and agnosia. The sensory disorder has a very special character, and changes the appearance of colors; thus, blue and yellow are seen as green (*dyschromatopsia*) and in the same way, white and different grays are seen as green (*chromatopsia*). Together with this strictly sensory disorder, there is the spatial chromatic alteration called *flat color* vision in which colors appear as fogs or colored masses that are detached from (or irradiated by) colored objects. Moreover, color agnosia hinders the understanding of color names, i.e. the abstract concept of colors. It is therefore imperative that all these alterations be taken into account in order to properly design tests and experiments. Thus, for example, the examination of subject M by means of Stilling's pseudo-isochromatic plates (Stilling, 1883) to detect color blindness is not at all suitable since dyschromatopsia and chromatopsia make the examination difficult, and in addition, color irradiation from the plates due to the spatial chromatic disorder makes them impossible to use. Also, due to the agnosic disorder, instead of using the usual color names which correspond to a conceptual abstraction, it is much more appropriate to use terms of a more concrete or immediate character; for example, straw color instead of yellow, or color of the sky instead of blue.

The complexity of this set of alterations and their unstable character depending on the conditions of stimulation have not facilitated color research in these patients, especially in the M case, until the alteration in brain excitability and the corresponding process of chromatic degradation was understood.

At the beginning of the examination of subject M, in the summer of 1938, once his wounds had healed, the state of his chromatic disorder was about the same as now. In general, he saw all objects as greenish although he was able to distinguish red. While walking down the street he came to believe that he had been lost in the city and had arrived in the countryside because he was seeing the ground with a greenish color like that of the grass. Seeing white and gray tones as green is what constitutes green chromatopsia, i.e. colored vision of objects that normally have neutral or white color. This occurs in violet-blind people, called *tritanopes*. It is an already known phenomenon although observed in just a few rare occasions. Although subject M is violet-blind, his color alteration is much more extensive. The considerable decrease in chromatic threshold even in the colors he best perceives as red and green, establishes a general chromatic degradation with predominance in certain regions of the spectrum. This decrease in color threshold depends on the type of sensory activity, so the result varies according to luminous intensity, facilitation, etc. Also, latency and persistence of colors must be carefully taken into account during the experiments performed in order to obtain useful results.

In the case of M, the chromatic deficit is considerably improved by a very intense illumination of colors, although the characteristic disturbances do not completely disappear. However, under facilitation by maximum muscular effort and medium illumination, color vision is normal. It is thus crucial the facilitation through intense muscular contraction of the whole body, thanks to which the subject instantly recovers normal color vision and is able to distinguish both the so-called primary colors and the subtlest color shades of any kind, chromatopsia disappears and white and gray recover their corresponding hues. Therefore, by changing facilitation intensity or stimulus intensity, the chromatic activity can be analyzed in a very diverse way.

The T case seems to show negligible disturbances in color vision, thus, in medium or low illumination, he perceives the yellow-blue pair with some difficulty, confuses blue with green very often and is rather uncertain in naming colors. It seems that he does not present greenish chromatopsia, but a careful examination of this phenomenon shows that when a white sheet of paper is moved far enough away, there is a tendency for the margins to be seen with small pale green spots. In the same way, yellow and blue colors tend to be perceived as green. But even in near vision, this phenomenon of greenish chromatopsia can easily occur in subject T by illuminating a white paper for an instant while the subject is in darkness. In this case, the paper appears full of green spots on a white background. This phenomenon is only noticeable for the right eye whose vision is weaker than that in the left eye. If the illumination, instead of being for an instant, is prolonged for a while, the greenish spots on white background are only perceived during the first instants, fading very quickly. This process indicates that the development of white color sensation in the T case shows a slowness that allows the observation of certain partial phases. In the M case under ordinary conditions, these phases are not completely overcome and give rise to greenish chromatopsia. Similarly, subject M, who might seem to have completely

normal color vision under facilitation, if he is examined in the facilitated state in conditions similar to those of subject T (very brief stimulation), also presents greenish chromatopsia and somewhat more pronounced than in T. We already know that no matter how intense facilitation is, it is not capable of completely restoring the normal function, and both subject M under facilitation and subject T inactive show a certain slowness in reaction time giving place to some functional lag or asynchrony which must be conveniently searched for in order to make it evident.

The Schneider case of Goldstein and Gelb (1918), which would correspond to an intermediate degree of disorder between the two cases studied here, does not seem to have had any color vision disorder, according to these authors. However, we believe that their case, examined following the principles and methods here exposed, would show much more significant disorders than the T case and very similar to those of the M case under facilitation.

6.2 Perception of the color spectrum

By studying the perception of the color spectrum in the M case by means of a monochromator spectroscope, color vision in his right eye has been determined for different luminous intensities, in the inactive state and under facilitation by strong muscular effort.

By using the minimum luminous intensity under which the right eye of subject M in the inactive state can perceive color, the color spectrum he perceives is then reduced to red and green: red is seen from a wavelength of 680 nanometers (nm) up to 605 nm where green appears, and extends up to approximately 540 nm. This means that under such conditions, the perceived red corresponds almost entirely to the normal orange region, and the perceived green includes entirely the normal yellow region. The remainder of the spectrum does not provide any color sensation. This first result indicates that red and green are the colors for which our subject is most sensitive, and that he has a special dyschromatopsic disorder since the colors seen do not correspond to their place in the spectrum, but are the result of a chromatic alteration and degradation of other colors. There are therefore no neutral regions as often described in color blindness; thus, yellow, to which the subject is blind in these conditions, suffers the indicated degradation or dyschromatopsia instead of showing a colorless region in the spectrum. It should be noted that the colors for which a normal subject in these conditions has more sensitivity are yellow and blue-green.

In a medium illumination intensity, the perceived red ranges from 750 nm to 595 nm where green appears, and green extends up to 435 nm. Thus, red and green expand their regions considerably, the first covering the normal red and orange regions, the second covering the normal regions of yellow, green and part of the blue region (see Fig. 25).
Finally, in very strong illumination, the spectrum expands a little more towards the ends; colors become much brighter and saturated and new hues appear, although we are still far from the normal aspect. In the normal spectral region of red, in addition to red, a faint orange hue is seen. In the normal region of yellow, a pale green region appears splashed

with paler green and even yellowish spots that show a tendency to generate yellow. Likewise, in the normal spectral range of blue, there are small spots or traces of pale blue on a pale greenish background. In the normal range of violet, the subject perceives reddish and greenish spots as stripes. Thus, although red and green in general dominate under very strong illumination, there are traces of the other yellow-blue pair, and violet shows a very special mixture. In this situation, dyschromatopsia and weakness to yellow and blue-violet are very well perceived; the former because of the tendency to a greenish tint in the background; the latter because yellow and blue-violet only appear weakly and in a very reduced region. The mixed coloration of violet becomes green as soon as the illumination decreases a little, and reddish traces disappear.

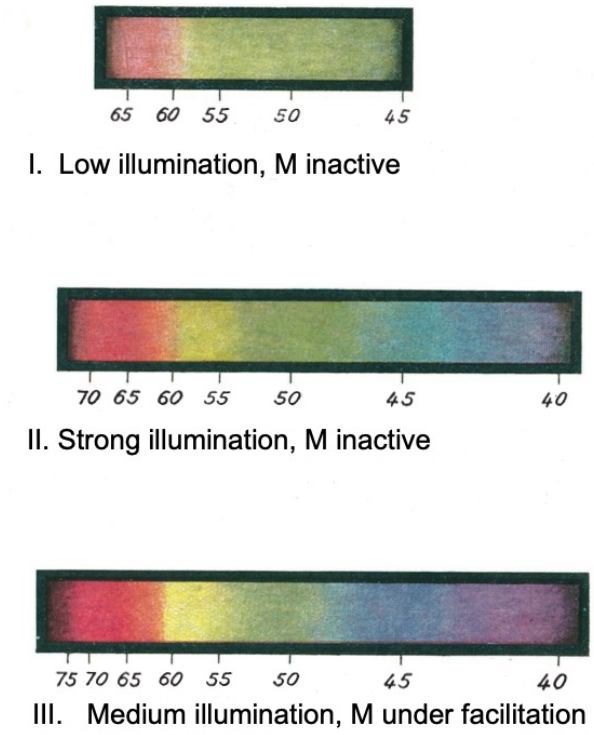


Figure 25. Perception of the color spectrum in subject M, right eye, according to illumination intensity and facilitation.
I: Illumination somewhat low and M inactive: he perceives red and green in the central region of the spectrum; thus, yellow is also seen as green, like the green of its own spectral region. These colors appear as little saturated and not very bright.
II: Strong illumination and M inactive. The perception of the color spectrum expands and now the missing colors in I appear: yellow and blue on a paler green background than the real green; the same happens in the violet region. (Due to the poor reproduction of the image, the greenish background in the yellow and blue regions is hardly appreciated). In addition, red and green look brighter.
III: Ordinary illumination and M under facilitation by strong muscular effort. The perceived spectrum becomes normal and colors show good saturation, thus, it is practically normal vision.

These results show that depending on the illumination used, the subject is blind or almost blind to all spectral colors except red and green, and that the other colors do not

become neutral or colorless, but change to an immediate color that is more stable according to the physiological characteristics of the subject.

In all these tests, the increase of visual field by the enlargement of the viewfinder slit does not seem to change the results.

By means of facilitation, color vision becomes normal, even in medium illumination. Not only does any trace of dyschromatopsia disappear, but colors appear completely normal in terms of brightness, saturation, etc., and red and green are now noticeably enhanced.

Results similar to those obtained with the spectroscope are obtained by other means, for example, using the color circle of Hering (1880) which includes twelve different colors. Subject M in the inactive state only perceives two colors, red and green, each filling one half of that circle. If the illumination is greatly increased, the perception is analogous to that already mentioned in relation to the spectrum: a greater number of hues can be distinguished. With facilitation by strong muscular effort, vision becomes normal, the twelve colors of the circle are perfectly distinguished, and the brightness of the colors already perceived in the inactive state increases. Thus, as said above, the chromatic disorder extends to all colors and is very pronounced for some of them.

When examining color vision in subject M by means of different colored pieces of paper in very near vision and in the inactive state, it is observed that, as the illumination increases strongly, yellow, blue and white acquire more and more their true tones tending to dominate in extension over a green background, even though this background is not completely erased no matter how much the illumination is increased. Conversely, when the illumination decreases, the true tones of the mentioned colors weaken, reduce in extension and a greenish tone tends to dominate. Violet, even in very strong illumination and near vision, never dominates over the altered background, therefore, violet is the color with the greatest deficit in the chromatic disorder of subject M.

Experiments with color mixtures provide results fully consistent with what has been exposed. The behavior of the violet in its transition to purple by the mixture of red and violet, is remarkable. Violet and purple are seen as green or red, depending on their spectral conditions and the degree of illumination. In ordinary illumination, violet is seen as green and purple as red; in strong illumination, violet has green and red stripes, the green strips being dominant; and purple also has green and red stripes but red stripes are dominant. Under facilitation, each color goes to its true tone and the strips disappear. By mixing on a screen light rays of the three primary colors, red, green and violet, in such a way that they partially overlap each other, it results that, from the seven different colors presented (red, yellow, green, blue, purple, violet and white), subject M in the inactive state can only distinguish two dominant colors, red and green that include all the others, as in the Hering circle. By increasing illumination, the subject sees red and various green tones with different intensities and traces of yellow and blue. By means of facilitation by muscular effort, all colors acquire their normal tone, the seven tones formed are perfectly distinguished, and colors that were already perceived well (red and green) increase their saturation and brightness losing the dirty and pale aspect with which they are usually perceived by the subject. The primary colors, red, green and violet, are then distinguished, as well as the colors resulting from the different combinations, i.e. yellow by a mixture

of red and green; blue by confluence of green and violet; and white by a mixture of red, green and violet (the three primary colors).

All these tests show that subject M presents a chromatic degradation for the whole spectrum, although much more pronounced in certain ranges, which means a different increase in color thresholds, due to the excitability disorder. Color discrimination increases as excitability is improved by intensification of the visual stimulus and also by facilitation. Thus, red and green, first limited to the central zone of the spectrum, expand towards the extremes of this one, encompassing other diverse tones which appear in increasingly evident traces when the illumination increases, the normal vision being reached only by means of facilitation provided the first moments of sensation have elapsed.

The behavior of white and grays goes in parallel with that perception of the spectrum. A white paper in weak illumination, no matter how big the paper is, appears with a dark green tone that when illumination increases becomes pale green or yellowish green; and finally, in strong illumination, an increasing number of larger and larger whitish spots are seen on a pale green background. Only by means of facilitation color vision becomes normal. The series of grays and even black behave in a similar way and present a more or less dark greenish tint; black always contains a certain amount of albedo, thus, it also presents a more or less dark greenish tint. Likewise, mirrors and objects with metallic shine are seen with a greenish tone of varying intensity depending on the circumstances. The greenish tint in white (greenish chromatopsia) resembles a pale green close to yellow, according to the comparison made by the subject between the color perceived and the twelve colors of Hering's circle.

6.3 Differential color sensitivity

From all that has been said, it can be understood that color discrimination in a subject with the chromatic degradation previously exposed must be extremely reduced, since all his chromatic spectral vision in medium or low illumination is reduced to a couple of colors, due to his dyschromatopsia in colors such as yellow, blue and violet.

By means of the aforementioned monochromator spectroscope, which allows the isolation of colors and the determination of their wavelengths along the entire spectrum, the different color bands perceived by subject M and by a normal subject (as a reference) are determined. Thus, the boundaries of the different color bands, from extreme red to violet, are determined. About 165 different tones and even more can be established along the spectrum, but by following a successive procedure with a single spectroscope, twenty to thirty different bands can be distinguished, taking into account that differentiation increases as the illumination increases.

The results of these differential color sensitivity tests under different degrees of illumination in subject M, and in a normal individual, are shown in Fig. 26. These tests are not at all easy to perform even in a normal subject and for this reason and greater certainty, different luminous intensities are used in the M case in order to ensure the

results. In the figure, the abscissa of a given experimental point indicates the central wavelength of a given color band and the ordinate indicates the difference between the wavelengths of the boundaries of that color, i.e., the spectral width corresponding to that color.

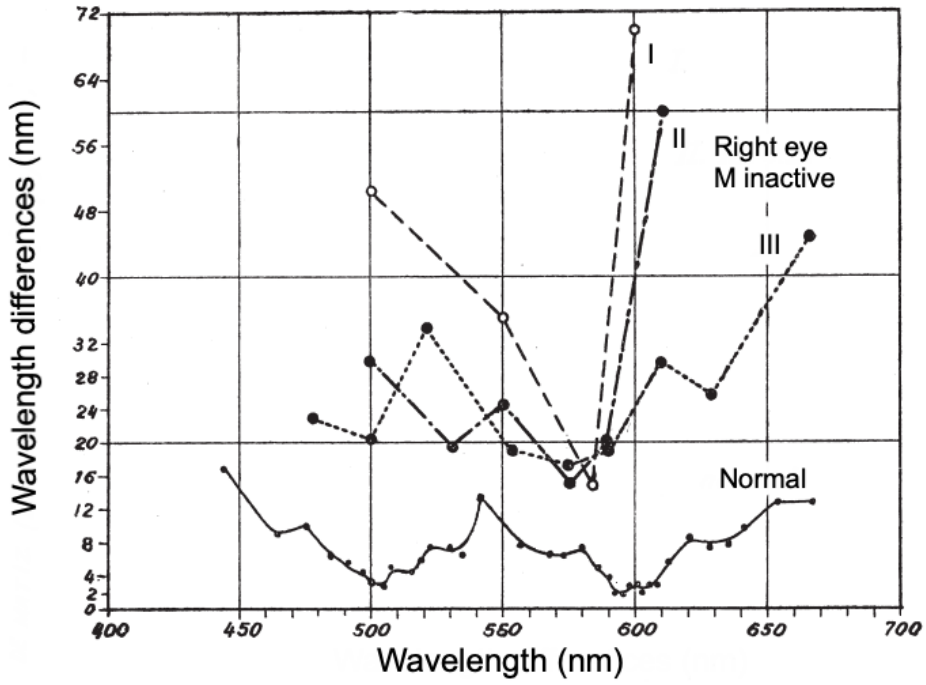


Figure 26. Differential sensitivity to colors. Perceived spectral widths versus color wavelength. The lowest curve refers to the normal subject, the other three refer to the right eye of M inactive, for different luminous intensities of the spectrum: I, low-medium intensity; II, strong intensity; III, very strong intensity. The curve of the normal subject corresponds to an intensity slightly stronger than type I.

In the lowest curve (normal subject) more than thirty hues are perceived whose spectral widths have very different values; widths are smaller (and therefore color changes are more abundant) in the yellow region and the green-blue region, two regions where color discrimination is greater. But instead, subject M using the right eye and in the inactive state shows a very reduced differential sensitivity since he perceives very few colors and with very broad spectral widths. In the normal individual, more than thirty spectral bands of different hues are obtained whereas in M, only five to ten bands depending on the illumination used. Concerning the greatest color discrimination in a normal individual, it only occurs in the region of yellow, where the spectral widths are smaller and therefore the hues are more numerous. This region corresponds to the change from red to green in subject M. This maximum differentiation coincides in the M case for the three types of illumination employed: type I medium-low intensity, type II strong intensity and type III extremely strong intensity. The curve of the normal subject corresponds to an intensity a bit stronger than type I. Differentiation increases in subject M as the illumination increases: the spectral bands become narrower and therefore there are more different spectral bands but, even in maximum illumination, the spectral band

widths in the region of maximum differentiation are approximately ten times the smallest spectral width perceived by the normal subject.

Concerning the other maximum of differentiation in the blue-green region of the normal curve, it is present only in very slight signs in M with very strong illumination. This lack of the other maximum can be interpreted as a result of blindness or strong weakness to the third primary color, blue-violet. Thus, the single maximum is established by chromatic combinations in the transition from red to green, i.e. in the yellow region.

In summary, in the tests to determine the threshold of differentiation of color changes, it is found that the color bands in the M case are too wide and scarce, thus showing a very high threshold, and the obtained curve shows only a single maximum of differential sensitivity in the yellow region.

As regards the distribution of brightness in the spectrum, i.e. the amount of white or resemblance to white shown by the different colors, no major anomalies were observed in the M case in the simple tests performed by the direct method of comparing the brightness of each color with white using a photometer. The characteristic bell-shaped curve shows the maximum brightness in the yellow region, as in the normal subject. Concerning the Purkinje phenomenon (Purkinje 1825), when illumination is reduced, the brightness of the spectrum shifts towards short wavelengths, as in a normal subject. However, more complete and precise quantitative experiments are still needed in this type of phenomenon. It should be noted that at the beginning of the examination of the M case, the Purkinje phenomenon was inverted, i.e. in low light, red seemed brighter than blue. We have also observed a clear trend towards the reversal of the Purkinje phenomenon in other brain-injured people with occipital lesion and visual field disorder.

7 Chromatic dynamics

7.1 Photochromic and photo-heterochromic intervals

From the features previously studied on the perception of the chromatic spectrum, we can get an idea of the nature of the alteration in color vision. Here, we shall expose more accurately the conditions of brain excitability in the M case in relation to the different colors.

When the luminous intensity of a color decreases greatly, it appears as colorless; in fact, any color with very tiny luminosity is seen only as whitish (simple luminosity), and only with greater luminosity, it is chromatic. Thus, there is a colorless interval (*photochromic* interval) between the threshold of simple luminosity and that of color perception. Only with respect to red there is controversy about whether it shows a colorless interval or not; followers of the dualist theory of vision such as Kries (1899), Köning (1894) and others deny it, whereas researchers such as Hering (1874, 1878, 1880) claim it. The photochromic interval is minimal or nil for red and increases progressively as the color wavelength becomes shorter. The fundamental disorder of brain excitability, consisting of slow reaction time, excitability deficit and especially asynchrony, alters the photochromic interval in a very special way. In addition to the increased threshold due to the excitability deficit, the colorless interval expands considerably and becomes very conspicuous even for red. Red and green, although with a photochromic interval larger than normal, do not show any other particularity. Instead, all other colors, including white (although it is not a spectral color in the true sense), show what could be called a *photo-heterochromic* interval, meaning that they go through a different color than their own before reaching their true normal tone. In these colors, the interval between simple luminosity and the color itself is not as simple as in a normal subject and shows an intermediate phase or very singular dyschromatopic alteration that evidences a color decomposition for a certain luminous intensity, giving rise to sensory manifestations (sensory levels) that do not exist in a normal individual.

By determining the strength-duration curves for the different phases of the photochromic and photo-heterochromatic interval, bundles of curves are obtained similarly to what was already described when dealing with the fundamental experiment in Part I (Fig. 5). The curves for these intervals are obtained by the same procedure as for the light excitability curve shown in Fig. 20.

The results obtained for the photochromic interval of red and green are shown in Figs. 27 and 28 respectively. In each figure, the lower curve corresponds to minimum

sensation of luminosity and the upper one to minimum sensation of color. The photochromic interval corresponds to the separation between the two curves. The scales for these two and subsequent figures are the same, taking the logarithm of luminous intensity in the ordinate to facilitate the representation of the curves.

All these tests refer to case M, right eye, the subject being totally inactive without any facilitation, which makes the experiment easier. The same subject under facilitation would give a smaller photochromic interval, i.e. curves closer together, and also lower threshold and faster reaction time. These last features would be even more pronounced in the T case. For a normal individual, the two curves in each of the two colors considered would be extremely close, would have a more pronounced curvature, and the useful time would not exceed half a second at most. Thus, as the brain excitability deficit increases, the curves show a higher rheobase threshold, a longer useful time and the separation between the curve for sensation of minimum luminosity and that for the color in question is greater, i.e. the photochromic interval increases. All these relationships are expected given the different light excitability curves for M inactive, M under facilitation, case T and a normal subject, shown in Fig. 20.

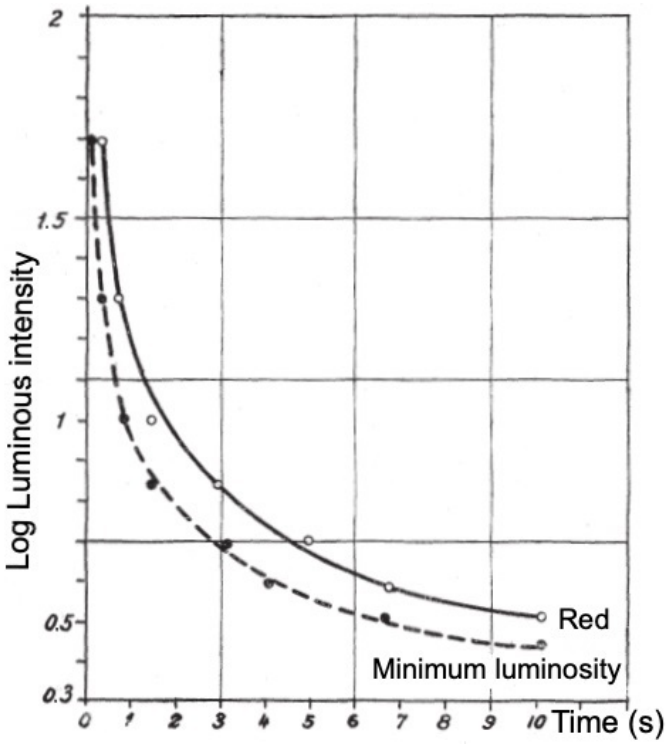


Figure 27. Photochromic interval for red in the M case inactive, right eye. Upper curve: strength-duration curve for minimum color sensation. Lower curve: strength-duration curve for sensation of minimum luminosity.

The photochromic intervals for red and green in the M case are very similar, with the interval for green slightly larger. In all these curves, each experimental point is the mean value of several measurements. The duration of the stimulus results perhaps a little

longer due to the experimental conditions imposed in order to discriminate between the sensation of luminosity and that of color, but this is not relevant.

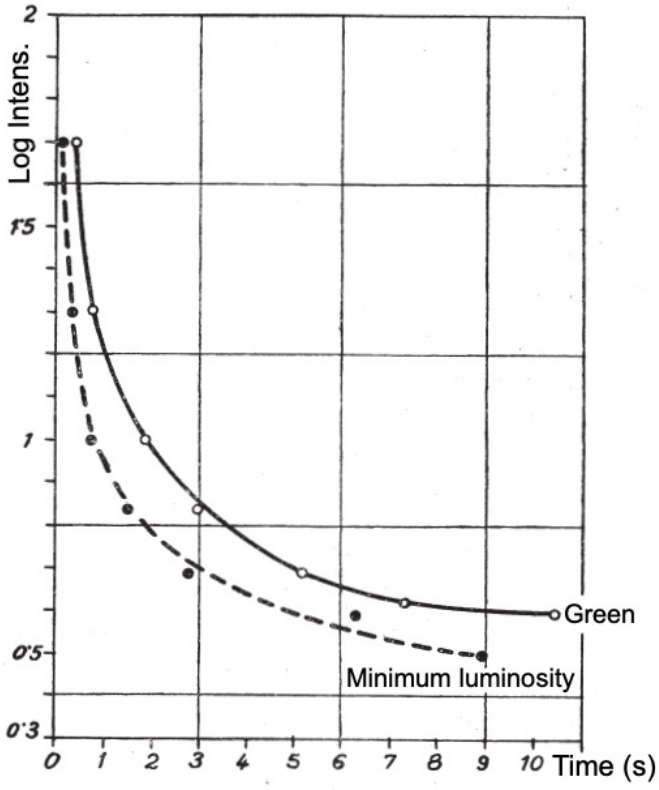


Figure 28. Photochromic interval for green in the same subject M and conditions as in Fig. 27.

Examining the interval in question for yellow, blue and white, under the same conditions as before, more phases appear. In the determination of the corresponding curves, only three sensory levels (phases) will be chosen: the phase of minimum luminosity, the phase of color that appears when the intensity increases and which does not correspond to the color tone we are studying, and finally the appearance of the color itself, which only appears in traces by means of small spots over the color of the preceding phase. Therefore, it is a complex interval that we have called photo-heterochromic to highlight the intermediate phase of altered color.

The phases in the M case for each of the colors mentioned are as follows. Yellow, after having passed the colorless phase, goes on to light green when the luminous intensity increases, and when the intensity increases even more, some yellow stripes are seen over the green background, and finally when the illumination is considerably increased, the area of yellow can dominate over that of green. For blue, there is an analogous process: colorless phase, green phase and phase of blue spots over a green background. Violet color changes from simple luminosity to green, and later shows a tendency to reddish and lilac. Finally, white, after the colorless phase, changes to light green, and later displays white stripes over some green. It should be noted that in the case of M, in the inactive state, no matter how much the light intensity is increased, he cannot perceive colors fully normally except red and green. For white and for the yellow-blue pair, when illuminated

very strongly, the most that can be achieved is to reduce considerably the abnormal greenish hue and that it appears in less quantity than the color under study. In order to reach the completely normal color, facilitation must be applied.

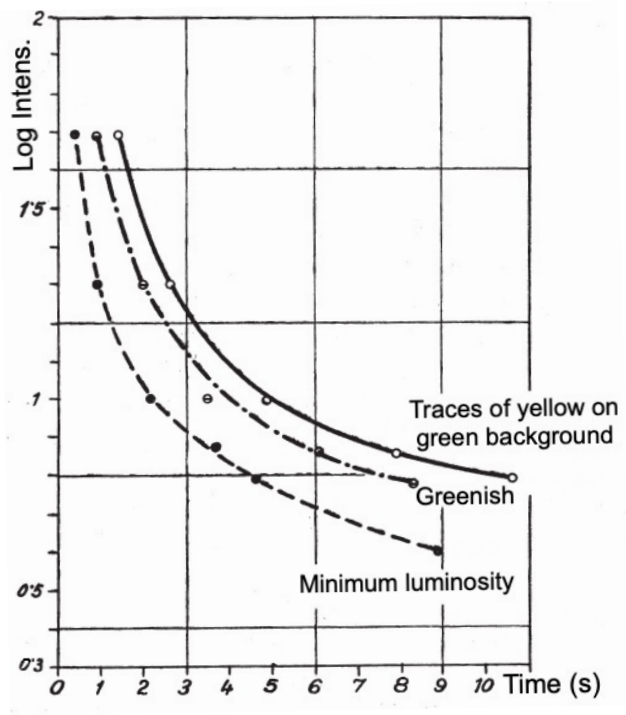


Figure 29. Photo-heterochromic interval for yellow in the same subject and conditions as in Fig. 27.

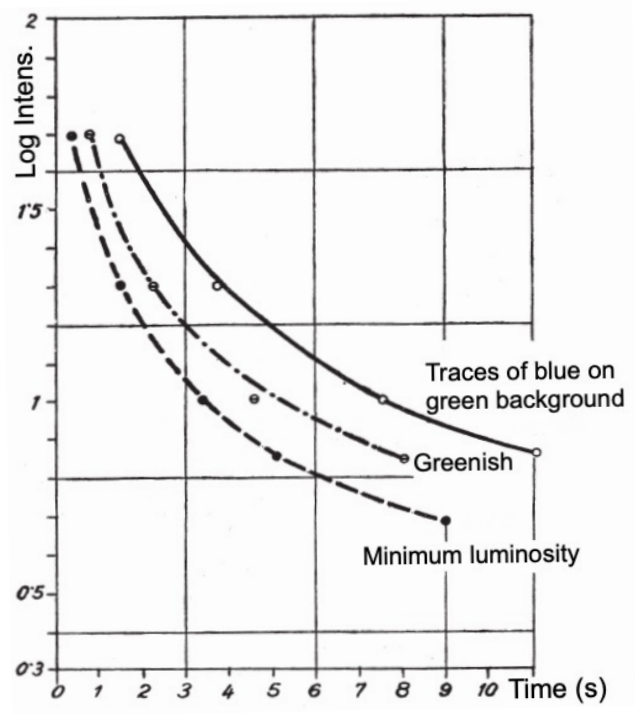


Figure 30. Photo-heterochromic interval for blue in the same subject and conditions as in Fig. 27.

The photo-heterochromic intervals for yellow, blue and white are respectively shown in Figs. 29, 30 and 31. The corresponding curves have, for all the phases, a smaller concavity and a higher rheobase threshold than those for red and green shown in Figs. 27 and 28. Therefore, yellow, blue and white are more difficult to excite, i.e. slower to react. Due to the triple phase that these colors present, the interval between the sensation of minimum luminosity and the color itself, even if only traces, is considerable, and would be even much greater if the threshold for a more complete display of the color itself is reached. Thus, for white it is necessary to increase from fifteen to twenty times the light intensity threshold corresponding to the phase of white traces in order for the green-white ratio to be inverted and white to appear with green traces. If light intensity increases even more, white becomes more pronounced but there are always traces of green. Thus, a fourth curve could be added, which would be placed well above the corresponding curve of traces of color over a green background, and which would indicate an inverse relationship, i. e., the color itself and some traces of green. Even so, this new curve would not be fully similar to those obtained for red and green, since these two colors appear without any mixture.

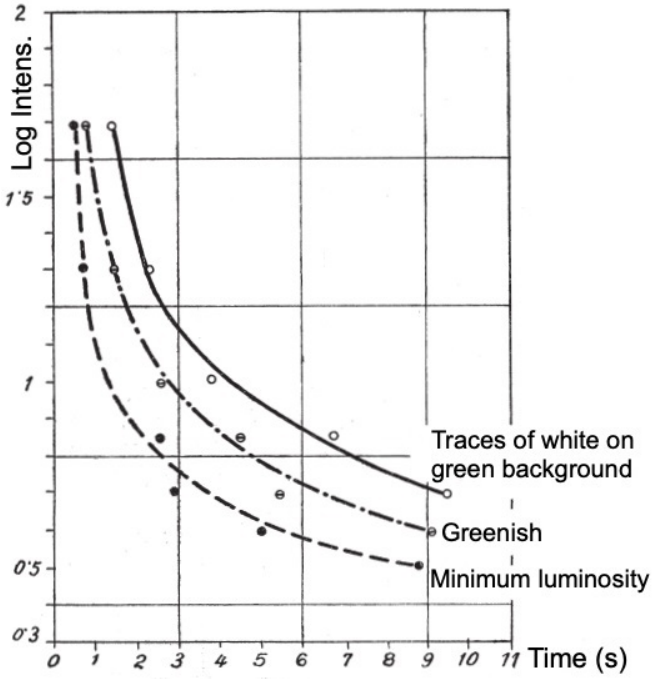


Figure 31. Photo-heterochromic interval for white in the same subject and conditions as in Fig. 27.

By facilitation, the bundle of curves for a photo-heterochromic interval is considerably narrowed, the curves tend to approach the axes and each other, the intermediate dyschromatopsic phase is considerably reduced, and in subject T the synchronization of these phases (out-of-phase functions) is so large that each dyschromatopsic phase is practically impossible to determine because it only appears in minimal signs.

In short, this type of experiment shows that the excitability deficit affects the entire chromatic system, but certain colors are affected more intensely than others. The decrease in brain excitability, thus causing asynchrony, leads to an increase in the colorless interval; the more affected the color, the greater the interval is. Furthermore, due to this asynchrony, a decomposition of certain complex colors into their chromatic components is generated during the photo-heterochromic interval.

In a normal individual there are very small differences in excitability for the various color sensations, and according to Kleitman and Piéron (1925) and Piéron (1932, 1934), colors are sorted according to the speed of perception, red first, green very soon after, and blue shows a much lower speed. Pathologically these differences become considerably more pronounced, affecting in greater proportion the less excitable colors, so blue is one of the most altered colors. Whereas in a normal individual the small differences in color excitability are easily overcome, in subject M the asynchrony is so great that it even tends to exclude colors that are more difficult to be excited, thus reducing the number of hues perceived.

7.2 Dyschromatopsia and chromatopsia. Simple and composite colors

The aforementioned tests on photochromic and photo-heterochromic intervals, and especially the alteration of certain colors when passing through the dyschromatopsic phase, constitute the fundamental experiment in the dynamic analysis of colors. The bundle of curves that can be obtained, especially for colors with photo-heterochromic interval, is just the asynchronous bundle of desynchronized (or partial) functions described in the general part (Part I) when dealing with the fundamental experiment.

It results from these tests that only the red-green pair maintains its true color whereas the other yellow-blue pair and also white show phases whose tones are completely different from their true colors, depending on the degree of illumination. This color change is called dyschromatopsia; thus, blue and yellow are seen as green although of different lightness to that of real green. Also, white in weak illumination is seen as green, and in ordinary and even strong illumination it maintains this greenish shade although paler, turning towards yellowish green or pale green. This case is also a dyschromatopsia, but due to the special character of white, which belongs to the neutral series (grey-white), this alteration of white that produces a colored vision is also called chromatopsia. This is in fact a consequence of dyschromatopsia in certain colors, so all these chromatic alterations result from the same process.

We are therefore faced with a special chromatic degradation that affects all types of colors except red and green; these remain unaltered and evolve directly to the achromatic phase when the intensity of the stimulation decreases. The other colors, for example, yellow and blue (the other chromatic pair), are seen as green in ordinary or medium luminous intensity, that is, they go through a somewhat close hue that must be considered as originated in the disaggregation of the chromatic function by virtue of an asynchrony of normally synchronized (united) functions.

7.2.1 Primary and composite colors

The perception of the spectrum by subject M under ordinary conditions of stimulation is essentially reduced to two unalterable colors: red for the long wavelength region, and green from the yellow region to the shorter wavelength region. The studied chromatic degradation and decomposition would allow us to classify colors in simple and composite colors. Simple colors, of higher excitability, persist; composite colors, of lower excitability, tend to be excluded and replaced by more stable hues resulting from the decomposition and that would participate in the formation of the composite color. If the simple colors red and green are called primary colors, the others can be called composite colors since in their decomposition by asynchrony they change to one of the two primary colors. Composite colors could also be considered as differentiated colors since they are located in spectral regions in between the primary colors, which means an increase in differential color sensitivity.

In the process of degradation, the differential yellow-blue pair is lost and these colors change to the next simpler hue or to certain mixtures, depending on the colors. Thus, yellow and blue become light green, and violet becomes green or red depending on its spectral position. In the case of white, considered the maximum composite color since its formation needs the whole spectrum, a yellowish green or light green color appears as a result of the mixture that is then established. The primary red-green pair subsists but somewhat weakened as we know, losing brightness and saturation.

In our brain-injured people, all kinds of sensory functions experience a special disaggregation due to asynchrony, and the most complex functions are delayed or excluded. Thus, in the case of colors, white is one of the most altered functions due to its most complex character. Even in normal individuals, there are different reaction speeds for different colors, and although the differences are small, they have been accurately determined by Kleitman and Piéron (1925) and Piéron (1932,1934) using a special spectrocolorimeter. In foveal vision, the development of color sensation until a steady state is reached has maximum speed for red, a little lower for green, much lower for blue and even lower for white, as can be seen in Fig. 32.

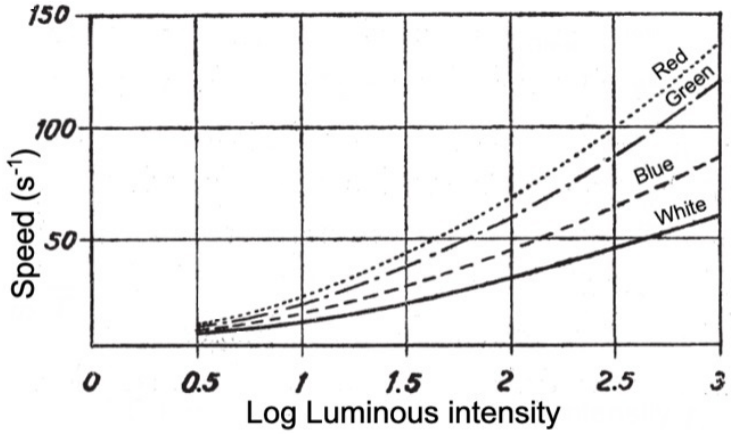


Figure 32. Speed in establishing color sensation as a function of the logarithm of luminous intensity, for different colors in foveal vision. Figure adapted from that of Kleitman and Piéron (1925).

Due to the deficit in brain excitability, these differences increase considerably, the colors of lower speed being the most affected, which tend to be excluded or to appear incomplete even in extremely high stimulation. Thus, the pathological alteration follows the same sequence that exists in the normal subject; red and green are much less deteriorated than blue or white.

If we observe what happens in the recovery from the chromatic disorder in brain-injured people who have become almost blind and slowly recover their vision by regression of the disorder, we find a similar sequence. Although our observations are not sufficiently precise in this matter, we can count on the indications of Economo, Fuchs and Pötzl (1918), Best (1920), etc., who point to red as the first color restored in the recovery from cortical blindness, followed by green and yellow, and much later by blue which may be missing for a long time. Although this recovery has not been possible for us to study it in detail, we can now specify the exact order in which it should be carried out in view of the characteristics of color excitability. For the moment, we shall simply point out that the order in color recovery coincides and perfectly fits the degree of color weakness in our cases.

By means of special tests, a similar order in color disturbance is obtained. Thus, in the experimental intoxication with mescaline in normal subjects, very diverse sensory alterations are shown, and with respect to colors, Beringer (1923, 1927) describes certain phenomena which are very important for the knowledge of the diverse excitability of colors: “The chromatic qualities disappear, the whole space is seen uniformly gray and colorless”. As for the dominant colors, the subject concerned says: “everything looks either green-blue or green-red. I see everywhere only red and green, and I look for blue and yellow”. Mescaline, a drug that reduces brain excitability, therefore produces a chromatic alteration in which the yellow-blue pair is most affected.

Therefore, in all the cases considered of chromatic alteration by modification of the nervous system, such as our brain-injured patients, cases of recovery from cortical blindness and cases of mescaline intoxication, the sequence of color alteration is always the same. The only basis for this is the alteration of brain excitability by which the small differences existing in the normal subject increase considerably and in different proportions.

7.2.2 Dyschromatopsias

We think that the dyschromatopsic degradation of composite colors presents a fundamental interest for the theory of color vision since it seems to be the usual form of color alteration in brain lesions. Weakness to yellow and blue-violet, and the confusion of blue with green or, rather, the perception of both colors as green, which occurs in cases of minor disorders such as in the T case, are phenomena that we have detected in a large number of injured people with occipital or other central lesions that disturb visual perception to varying degrees. This type of alteration was not only a predominant and constant phenomenon in these brain-injured people, but it was the only one observed. However, in the literature on chromatic disorders due to brain injuries, this type of alteration in the yellow-blue pair is rarely mentioned. This is probably due to a defect in

the examination of patients because red-green color blindness from retinal anomalies is much better known and frequent, and it is possible that attention has been focused only to this type of color disorder.

Two types of dyschromatopsia can be distinguished; one for colors that become green such as blue and yellow, and another for those that become red such as purple and sometimes violet.

The intensity of dyschromatopsia depends on the degree of brain disorder and the dynamic conditions involved in sensory excitation (facilitation by muscular effort, by binocular effect, etc.). Thus, subject M can exhibit various degrees of impairment depending on the conditions of the experiment. However, it should be noted that even in the optimal state of excitability (binocular vision and facilitation by maximum muscular effort), asynchrony is still present to some extent. This allows to obtain still a photo-heterochromic interval (dyschromatopsia) by sufficiently reducing stimulation intensity. Subject T usually distinguishes the twelve colors of Hering's circle; but in weak illumination, yellow and especially blue tend to become green to some extent, as does white, although the process is much less pronounced than in subject M. Also, in a very brief exposure of such colors, some tendency towards green arises, and even in ordinary conditions of medium illumination, the mutual confusion of green with blue is very frequent. Detailed indications about the various types of vision (or levels of excitability) in relation to colors will be given further on when dealing with chromatopsia.

In relation to dyschromatopsia, it is interesting to comment on the syndrome that Poppelreuter (1917) called *Psychische Farbenschwäche* (psychic color weakness), according to which the different shades of a given color are not perceived in a first stage, i.e., there is a reduction in differential sensitivity; and in more acute cases, colors with some similarity get confused with each other. This author states that the cardinal symptom of psychic color weakness in occipital lesions consists of the confusion of green and blue with each other, and he sees in this symptom a completely different behavior from that of not distinguishing colors due to retinal abnormalities. From the descriptions of Poppelreuter, it seems that he ascribes the referred syndrome to a psychic deficit for the concept of color (*Schwäche in der Auffassung des Farbentones*). On the contrary, we think that it is not necessary to admit complex intellectual disorders since it is simply a color weakness of sensory origin, a deficit in differential sensitivity due to an alteration in brain excitability, and a dyschromatopsia that turns blue into green. The disorders described by the aforementioned author refer to subjects with much lighter disturbances than those of subject M; at most, the chromatic alteration consists of the confusion of colors with similar shades such as blue and green. These are subjects with disorders of similar intensity to those of the T case and even much less, but properly examined would show more complex alterations, as has been seen in the T case concerning greenish chromatopsia when the stimulation with white color is extremely brief.

7.2.3 Chromatopsia

The fact that white and the whole series of greys even to black are seen as green constitutes green chromatopsia or colored vision, which in our cases acquires a more or

less intense green hue for reasons that will be seen later. This singular disorder contributed to considerably hamper the interpretation of the chromatic alteration in the M case, already so complex due to the peculiar physiological conditions. This greenish perception of colorless objects, i.e. of the neutral series (white, gray and black), is known to be characteristic of subjects with *tritanopia* (violet blindness), an extremely rare disorder.

Chromatopsia disappears completely by means of maximum facilitation, although due to some slow development of white sensation, there is still some greenish tint in the first moments, which disappears quickly being replaced with full white. In the inactive state of subject M, a very important attenuation of the greenish tint is achieved when white is strongly illuminated and also observed in very near vision, although there are always small traces of green. Even the solar disk would be seen with some traces of green. As for grey and black, subject M sees them as green too; the medium grey becomes a medium shade of darker green than that perceived in white, and black is perceived as a very dark green. If black is very strong and bright, as on a shiny satin surface, then mixed green and dark strips appear. All kinds of white light show some green. It is not surprising then that black is seen with a bit of green, since almost all blacks reflect a certain amount of light and never fully meet the conditions of absolute black.

Chromatopsia was first investigated in subject M, and much later in subject T, who only shows it when the stimulation is very brief. In this subject it is revealed by illuminating a white paper for an instant in darkness, that is, the greenish tone appears only in the first instants of the establishment of the sensation of white, since his asynchrony is extremely small compared to M. More recently we have verified the existence of chromatopsia in a former brain-injured patient with significant visual field alteration of an intermediate type between concentric reduction and homonymous hemianopsia. This subject presents a state of brain excitability for all visual functions very similar to that of subject M in monocular vision and under facilitation by muscular effort. We have therefore proven accurately the existence of chromatopsia, and in different degree in three different brain-injured subjects.

In both the M and T cases, there are various types of vision according to the conditions of excitability (facilitation, binocularity, etc.), and the eye being used since there is a small difference in vision between the two eyes. Therefore, by combining the various factors, very different states of excitability can be obtained in which chromatopsia is present to varying degrees. Examining carefully such types of excitability, the following phenomena are observed in subject M, under intense illumination of a white piece of paper:

In binocular vision and under facilitation by maximum muscular effort, no green is seen; only by paying close attention he can perceive traces of green in very peripheral vision and at the beginning of the sensation.

Using only the right eye and facilitation by maximum muscular effort, the central part of the sheet is seen white, and peripheral green spots over white background are more abundant than in the previous case.

In binocular vision and in the inactive state, greater amount of green is seen in peripheral vision than in the previous test, and some green dots over white background are seen in the center.

Using only the right eye and in the inactive state, white predominates but there is a large amount of green.

Using only the left eye and in the inactive state, white also predominates but with even more amount of green than in the previous case.

Even under the best conditions of brain excitability (binocularly and with facilitation by muscular effort), a greenish hue is perceived if the observation time is very short. Also by strongly reducing the illumination, full and permanent green coloration is obtained even in binocular vision and with maximum facilitation.

There are therefore important differences with subject T who, in order to perceive a greenish hue using only his right eye, a white paper in the dark must be illuminated only for an instant. This means that only in the first moments of the sensation there is a chromatic asynchrony causing chromatopsia. If the stimulation lasts longer, the green hue disappears very quickly and a normal white appears. In this subject, greenish perception is minimal and only appears as green spots, very close together, over a grey background, and only in the right half of the visual field while the left half is completely white. Chromatopsia appears in the weakest part of the visual field, and although there is a concentric reduction involving the two halves of the field, the right side (contralateral to the brain lesion) is somewhat weaker. If in the test with illumination for an instant, facilitation is applied (whose effect on this subject is much weaker than in M), the green spots become much paler changing to yellowish and appear more spaced out. Using the left eye, which has slightly better vision, no trace of green is obtained even if the white paper is illuminated for an extremely short time. It must be noted that if the right eye (the worse eye) is used, even by reducing illumination conveniently, chromatopsia cannot be obtained in a stable or permanent way, therefore it is not possible to determine excitation curves corresponding to the different stages as we did in the M case.

As regards the third subject mentioned above who has presented greenish chromatopsia, a behavior very similar to that of subject M in monocular vision and under facilitation is observed. This means that only under very intense illumination, the greenish hue of white disappears, so in medium or ordinary illumination, the abnormal greenish hue appears in a permanent and pronounced way. Also, a sheet of yellow or blue paper is altered, presenting an intensely green area in the lower left quadrant of the visual field (weaker region in this third subject).

Chromatopsia, like so many other new phenomena studied here, goes easily unnoticed by patients who suffer from it, and even in well-educated people, as in the case of our third patient indicated, we have been able to discover it quite pronounced after several years of having suffered the brain lesion, without the subject being aware of its existence during all that time.

The fundamental experiment of diversely illuminating a white object to obtain different intensities of green, ranging from total dark green in very low illumination to traces of extremely pale green or yellowish in extremely strong illumination, is equivalent to perform the transition from a reduced perception of the chromatic spectrum to a more complex and differentiated perception of it.

The M case and all these brain-injured people who present the chromatopsia described above, can be considered either tritanopes or tritanomalous, depending on their

brain excitability, giving rise respectively to blue blindness or to blue weakness, blue being the third primary color according to Young-Helmholtz trichromatic theory (Young 1802, Helmoltz 1852). However, we know that the chromatic disorder is more general and not circumscribed to blue; yellow also disappears, and the red-green pair loses saturation and brightness. We must also consider the dyschromatopsia of the yellow-blue pair, which degrades these composite colors giving rise to green without the appearance of neutral spectral zones as occurs in many other cases of retinal type blindness. Thus, we find that under the usual conditions of subject M, he sees a large part of the spectrum (much more than half) only green, and the rest of the spectrum, red, and both colors with a weak saturation. For this reason, the excitation of the entire spectrum, which normally produces white, cannot give in that subject more than a mixture of the indicated colors giving rise to a yellowish green or a pale green. Thus, the explanation of green chromatopsia is simple, a direct consequence of the chromatic deficit in this subject, since his inability to see white is produced by the lack of appropriate ingredients in the spectrum. In this case there is not enough provision of luminosity nor chromatic neutralization of colors with each other.

By representing the dyschromatopsia of subject M in the triangle of colors (Fig. 33), it is easy to appreciate the difficulty to form white. In the Maxwell triangle of colors (Maxwell 1860), each color located on one side of the triangle has its complementary color in the diametrically opposite position with respect to the center, therefore, the combination of both colors in the center produces white. According to the representation of this triangle for case M in Fig. 33, white cannot be achieved by joining complementary colors, and the fusion of the whole spectrum only gives a pale yellowish green hue.

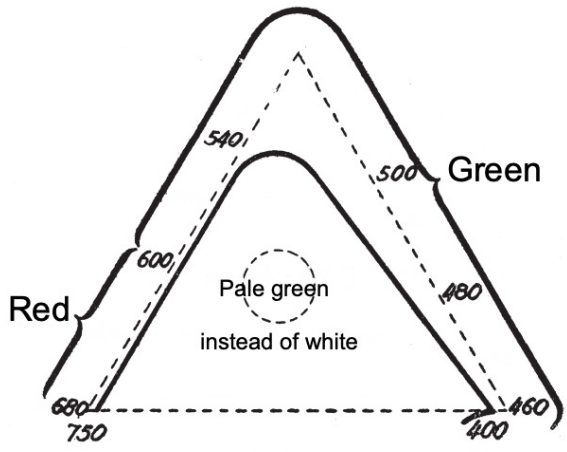


Figure 33. Triangle of colors in the M case in the inactive state. Wavelengths are indicated in nanometers. The braces indicate dyschromatopsias and the central circle represents green chromatopsia.

Therefore, chromatopsia is not due to some special contrast phenomenon, as has sometimes been hypothesized, nor to even more problematic absorption phenomena, but depends solely on the dynamic conditions of brain excitability. The dynamic conditions, by altering color excitabilities and degrading the spectrum, modify the conditions of spectral addition. Chromatopsia is therefore at the heart of color disturbance and

constitutes the synthesis of the whole disorder. It is also shown that the process of formation of white is in agreement with the theory of fusion of the simplest colors (Young 1802, Helmholtz 1852), thus white is the most complex color or function.

7.3 Inversion of color isopters

The special chromatic alteration in the subjects we are studying, especially subject M, still allows us to deal with a very interesting complementary aspect which is the arrangement of colors in the visual field, i.e. color isopters. In a normal situation, the periphery of the visual field is blind or extremely weak to color, and the thresholds for the different colors have different limits giving rise to color isopters. From the periphery to the center, the outermost color is white; the next are blue and yellow; more centrally, red, and finally green is the innermost, the latter being the least extensive in the visual field.

In our two cases, the layout of these limits (color isopters) is totally different; the outermost color is red, even more peripheral than withe. Thus, all the colors are distributed in two types of isopters, one more eccentric for red and another more central for the different greens resulting from the alteration, arranged according to their degree of lightness. Since sensitivity in the visual field decreases rapidly from the center to the periphery and colors show very different excitabilities, they are positioned along the visual field meridian according to their new excitability levels.

Due to the strong concentric reduction of the visual field, especially in subject M, colors occupy a very small area in the field, although by means of facilitation a considerable enlargement is achieved; but despite this enlargement, the position of color isopters is still inverted, i.e. the red-green pair more peripheral than the yellow-blue pair. By studying color isopters in subject M, inactive and under facilitation, and in subject T, the development of the alteration of color isopters can be easily followed because the different cases show different arrangements of them.

The position of color isopters in the right eye of subject M in the inactive state is shown in Fig. 34. The red isopter is the most external, the green is much more inside, and the isopters for white, yellow and finally blue are situated from 5 to 3 degrees. These three colors are shown only as traces of their true color on green background. The different hues can be perceived by using colored tests of 1 cm in diameter under good illumination. The isopters shown in the figure only reveal one of the aspects of color function since, due to the excitability conditions, very diverse changes occur in the visual field. Thus, by carefully performing the chromatic test from the periphery to the center, either the photochromic or the photo-heterochromic interval is found again, depending on the type of color. The values obtained for the right eye of subject M in the inactive state are shown in Table 8.

As indicated in Table 8, with regard to the sensation of minimal luminosity only, red and white are almost together although red is more eccentric than white, and yellow is more eccentric than green. With regard to the emergence of color sensation,

dyschromatopsic greens emerge for white, yellow and blue (in order of lightness), and are situated more externally than the real green. The emergence of the true color for these three composite colors occurs only in traces and more centrally than the real green. We can then say that color isopters show an inverted position since the red-green pair is much more external than the yellow-blue pair. As for white, it is in an intermediate position between both pairs of colors, always bearing in mind that white, yellow and blue are only perceived as traces. Colors are then arranged according to their characteristic excitability, complying with the above-mentioned relationships on chromatic intervals of diverse complexity.

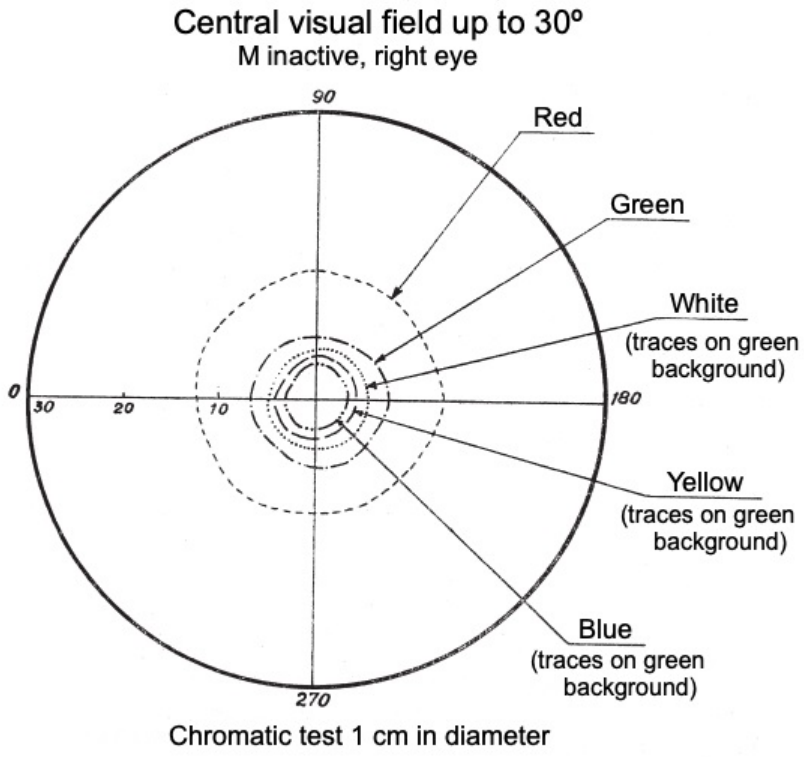


Figure 34. Position of color isopters in the visual field of the right eye of subject M in the inactive state. The limits indicated are only for colors; each color can produce a sensation of simple achromatic luminosity at a much higher eccentricity.

Table 8. Degrees of visual field for limits of minimal simple luminosity and minimal color in the right eye of subject M in the inactive state, using colored tests of 1 cm in diameter.

Color	*Minimal simple luminosity*	*Minimal color sensation*
Red	24º	……...13º pure red
Green	14º	…….....6º pure green
White	23º	12º dyschromatopsic green; 5º traces of white
Yellow	17º	9º " " ; 3º traces of yellow
Blue	14º	7º " " ; 3º traces of blue

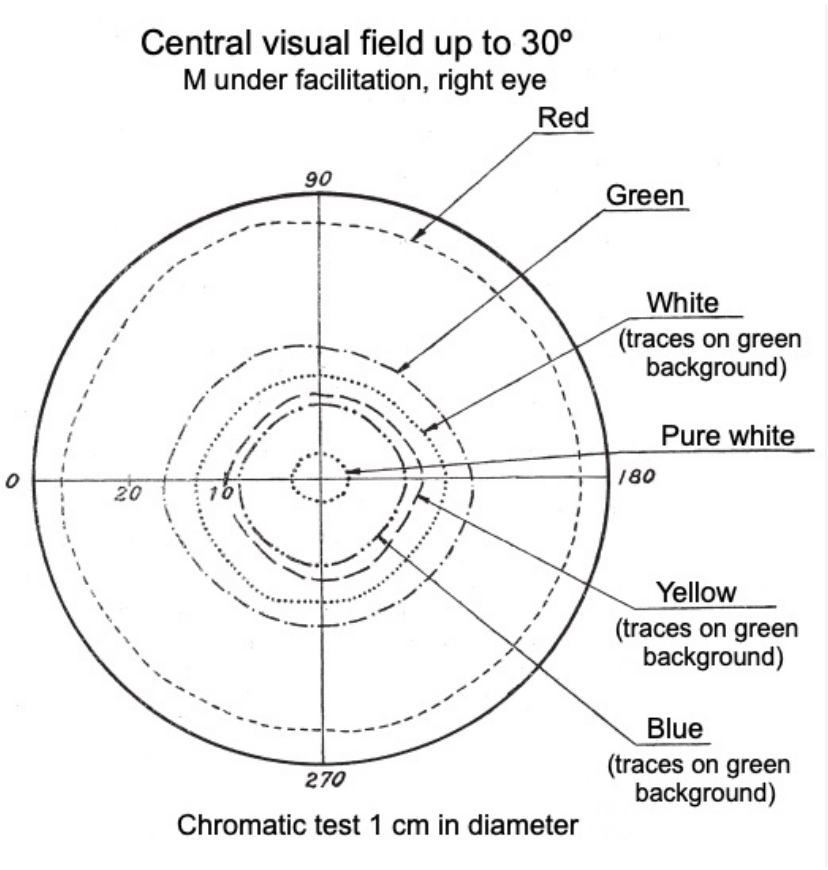


Figure 35. Position of color isopters in the visual field of the right eye of subject M under facilitation by strong muscular effort. Same arrangement as in previous Fig. 34 but color isopters are more eccentric here. There is the important novelty that pure white can be seen at 3º, and yellow and blue are almost completely free of green at 0º.

The same tests for the right eye of subject M but now under facilitation by strong muscular effort result in more enlarged color isopters (Fig. 35), approximately double or triple depending on the colors, but the sequence remains the same as in the inactive state. However, there is the important novelty that pure white without green can be seen at about 3º, and yellow and blue are seen in the center (0º) almost entirely devoid of discromatopsic green hue. As in the previous test, photochromic intervals of diverse complexity are obtained, as shown in Table 9.

Compared to the previous test in the inactive state, it can be seen that the intervals are smaller, especially the photo-heterochromic intervals; pure white is obtained close to the center, and both yellow and blue with almost no green trace are obtained at the center. A larger number of phases can be determined in the composite colors, as is indicated in Table 9: simple luminosity, dyschromatopsic green, traces of color, and pure or almost pure color. We see that facilitation makes that the limit of traces of white in inactive state (at 5º) becomes the limit of full white (at 3º), and similarly for the other composite colors. But as we know, facilitation is not able in the M case to completely restore the normal

function, even if excitability improves very significantly. A shorter interval under facilitation means, as has been said several times, less asynchrony. By means of facilitation, the interval is reduced to a little less than half the interval in the inactive state.

Table 9. Degrees of visual field for limits of minimal simple luminosity and minimal color in the right eye of subject M under facilitation by strong muscular effort, using colored tests of 1 cm in diameter.

Color	*Minimal simple luminosity*	*Minimal color sensation*
Red	45º	…… 27º pure red
Green	35º	……..... 17º pure green
White	40º	22º dyschr. green; 13º traces of white; 3º pure white
Yellow	32º	20º " " ; 10º " yellow; 0º almost pure yellow
Blue	32º	20º " " ; 9º " blue; 0º almost pure blue

For subject T, an arrangement of color isopters intermediate between the normal case and the M case is obtained; so there is already a trend towards a more normal color function. There is no photo-heterochromic interval for yellow and blue or it is insignificant and difficult to appreciate; only for white, a certain dyschromatopsic phase appears in the first moments of the sensation, already mentioned in previous pages. In addition, due to the greater difficulty in differentiating blue from green, there may be something like a dyschromatopsic phase in the former.

By examining, as in the previous cases, the temporal meridian of the right eye (of worse vision in this subject) and using the same illumination in the perimeter as in the previous tests, the limits obtained for the chromatic intervals in the visual field are shown in Table 10.

Table 10. Degrees of visual field for the limits of photochromic and photo-heterochromic intervals in the right eye of subject T, using colored tests of 1 cm in diameter.

Color	*Minimal simple luminosity*	*Minimal color sensation*
White	42º	37º fleeting traces of green; 34º pure white
Red	39º	…….. …….. 32º pure red
Yellow	29º	……. 22º pure yellow
Green	29º	……. 21º pure green
Blue	21º	16º green; ……... 12º pure blue

Color isopters for this case are shown in Fig. 36. Their positions only undergo some alterations without reaching such important inversions as in the previous case. In this case

T, only a single photo-heterochromic interval can be considered (in the blue color) since in white, chromatopsia is extremely fleeting and unstable. In addition, photochromic intervals are very small compared to subject M under facilitation, for example for the red-green pair. The anomaly in color isopters lies in the fact that yellow is inside the red and at some distance from it, and that blue isopter is the most internal. Also, although the white isopter is the most peripheral, it is very close to the red isopter. There is, then, a tendency to a certain inversion of color isopters since the yellow isopter is more constricted than the red isopter, and the blue isopter more constricted than the green one, whereas the isopters for the yellow-blue pair are more peripheral than the red isopter in a normal case.

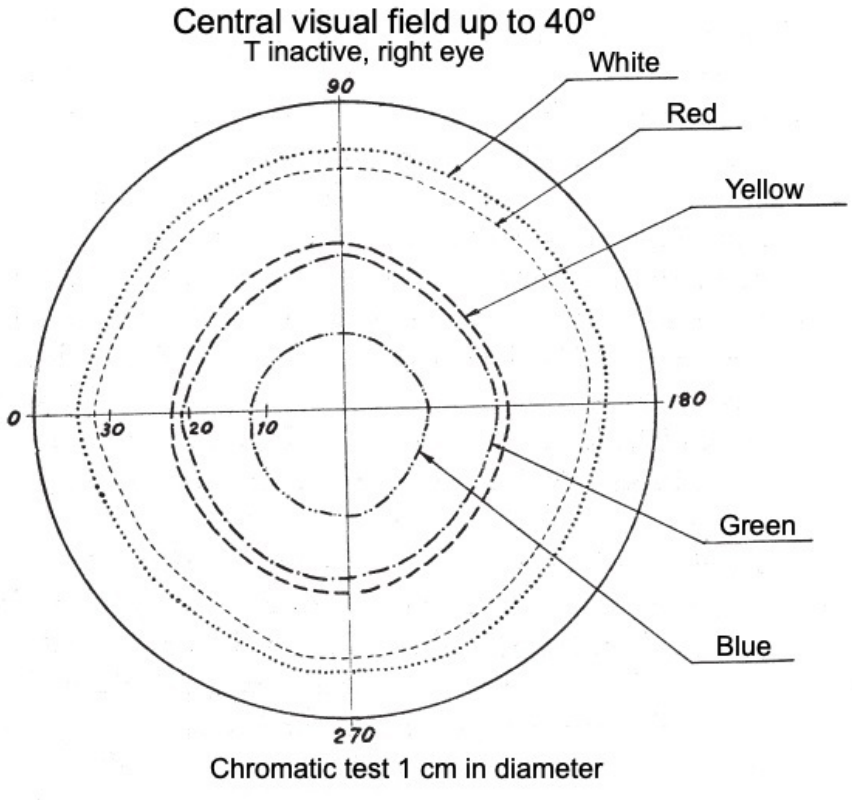


Figure 36. Color isopters in subject T. Abnormalities: the yellow isopter is more constricted than the red isopter, and the blue isopter is the innermost.

Such alterations in color isopters, from total to moderate inversion, are clearly in relation to the excitability characteristics in each case.

All these results suggest certain considerations about the theory of functional duplicity of the retina. This theory postulates a different functionality for the two types of receptors in the retina, cones and rods. Cones are ascribed to diurnal vision, especially to colors, and rods to night vision (Parinaud 1898, Kries 1905). The Purkinje phenomenon (Purkinje 1825) is invoked as particularly demonstrative of that theory, and it is generally accepted that in low luminosity, vision is ensured by sensitive blue and blue-green rods,

whereas in high luminosity, only cones excitable by long wavelength radiation would intervene.

However, the inversion of color isopters does not fit in any way with this theory of duplicity. Even in cases of less severe color disorders than those mentioned above, a large number of brain-injured patients had an inversion consisting of seeing red more peripherally than blue. Also, some sporadic cases can be found in the literature without special comment. In extreme cases such as subject M and the third brain-injured subject mentioned in the preceding pages, the red isopter is the outermost, much more peripheral than the isopter for white and all the others. In contrast, the blue isopter is the most internal even in cases of very moderate alteration. Such an arrangement should not be attributed to anatomical differences of the receptors but rather to the differential activities of the brain centers that undergo a major change by varying their nervous excitability.

Normal color blindness at the periphery of the retina does not seem to be caused by simple anatomical changes in the receptor elements. Its origin is probably to be sought in the functional features of colors, starting from a global conception of color vision in relation to excitability phenomena.

7.4 Alteration of chromatic induction phenomena

We shall complete the study of chromatic dynamics by examining the behavior of chromatic contrast phenomena, especially in the M case and in its two main forms: simultaneous or edge contrast and formation of afterimages or successive contrast.

Edge contrast presents in subject M the peculiar effect of a very important magnification. When a red and a green paper are presented to him together, strips of a more intense color appear in the edge zone. These edge strips are very bright and very wide; they are 22 mm wide at the red side and about 15 mm wide at the green side; widths that could never be reached in a normal subject. This effect is obtained without difficulty and quite rapidly. The same occurs with yellow and blue; under the same conditions, a color-enhanced edge strip 12 mm wide is obtained at the yellow side, and 15 mm at the blue side. These colors, which the subject usually perceives as pale green, look almost normal in the enhanced edge zone, although greenish dyschromatopsia does not completely disappear.

By means of facilitation, a significant reduction in the increase of edge contrast is obtained, thus resembling the behavior of a normal subject. The width of the contrast strip is reduced to 1/5 of the previous value and its saturation is also reduced. This singular behavior in edge contrast is known as typical of people with color weakness (or *anomalous*)**,** in whom edge contrast acquires great intensity. Where normal people see only a narrow edge contrast, anomalous perceive wide areas of intense color; and whereas a normal subject needs a fairly long time for the edge contrast to appear, in anomalous it is almost instantaneous (Nagel 1907, Guttmann 1920, Koffka 1922, and others).

Subject M can certainly be considered affected by color weakness and can be considered tritanomalous given his blue-violet weakness even under optimal conditions of stimulation. In his usual state, he should really be considered as affected by *tritanopia*

or blue-violet blindness, and since color blindness goes in pairs, blind to the blue-yellow pair. The T case fulfills the conditions of tritanomalous given his blue weakness, blue being the third primary color in the trichromatic theory. This tritanomaly is a very rare alteration and was reported by Engelking (1925).

Regarding the interpretation of the enhanced edge contrast, it could perhaps be explained by an increase in color fatigue, which would favor an increase in the complementary color, although more detailed research would be needed.

Concerning the successive contrast, i.e., the formation of afterimages, we find in the M case the disappearance of the negative afterimage at the expense of an enhancement of the positive afterimage which persists for a long time due to the slowness of the visual function.

When subject M stares at a red surface for a certain time and then immediately looks at a neutral surface, he does not see green, as would be normal, but continues to see red for a period of time. The same result is obtained with any other color. In all cases, it is impossible to somehow obtain the complementary color that forms the negative afterimage, and he always sees the same color he was initially looking at. If he is instructed to look at a yellow surface until yellowish stripes are generated on a green background, and then look at a neutral surface, the image remains the same, and after a while the yellowish stripes on green background disappear and the whole surface becomes green until the whole image disappears completely. By means of facilitation by muscular contraction, which accelerates the development of sensation thus considerably decreasing its persistence, a negative after image can be obtained although weakly developed.

Pötzl (1916, 1928) also found a chromatic induction disorder in occipital lesions altering brightness and color, especially in patients who being blind in the acute phase recover their vision. An increase in positive afterimages and a decrease in negative ones can then be observed, without the author providing more specific data on these phenomena.

Finally, the alteration of the afterimages suggests the hypothesis that such subjects could behave like the *eidetic* subjects of Jaensch (1923, 1930), that is, they would have a form of perception (without negative afterimage) that many children normally present, which leads them to take representations and perceptions at the same level, thus contributing to create a special state of fantasy; later, but before puberty, the negative afterimage appears and perceptions are better distinguished from representations. In our subjects, the change suffered in sensory activity with the emergence of eidetism could be the cause of the distortion of perceptions, pseudoagnosia, autotopagnosia, exclusion of pathological defects, etc. In other patients, singular phenomena such as the phantom limb of the limb amputee, hemianopsic hallucinations, or the mental state of blind subjects by central lesion who assert good vision (Anton syndrome, of which we have studied some cases in brain-injured people), and many other manifestations of anosognosia (ignorance of one's own disorder), may be attributable to eidetic-type changes in sensory structure. But all this is conjecture, and precise investigations would be needed before the mechanism of such phenomena could be determined. In any case we would have a precise physiological and experimental base for further elucidation

8 Theory of color differentiation

8.1 Color differentiation action

The two subjects studied here, as well as others whose degree of alteration is very diverse, show many types of color degradation which can be gradually classified from color *anomaly* to color *blindness*. This can already be exemplified with only the two cases, M and T, putting into play various summation effects (binocular effect, muscular effort, etc.). In the most favorable conditions of excitability, the color vision obtained corresponds to that of *anomalous trichromats* according to Kries (1899) and also Guttmann (1908, 1909,1920) and Nagel (1907), whose characteristics are: the times necessary to recognize colors are extremely increased, the limit of a hue is confusing, colors are not perceived in the periphery of the visual field but are seen relatively well in the center. Such characteristics are fulfilled very well by subject T who can see the three primary colors but he presents blue color weakness and can therefore be considered as affected by tritanomaly, a rare anomaly so far. The same happens to some extent in the M case under optimal vision conditions (binocular effect and facilitation by muscular effort). But this case in an inactive state already presents the characteristics of a tritanope, i.e. blue color blindness, a defect that includes the opposite color, yellow, without all the other colors being totally undamaged. Therefore, all kinds of transition forms are obtained, from normal color vision to the deepest deficit, but always based on the fundamental alteration of the third primary color according to the trichromatic theory.

Both tritanomaly and tritanopia are little known disorders, but this does not prevent them from being common forms of color alteration in lesions affecting the visual area. In several cases we have studied with slight disorder, what is characteristic is a certain blue-yellow weakness with a predominance of deficit for blue, as well as a certain inversion of color isopters so that red is more eccentric than blue in the visual field. This form of alteration is, as stated, the result of changes in nervous excitability of colors.

A theory of color vision or a meticulous interpretation of all these types of phenomena cannot be expected here, where only the description of the outstanding manifestations and a general explanation of brain excitability conditions have a place.

Considering the facts observed from the point of view of the two most important color theories, the trichromatic theory of Young-Helmholtz (Young 1802, Helmoltz 1852) and the complementary pair theory of Hering (1874/1878, 1880), we find that these theories only partially agree with the phenomena studied. Let us take as an example the case of the white color. In the trichromatic theory it is accepted that white is the result of

the excitation of the three primary colors; red, green and blue-violet. In the case of blindness to the third primary color, the fusion of the remaining colors cannot produce white and a more or less greenish-yellow or pale green is obtained due to the fusion of red, green and the green from the altered yellow and blue, thus explaining chromatopsia perfectly. In this case, Hering's hypothesis that the visual white substance is responsible of the formation of all the grey series and white, cannot be sustained. If the activity of that substance were to decrease, grey would be obtained instead of white, but not a greenish hue.

A different situation is that of yellow. In trichromatic theory, yellow is a composite color obtained by fusing red and green. In Hering's theory, yellow is part of the yellow-blue visual substance and both colors are altered simultaneously. In our case M, there is no difficulty in thinking that yellow is a composite color since it appears degraded, it is seen as green, and therefore it is not primary. We also know that the type of blindness in M is predominantly to the yellow-blue pair, although the remaining colors are not totally undamaged. This paired blindness is the best argument in favor of Hering's theory, although it fails in relation to chromatopsia and white, and this is important.

In cases of slight alteration, such as the T case, the predominant alteration is found in blue; then there is no chromatic pair disorder and Hering's theory is again questioned. The fact that the yellow-blue pair is perceived altered by the dyschromatopsia of subject M, i.e., with a green tint, is not in favor of these colors being simple nor of the yellow-blue substance of Hering. Thus, it can be said that the phenomena, in general, are more easily explained by the trichromatic theory, although it is necessary to appeal to that of Hering for the alteration of the chromatic pair. However, the third primary color, blue or blue-violet, also is altered by dyschromatopsia, which is not a fact in favor of it being a simple or primary color as accepted by the trichromatic theory.

Each of the two main theories of color vision, which we consider sufficiently well known, is based on important facts and arguments, but both also face difficulties that neither of them has been able to overcome from their own perspective. In general, physiologists side with the trichromatic theory, and psychologists side with Hering’s theory. Many attempts have been made to bring the two closer together by trying to involve different stages, both peripheral and central, in the chromatic process (Hecht and Shlaer 1936, Müller 1917, Piéron 1939, Troland 1920, 1921, etc.), giving increasing importance to the chromatic processes that should take place in the centers of the cerebral cortex. Now, it is clear that in view of the nature of the cases studied here, it is only at this stage of nervous centers that we should place ourselves, and for the same reason the whole chromatic issue must be focused in this study on the alterations in the brain excitability of colors. Colors already present small differences in a normal individual with respect to reaction speed and color establishment, and these differences are pathologically amplified, the slower colors being more impaired and tending to be excluded.

Thus, blue (the third primary color of the trichromatic theory), being the slowest color in a normal person, is the first to be altered, so tritanomaly and, in more pronounced cases, tritanopia, should be considered typical forms of color disorder in brain lesions involving vision. As has been said, a characteristic of the alterations studied in our brain-injured patients is that the most complex functions are the most affected, splitting into

partial functions and being reduced to simpler activities. This is mainly fulfilled for white, which is the maximum composite color in the Young-Helmoltz theory, since the three primary colors must participate in its formation. As for the other colors, it is observed that red and green are the only colors that remain unaltered, whereas yellow and blue are green dyed due to dyschromatopsia. Therefore, it can be said that red and green are the only two primary colors and would form the only elementary pair, whereas the other yellow-blue pair would be formed from the primary pair: yellow by combination of red and green, as assumed in the trichromatic theory, and blue from green with some other hue and in special conditions of luminosity. This pair, yellow-blue, would be composite colors resulting from the action of *color differentiation* in brain centers.

The conditions for color vision depend entirely on the excitability relationships established according to the degree of activity of the nervous centers. Red and green are the most stable colors because they have the highest reaction speed, and the other colors would be generated by progressive differentiation from them. Yellow, for example, would need the previous existence of red and green and with a certain degree of intensity. If this pair is diminished, there would not be enough chromatic contribution to originate the yellow color because the combination of red and green would only form a yellowish green that cannot be differentiated from the real green. Likewise, if blue is weakened, it can easily be confused with green which seems to intervene in its formation. In this way, these two colors, yellow and blue, appear little differentiated, rather degraded, adopting a tone of simpler colors that participate in their formation. If the excitabilities of colors are very close as in a normal individual, the colors that we can consider composite (yellow-blue pair) are well differentiated and easily formed, as well as white and grays. Instead, if asynchrony occurs, all composite colors succumb because their processing is incomplete and show abnormal hues that reveal their composite nature.

If such an interpretation proves to be valid, it could be said that all the colors, more completely or incompletely, are developed from red and green which seem to be simple. Summarizing, it can be said that chromatic differentiation has its physical brain base in the degree of nervous synchronization of the centers. This differentiation by means of the combination of simpler colors would be more in line with the trichromatic theory than with that of complementary colors. Finally, color degradation according to the conditions created by asynchrony generates a dynamic reduction of color differentiation by excluding the more complex and less excitable colors.

8.2 The problem of color processing in the brain

Under the influence of the theory of brain localization, attempts have been made, using the anatomical-clinical method, to determine a localization for color vision within the occipital lobe, on the basis of subjects with occipital lesions who showed more or less intense chromatic disorders. However, because of all that is indicated in the general part about the action of nervous centers and the dynamic conception of the brain, such a localization, which has never crystallized into something certain, is difficult to admit.

From our observations in many brain-injured people, and especially in the subjects studied here, it follows that any localization in areas of the cerebral cortex, excluding the marginal areas which are practically the projection pathways, seems impossible. Brain centers appear to act only as nervous masses that activate the receptors, without determining specificity of any kind. We have seen quite intense chromatic alterations in lesions of the left hemisphere, right hemisphere and in zones both close and distant to the calcarine cortex. In all cases the intensity of the disorder depends on the amount of brain mass destroyed, provided that the lesions are not too far from the occipital lobe, since for a given intensity of injury, its effect on vision decreases as the distance to the visual projection cortex increases. In favor of excitability processes as a basis for color vision and ruling out any kind of anatomical localization, we can only cite Stein (1928, 1930), who admits or supposes a chronaxic characterization of colors similar to that of the different tactile qualities. But this functional conception represents a rarity in the midst of the theories of anatomical localization which, being more or less diffuse, are widespread everywhere.

A rigorous research shows that there is no reason to think about localization of color or any other type of activity in the visual domain (form, motion, orientation, etc.), and we can only talk about the greater or lesser degree of organization and differentiation of the visual function in relation to the level of brain excitability. The central lesion dynamically reduces all sensory activity in the brain, and a dynamic reduction is established in the visual system by virtue of which the less excitable functions are the first to be altered, they tend to be delayed or totally excluded. This applies both to colors and to any other type of function as we shall see next. The alteration is therefore global and according to the excitability of each function.

Visual forms

9 Visual field

9.1 Concentric reduction

In this chapter, we shall study visual forms, a type of sensoriality that corresponds in psychological language to perceptions, that is, to the spatio-temporal organization of visual stimuli. We shall start with a matter of primary importance which is the structure and modifications of the visual field. Next we shall deal with spatial localization, which in our subjects appears under the remarkable phenomenon of flat color vision (or color irradiation). Finally, we shall address phenomena of greatest differentiation such as spatial discrimination (acuity), motion perception and conceptualization of complex forms and configurations of objects.

The study of the visual field in the cases presented here is very appropriate, given its simplicity and characteristics, to demonstrate the physiological disorder that leads to asynchrony and dynamic reduction. In our two cases as well as in the Schneider case of Goldstein and Gelb (1918), there is a visual field alteration of the type *concentric reduction*, whose different intensity shows the degree of brain alteration in each of these three cases; highly intense in case M, of medium intensity in case Schneider and moderate in case T.

As already mentioned in Part I of this work, the concentric reduction of the visual field poses a serious and unsolvable problem to the theory of cerebral localization, within which there is no possible interpretation since it is impossible to imagine an anatomical disposition in the cerebral cortex that concentrically disturbs both eyes equally, being the lesion in only one hemisphere. Moreover, in this type of visual field alteration, it is characteristic that the brain lesion is located outside the visual projection area of the cortex, generally in the parieto-occipital region, i. e. at a great distance from the calcarine cortex or striated area. By contrast, the explanation of the concentric reduction is very easy and immediate within the dynamic conception we advocate, since it is only a dynamic reduction in its simplest form.

In the study of the concentric reduction in our two cases, using a white test of 0.5 cm in diameter at the perimeter, we find very different degrees of reduction. Subject M in the inactive state presents a very intense reduction since the limit of vision only reaches up to 6º in the temporal meridian of his right eye and up to 4º in that of his left eye (Fig. 37), whereas a normal subject in the same illumination conditions reaches up to 90º.

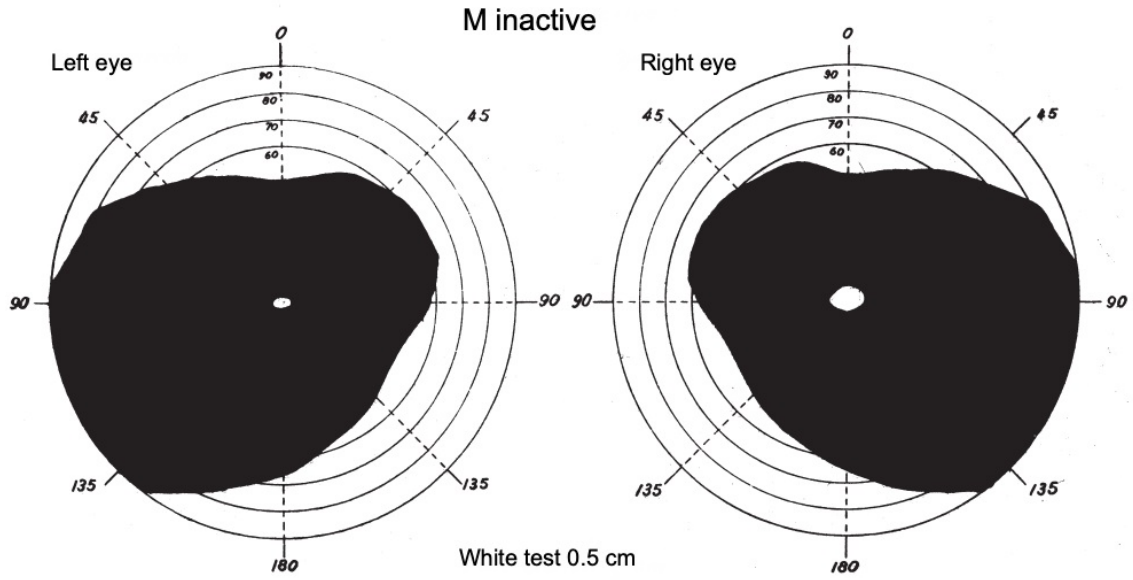


Figure 37. The visual fields of subject M in inactive state. White test of 0.5 cm in diameter. Note the intense concentric reduction, somewhat more pronounced in the left eye.

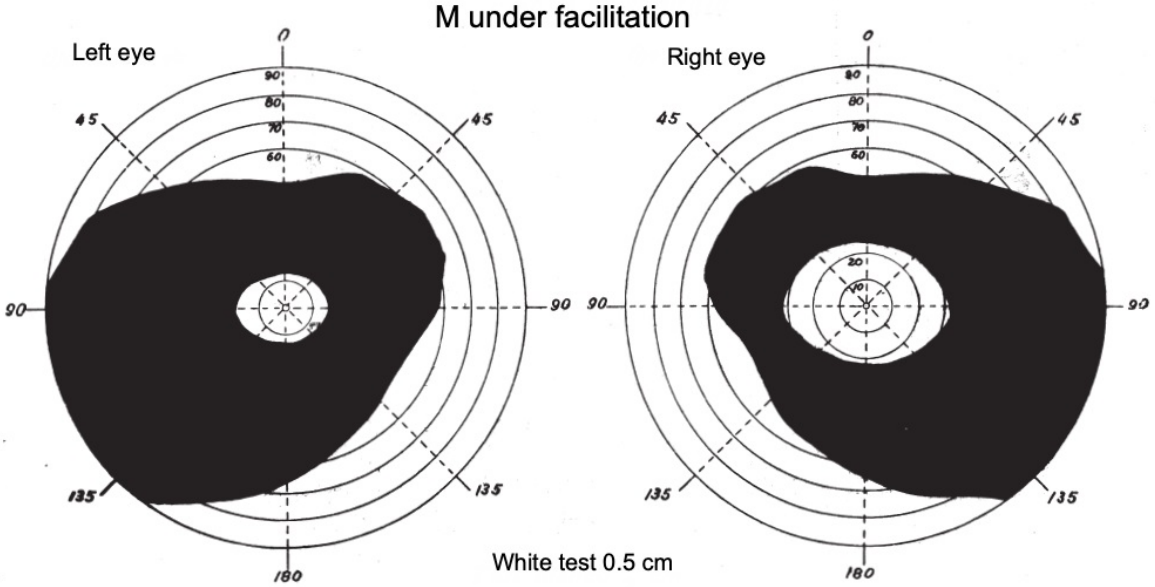


Figure 38. The visual fields of subject M under facilitation by maximum muscular effort. An enlargement of about five times with respect to the previous inactive state is obtained.

Therefore, vision is reduced to macular vision approximately. In the study of color isopters described above, the limits are wider because a test of 1 cm in diameter and greater perimeter illumination were used to properly investigate color manifestations. For a proper comparison with a normal individual, the limit of vision is determined not by the simple sensation of luminosity produced by moving the test from the periphery, but by a vision that provides a more defined sensation which in M corresponds to the appearance of a greenish tint on the white test. This occurs at 6° with the 0.5 cm diameter test and at 10° with the 1 cm diameter test, always using sufficient illumination for a normal subject to reach 90° with the 0.5 cm diameter test.

The concentric reduction is not perfectly circular but the visible field maintains a somewhat elliptical shape, i.e. the area of vision extends a little more along the horizontal meridian. This significant reduction does not mean that peripheral areas are absolutely blind. As said, it is enough to increase the diameter of the test to achieve a wider area of vision. Thus, by sufficiently intensifying the luminous stimulus, the area of vision is extended to the entire visual field even if the subject is free of any facilitation, as we shall see later. Therefore, the disturbance is relative and there is no absolute blindness in the rest of the field. Excitability decreases rapidly from the center to the periphery of the field in a normal case, and the brain lesion causes a general disturbance in the field that given its functional structure, the more peripheral the area the more pronounced the disturbance.

If facilitation is applied by means of a strong muscular contraction of the whole body, the increased brain excitability leads to an enlargement of the visual field of about five times that of the inactive state. For the right eye, for example, the limit of the field of vision is increased from 6° to 30° (Fig. 38). This effect of facilitation on the visual field was already found at the end of 1939, when the action of muscular contraction was discovered and applied to the investigation of a large number of functions. However, the enlargement obtained does not completely eliminate the field reduction because this type of facilitation is not capable of restoring brain activity *ad integrum*, as we know from other examples. But somewhat larger enlargements of the visual field can be obtained by combining several types of facilitation (muscular effort, binocular effect, etc.), although we shall now consider only the above effect for simplicity. It is necessary to point out that the enlargement of the field by means of facilitation, with respect to the inactive state, increases as the intensity of the stimulus used decreases. Thus, for example, a stimulus perceived in the inactive state at 6º, can be perceived at 30º (five times higher) with facilitation, whereas another more intense stimulus perceived in the inactive state at 12º, can be perceived at 35º or 40º (only about three times higher) with facilitation. Therefore, enlargement through facilitation decreases as stimuli become more intense, as we shall see in the corresponding examinations below. In general, for stimuli normally used in the visual field examination, magnification of the field through facilitation is four to five times the value in the inactive state.

As regards the second case, subject T, the concentric reduction is much more moderate, as can be seen in Fig. 39. The field of vision can reach up to 50º with the 0.5 cm white test, the field being slightly larger in the left eye than in the right eye, and showing an elliptical contour as in the above case. However, visibility is diminished in this case by the presence of a small *annular scotoma* which was much more widespread at the

beginning of the examination of this subject. This type of scotoma results from the special dynamic conditions of the concentric reduction. In the examination with the perimeter, moving the test from the periphery to the center, the test becomes visible at a certain moment, and continuing slowly towards the center, the test stops being seen very soon because the subject's vision becomes fatigued because it takes place in weak zones**.** But by continuing towards the center, the test object is seen again due to the stimulation of increasingly sensitive areas. The zone in which vision is interrupted is the scotoma, which appears in all meridians giving rise to an annular scotoma, fully functional, dependent on the conditions of examination in an unstable and easily fatigued field.

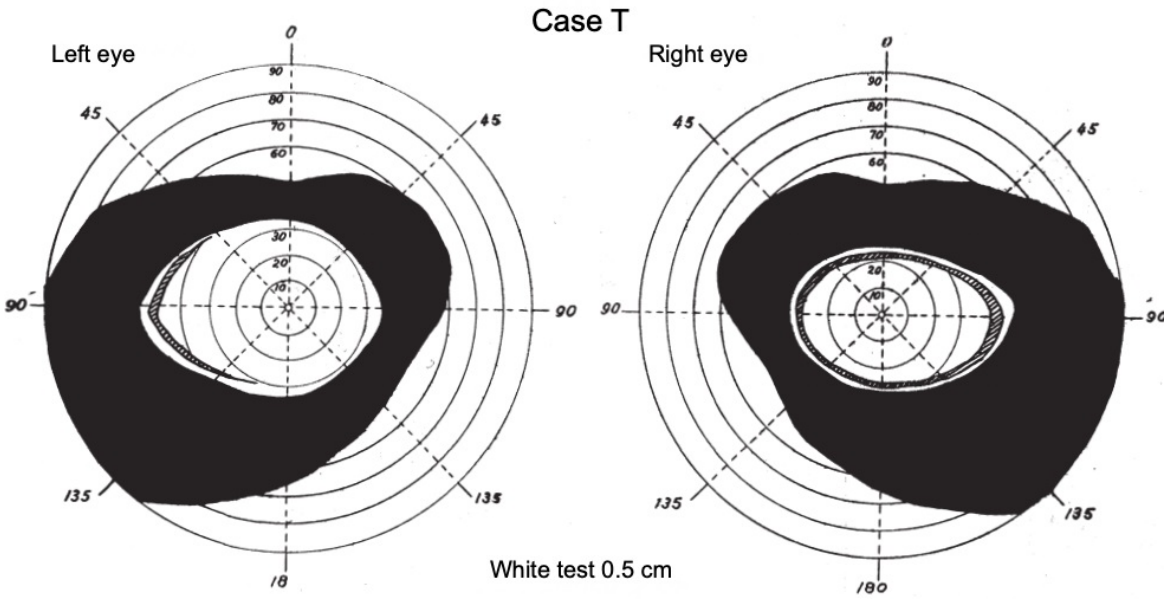


Figure 39. The visual fields of subject T. Compare with Figs 37 and 38. Note the small annular scotoma in right eye and an incomplete one in left eye (the better eye).

Goldstein and Gelb (1918), in a series of brain-injured people from the First World War, with visual alteration of the type concentric reduction of the visual field, were the first authors to give a rational explanation of the annular scotoma. Previously, these scotomas had been verified on different occasions, but without a plausible explanation they were wrongly admitted as a hysterical manifestation. This erroneous belief also occurred in the first observed cases of concentric reduction of the visual field, although the enormous number of subsequent observations in brain-injured people led that view to be abandoned. In other publications, we shall deal with different types of annular scotomas that occur along with other dynamic visual field alterations such as polyopia, macular deviation, etc.

As we already know, the T case offers the possibility of a small facilitation, so it is possible to obtain a small magnification of the visual field which demonstrates precisely the weak effect of facilitation on this subject, also due to the larger size of the field in the inactive state. Thus, in the case of a quite peripheral vision, the enlargement of the visual field by facilitation would be minimal.

Finally, Fig. 40 shows the visual field of the Schneider case of Goldstein and Gelb (1918). It can be seen that the degree of concentric reduction is between the more severe case M and the less severe case T. It can also be said that the M case with facilitation is less severe than the Schneider case. Hence, it would be easy to deduce the intensity of the disorders in the Schneider subject for all types of visual functions and for any other sensory system. The above-mentioned authors have described only a very small number of disturbances and with a very different interpretation, as indicated in the general part (Part I) of this work.

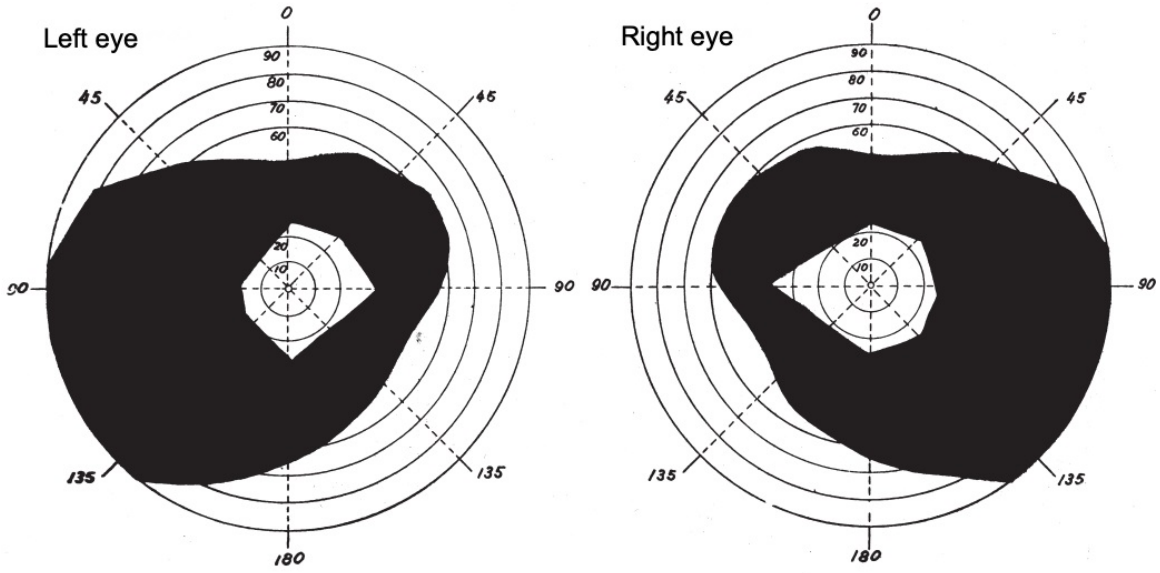


Figure 40. The visual fields of the Schneider case of Goldstein and Gelb (1918). Concentric reduction to an intermediate degree between that of the M and T cases. Subject M under facilitation can be similar to the Schneider case.

9.2 Excitability relations in the visual field

We shall now deal with the experimental research of the concentric reduction of the visual field by applying the dynamic analysis (fundamental experiment, synchronization by facilitation, etc.). The tests to be performed are easy since they are reduced to examining the vision with the test object along the perimeter in different conditions of stimulation: varying time, intensity, facilitation, etc. However, this simplicity of analysis is hampered by the irregularities that peripheral vision may present. The patients sometimes have great difficulty in keeping the eye stationary looking at the central fixation point, and easily look at the peripheral stimulus. In addition, we must expect the appearance of peripheral fatigue, changes in attention, etc. For these reasons, these examinations can be very painful and must be prolonged in time and repeated sufficiently until usable data are obtained. It is not realistic to expect a completely exact match between different examinations, but only approximate, as there is always some margin of variation, even in a normal individual.

First of all, it should be recalled that the visual field is not functionally uniform even in a normal situation, and all kinds of functions are quickly weakened from the center to the periphery. Figure 41, taken from Charpentier (1903), shows how the sensitivity of the retina decreases from the center to the periphery; it shows the additional illumination that must be given to the test object to distinguish it from the background. Sensitivity decreases very rapidly from 40º. It is therefore understandable that in an overall impairment of visual function, the peripheral zones of the visual field fail much more easily than the more excitable central zones, and thus a concentric reduction of the visual field occurs.

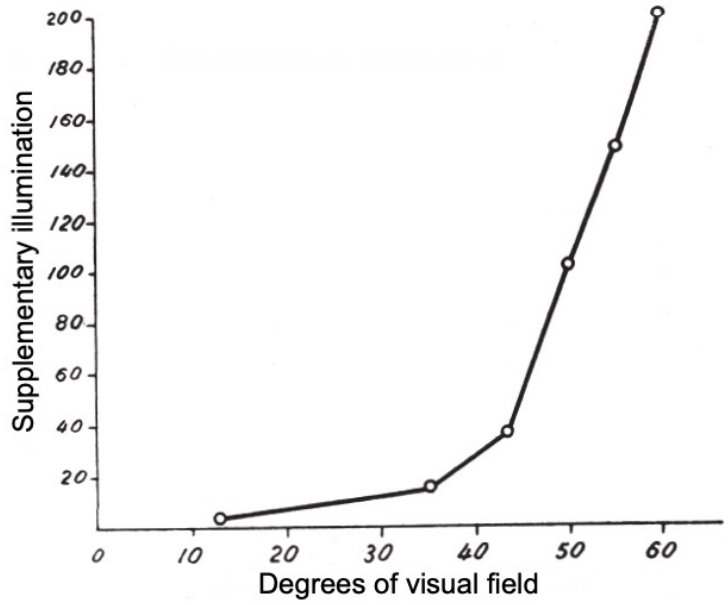


Figure 41. Additional illumination of the test object versus the degrees of the visual field. Sensitivity decreases strongly from 40° (Charpentier 1903).

Asynchrony affects all kinds of functions in such a way that normally less excitable functions are proportionally more disturbed. This explains that the weakest functions are excluded (dynamic reduction) and can only be recovered by extremely high intensity stimuli.

Due to this asynchrony, the already existing excitability differences in the normal individual undergo a considerable enlargement, as we have mentioned above regarding the photochromic interval in colors. An example of this enlargement of the normal interval is shown in Fig. 42, which shows the strength-duration curves obtained with stimulation at 0° and 20° of the visual field, for the right eye of subject M in the inactive state, using a white test object 1 cm in diameter. In addition to the large difference (interval) in the rheobase level, the different curvature of the curves indicates the different reaction speed, much lower at 20º of the visual field. By applying facilitation by muscular effort, the mentioned enlargement decreases considerably and is reduced to approximately one quarter, but it is still very large compared to the value of the normal excitability interval obtained under these experimental conditions between 0º and 20º. As

the interval decreases by means of facilitation, the corresponding curves show a more pronounced curvature, increasing the reaction speed as indicated above in other experiments.

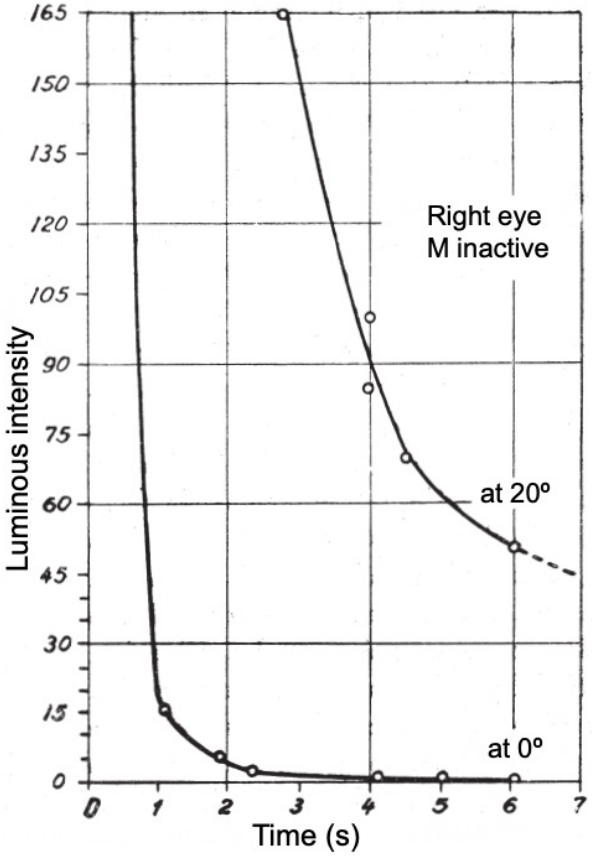


Figure 42. Curves of luminous excitability (strength-duration) at 0º and at 20º of the perimeter using a white test object 1 cm in diameter, for the right eye of subject M inactive. There is a considerable widening of the normal interval.

The great decrease in excitability at 20º that subject M experiences in the inactive state is understandable given the intense concentric reduction, so to achieve vision in this zone, very intense and long-lasting light stimuli are necessary compared to the more central zones. If these types of strength-duration curves are determined for a series of even more peripheral degrees, a bundle of curves is obtained which, compared with the analogous bundle using facilitation and with that of a normal subject, would provide a complete description of the enlargement of the various intervals. But now we must restrict ourselves to simpler relationships since such determinations would mean a huge task in this kind of brain-injured subjects, and such comparisons can be obtained much more simply as we shall see later.

Another excitability relationship to be considered is the one existing between the time elapsed until the sensation is obtained and the perimeter degrees where a stimulus of certain intensity is placed; thus, the reaction speed in different regions of the visual field can be obtained. In the previous figures on the concentric reduction of the visual field, it is not entirely correct to directly compare normal vision with that in pathological

subjects M, T and Schneider, since at the limit of peripheral vision, the time required to obtain sensation is very different in these cases. Therefore, to correctly establish the comparison, it is important to note that at the peripheral limit, whereas a normal subject takes only a few tenths of a second to perceive the stimulus, subject T needs a minimum exposure of about two seconds, subject M under facilitation needs four seconds or more, and about seven seconds if he is in an inactive state.

The increase in time, i.e. the loss of reaction speed, is not exclusive of the most peripheral areas since the excitability disorder, being global, is present in all regions of the visual field, as shown in Fig. 43 for a given stimulus along the horizontal meridian.

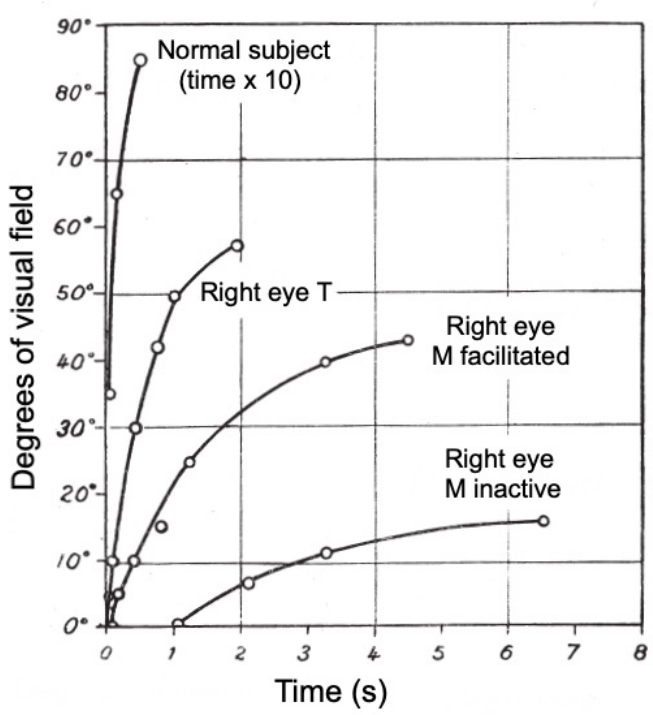


Figure 43. Reaction time in the perimeter for the right eye of: subject M inactive and under facilitation, subject T and a normal subject, using the same stimulus in all cases. The time for the normal subject is multiplied by 10 in order to indicate it in the graph.

For subject M in the inactive state, the speed of reaction is very low along the meridian that he can see: at 0°, about one second of exposure is needed, and at about 13° (the limit of the visible field), six seconds are needed. By contrast, under the action of facilitation by maximum muscular contraction, a remarkable economy of time is achieved with the same stimulus, as well as an enlargement of the visible zone. However, he is still very delayed compared to subject T, and the latter is still very delayed compared to the normal individual, as shown in Fig. 43. These tests show us how the excitability in the visual field decreases from the canter to the periphery in each case, expressed by the decrease in the slope of the curves as well as by the useful time at the limit of peripheral vision.

As indicated, the intense concentric reduction in the M case, and the more moderate reduction in the T case, do not mean absolute blindness in the remaining visual field; it is sufficient to intensify the light stimulus adequately to reach vision in the most peripheral regions, even if the subject is free of any facilitation. That is, it is sufficient to compensate for the large decrease in excitability with an increase in stimulation. This is shown in Fig. 44, where the degrees of peripheral vision are related to the intensity of the stimulus needed to produce sensation in the various cases we have studied.

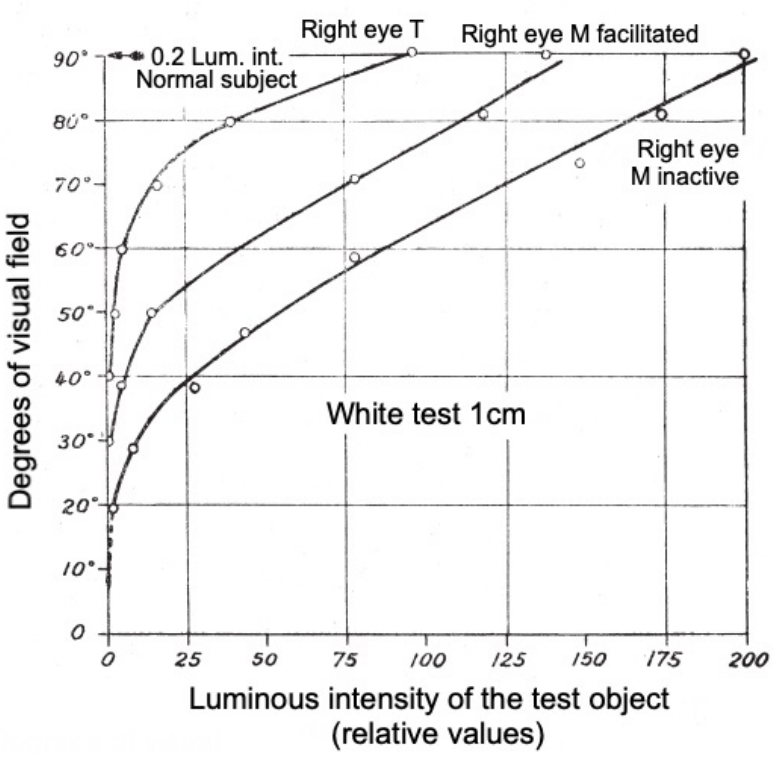


Figure 44. Degrees of the visual field of the right eye versus required stimulus intensity to produce sensation, for three types of excitability: subject M inactive, subject M under facilitation by maximum muscular effort and subject T inactive.

As in Fig. 43 above, the slope of the first part of the curves in Fig. 44 indicates the field excitability status. The curve for the T case presents a greater slope, thus with a moderate increase in the stimulus intensity, the most peripheral zones of the field are easily reached. By contrast, the increase in stimulus intensity in the other two cases (M inactive and facilitated) needs to be very large, especially in subject M inactive, which needs a significant stimulus intensification from 30° onwards. As for M with facilitation, he occupies an intermediate position between T and M inactive, showing a saving of stimulus intensity, with respect to the inactive state, much higher at low intensities than at very high ones. Such curves would correspond to the recruitment of the rheobase levels of the strength-duration curves in different degrees of the visual field (Fig. 42). The necessary values of the stimulus in the most external regions of the field are

extraordinarily high in the case of M inactive, reaching, for 90º of peripheral vision, about one thousand times the stimulus in the normal subject. However, with facilitation, about six hundred times or little more is enough. As mentioned several times, the normally less excitable functions undergo a proportionally much greater deficit than other activities of less physiological demand. For this reason, there is a progressive increase in the functional deficit from the center to the periphery of the visual field, as shown in all the tests carried out. This explains the origin of the concentric reduction of the visual field, without this meaning a complete abolition of the extreme peripheral regions but only their lack of excitability in the face of ordinary stimuli of moderate intensity.

Finally, we must also consider how the peripheral visibility of a stimulus of constant intensity increases by increasing the intensity of the facilitation applied by means of muscular effort. For this purpose, subject M must stand facing the perimeter which is placed at a sufficient height, with his right eye looking at the central point. Once the outer limit of vision is reached without any muscular effort, the enlargement achieved is determined as a function of the weights held by the subject, in the same way as described in the experiments on electrical excitability of the retina. The results obtained are shown in Fig. 45, which shows two curves for test objects of 0.5 cm and 1 cm diameter respectively. As in the curve showing the voltage saved by facilitation (Fig. 11), the visual field increases rapidly with facilitation at the beginning of the curve, and then more and more slowly. In proportion, the smaller test leads to a larger increase. Thus, the limit (in degrees) of the visual field becomes about four times larger for the 1 cm test object, whereas it becomes about seven times larger (extremely high value) for the 0.5 cm test object.

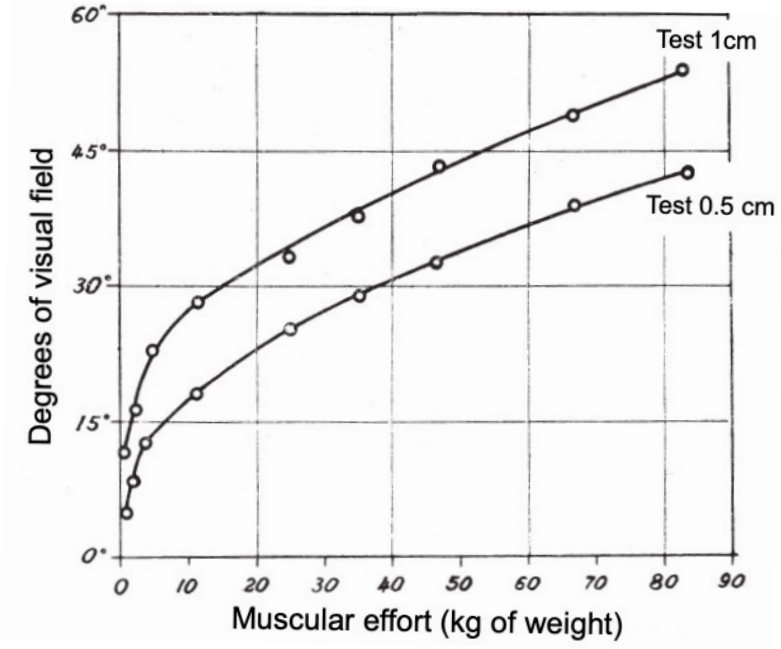


Figure 45. Enlargement of the visual field by facilitation. Degrees of the visual field of the right eye of subject M as a function of the muscular effort measured in kg of weight held by subject M, with white tests of 0.5 cm and 1 cm in diameter.

The experimental data in the curves are the average of several determinations, since the conditions of the experiment (fixation at the central point and attention to the peripheral vision, in addition to the considerable effort to hold very high weights) do not allow very precise assessments. However, they are good enough for our current aim of graphically expressing the course of the visual field enlargement as a function of facilitation by muscular effort.

These facilitation curves are similar to those shown in Fig. 44 on recruitment of peripheral degrees by intensification of the stimulus luminosity, but in this facilitation experiment, the stimulus remains unchanged. The fact that it can be perceived in increasingly peripheral areas because of an increase in their sensitivity, is due to the central summation produced by the muscular effort.

9.3 Visual field organization

The visual field represents a functional unit which is globally impaired in the central disorder studied here. Only superficial and incomplete determinations can lead to the idea that only peripheral regions are altered in concentric reduction. Since the impairment is general, the visual field reduction detected by perimetry using a standard test object can be sufficient to presume to what extent both the more central regions and those beyond the limit of vision that might be believed to be blind, should be affected. There is an alteration in excitability throughout the visual field, but the more peripheral the regions, the greater their deficit. For this reason, we can consider a dynamic reduction in which excitability is modified throughout the entire functional field, although with the indicated degree of diversity. This means an increase of varying intensity of the excitability differences (intervals) that already exist from one region to another in a normal individual.

In relation to the unitary functional character of the visual field we can cite the remarks of Pppelreuter (1917). This author points out that there are no absolute scotomas in the visual field as a consequence of cortical lesions, so it is sufficient to increase the intensity of a luminous stimulus to reach vision residues in regions where they would otherwise appear blind. In concentric reduction, the field expands by increasing the luminosity of the test object; also in hemianopsia, this author found that the "healthy" half does not remain absolutely unharmed and may show a certain degree of concentric reduction, or in any case a reduction of color isopters. There is thus a functional unit in the activity of the visual field. However, more specific considerations on excitability or more varied experimental analyses are not mentioned by this author.

Other phenomena, which we will only mention now, also reveal the dynamic peculiarities of the visual field, i.e the interdependence between its parts. Such phenomena are monocular *polyopia,* macular deviation (*pseudofovea*), *autokinetic* phenomenon, *metamorphopsia* (distorted vision), etc., all of which are manifestations we have been able to observe in the subjects studied here and in many others. Very remarkable and of great theoretical importance is monocular polyopia, which we have studied for the first time in an injured subject with parieto-occipital lesion at the end of

1936, and later in some other cases. In 1938 we determined the physiological laws of the phenomenon, which allowed us to collect a large number of cases with brain injuries and lesions of other nature. The cases described of polyopia are very few in total, having been reported by very few authors (Goldstein 1939, Quensel 1927, Pötzl 1933, etc.). However, although very few patients describe the disorder spontaneously, when attention is paid to certain circumstances of the visual field impairment, the phenomenon can be brought to light in a large number of cases in a constant and regular way. Some hypotheses about the occurrence of the phenomenon have been formulated by Pötzl (1933), and more positive interpretations by Goldstein (1939), however, these interpretations are not sufficient to explain the most complex variants of polyopia that we found. For the moment we shall only indicate that changes in the localization of stimuli occur, and a single stimulus in monocular vision may cause multiple localization due to an anisotropic visual field configuration.

Polyopia in the M case is shown to be related to the intensity of the luminous stimulus; the weaker the stimulus the more intense the monocular polyopia, and facilitation reduces it. With a stimulus of suitable intensity, the following three types of vision are obtained in subject M as well as in many other subjects:

1) The test object in central vision gives rise to triplopia; the real stimulus is seen in the middle, and somewhat less intense virtual images are seen on both sides.

2) In peripheral vision, only diplopia (double vision from a single stimulus) is obtained, with the virtual image less peripheral than the real image.

3) In a paracentral region at a certain distance from the central point of the visual field, a single image is obtained from a single stimulus, so there is a region without polyopia.

In subject T, polyopia is not very pronounced at present but it was very pronounced when we first studied this subject in 1938, allowing us to unravel to a large extent the mechanism of this phenomenon. At present, the virtual images in this subject are reduced to faint shadows; apart from this, he complies with the general rules set out in the previous case.

The phenomenon of macular deviation can be easily studied in subject T due to the greater extension of his visual field; the central vision, with maximum visual acuity, can be moved to about 15 degrees under certain experimental conditions that we cannot detail now. Fuchs (1922), a psychologist like Gelb and collaborator of Goldstein at the Brain Injury Research Institute during World War I, described the formation of a pseudofovea in the healthy half of the visual field of hemianopics. We found in 1938-1939 that, without the need to have hemianopsia, many subjects with concentric reduction of the visual field show a certain deviation of the foveal vision that does not correspond to the anatomical fovea, this deviation being much more pronounced in certain experimental circumstances. Thus, the entire functional layout of the retina is altered, relocating its functions (colors, luminosity, acuity, etc.) to regions that do not normally perform them.

We can also mention metamorphopsia (alteration of the shape of objects) and the autokinetic phenomenon consisting of the apparent motion of motionless light stimuli. This last alteration was initially extremely pronounced in subject M, and currently is only minimally displayed by means of low-intensity stimuli. All these peculiar manifestations

of the visual field, along with other cases, will be exposed in specific publications. We only mention them here to emphasize the functional complexity and nervous dynamism exhibited by the visual field, which seems so simple and static structure.

10 Flat colors. Visuospatial localization

10.1 Flat color vision

Besides the chromatic sensory disorder, our subjects present a singular chromatic spatial alteration: *flat color* vision. This is a color "irradiation"[7] that makes objects appear wrapped in a colored atmosphere, so that when an object is grabbed or a colored surface is touched with the hand, the subject has the visual impression that his hand is sinking into the color. Colors therefore do not seem to be adhered to the surface of objects, the vision of a colored surface is lost and instead a color irradiation appears that tends to erase the relief and contour as well as the inclination of the surface, the color appearing on a frontal plane (flat color vision).

This disorder is extremely pronounced in subject M and was one of the first notable phenomena found in this subject in the summer of 1938. It is also present in subject T but less markedly so according to his less severe brain lesion.

This disorder was first described by Gelb (1920) in the work entitled *Über den Wegfall der Wahrnehmung von Oberflächenfarben* (On the loss of surface color perception), when he was working with Goldstein at the Institute for Brain Injury Research. In this work, the loss of surface color was studied in two subjects with a parieto-occipital lesion. They presented flat color vision in accordance to the research of Katz (1911/1930/1935). From then until now, although some authors have dealt in passing with somewhat similar disorders (especially color irradiation), the flat color disturbance has not been researched. Gelb's descriptions are entirely from the psychological point of view. But here, in our subject M, who presents a much more intense chromatic spatial alteration than the two cases of Gelb, we shall address the disorder in a direct and simple way on a strictly physiological basis, showing that flat color vision is the result of a visuospatial localization disorder. Something similar occurs also in touch in these subjects, and could be called flat touch or irradiated localization due to the loss of precise localization. As said, both sensory systems are disturbed to the same degree in the central syndrome, therefore, in the case of spatial localization, the alterations that we now study in the visual system will be found again in a more or less similar way in the tactile system.

As for the patients of Gelb (1920), in addition to flat color vision, they also presented a concentric reduction of the visual field, a decrease in acuity, less adaptation to light, color weakness and severe visual agnosia; and merely the type of visual field alteration

[7] It must be understood as a diffuse spatial localization of the color of an objet.

is enough to ascribe them to the central syndrome. They are therefore types of brain disorder of the same type as that of our two subjects and subject Schneider, and as for their intensity, already Gelb indicates that they show a much less pronounced agnosia than that of the Schneider case studied by this author together with Goldstein (Goldstein and Gelb 1918, 1919). Indeed, in the most pronounced case of flat color vision, the concentric reduction is somewhat less intense than in Schneider, and can be equated with that of case M under facilitation by maximum muscular effort. Thus, that case shows a much more intense brain alteration than that of our second case, subject T. Since these patients of Gelb can be included in the central syndrome, they would have all the characteristics studied (alteration in all sensory systems in both halves of the body, asynchrony, permeability to summation, etc.). Flat color vision is only one aspect of the visual system disorder, and this in turn is a part of the brain disorder that necessarily involves other sensory systems.

Concerning the interpretation of this spatial color disorder, other authors (Pötzl 1928, Stein 1928, 1930, Kleist 1934) besides Gelb (1920) have discussed it without achieving a simple and satisfactory explanation. In general, the disorder is assumed to be dependent on both a sensory impairment of color vision and an associated apperceptive visual agnosia; all interpretations being made from a psychological standpoint and discarding the possibility of finding a physiological basis for the disorder. In this brief study, we shall prove that the pathological flat color vision results simply from a disorder in visuospatial localization, more specifically, from the loss of the normal precise localization, which is replaced with a diffuse or irradiated localization, in the same way that we will have the opportunity to study it in the tactile system.

10.2 Spatial localization disorder

Normally, colors and luminosities occur under various spatial aspects. When colors are adhered to objects and surfaces, they appear as *surface colors* with a defined location. In contrast, there are other situations such as the colors in the spectrum, the blue of the sky, etc., that show a less precise spatial location, they appear to us as in a plane, hence their *flat color* character (Hering 1880, Katz 1911/1930/1935). When this characteristic increases, they acquire an aerial character, and flat colors become *spatial colors* appearing as colored masses or fogs. As already mentioned, the pathological disturbance in our subjects lies in the fact that surface colors (with well-defined location) are lost and it is not possible to perceive them other than as flat colors.

10.2.1 Properties of the pathological flat color vision

In Gelb's patients, the darker the colors were, the more swollen they became, and this also happens in our two subjects, especially in subject M, who will be our main reference. When colors are detached from objects, they show a soft character that makes them penetrable, so that when touching a colored surface, the hand sinks into the color and gets tinged with it. Following the nomenclature used by the patients of Gelb, colors can be

classified into *thick* and *thin* according to their penetrability, so that the colors less luminous “irradiate” much more than the clearer ones. The former, besides easily tinting the objects in real contact with them, produce a sensation of certain softness to touch, whereas thin colors tinge much less and are harder to touch. In the M case, red is the thickest and softest color because it irradiates to the maximum, and white, which is perceived as light green, is the one that irradiates least. In between these two colors there are all the others regarding their irradiation capacity.

The color detached from a piece of colored cardboard is perceived on a frontal plane even when the cardboard is inclined with respect to that plane, unless the inclination is quite pronounced. The inclination necessary for this frontality to be lost depends on the color of the cardboard. Thus, red tolerates deviations of the cardboard from the front plane much more than yellow or white.

The concept and recognition of objects and forms is altered due to the disorder of irradiation, although they are already intrinsically altered in our subjects as we shall see later. The detachment of colors from objects tends to suppress the relief and contours of them, making their form more uniform and simple.

When writing with a pencil on white paper, our subject M has the sensation that the tip of the pencil is sunk in the light green of the altered white. The tip is as if impregnated with color, which being light color, its irradiation (delocalization) is very weak. If the pencil strokes are thin, they disappear under the irradiation and no difference with the background is perceived.

Subject M has the most intense color irradiation anomaly known so far, which decreases considerably under very strong illumination. In ordinary illumination and at the accommodation distance, the irradiation is so manifest that even under the most intense facilitation it cannot disappear from the less light colors such as red, instead it disappears for the other colors. In the other subject T, irradiation is very small and only pronounced in red.

10.2.2 Irradiated localization

The above phenomenology depends on the conditions of color irradiation, which varies greatly with excitability, and this in turn depends on the particular color, its illumination, exposure time and facilitating action.

When subject M is shown cards of different colors, and is asked to sort them according to the intensity of irradiation, he does it as follows (from highest to lowest irradiation): red, green, blue, yellow and white. All these colors irradiate upwards or towards the subject who is looking at the card, and also towards the sides, although somewhat less. With regard to black, which looks as a dark, somewhat greenish fog, he has the sensation that instead of irradiating upwards, the color is as if it were sunken, i. e., irradiating towards the bottom and in a slightly greater proportion than red. This distal character of a black surface's irradiation may be a secondary subjective alteration, and if we discard this peculiarity of black, it can be stated that the degree of irradiation depends on color luminosity.

Frontal irradiation is much more intense than lateral irradiation, the latter being about one third of the former. By measuring the irradiation of colors according to the indications of subject M at a distance of 25 cm and in medium illumination, the values obtained are shown in Table 11.

Table 11. Spatial irradiation of colors in the M case in the inactive state, using colored cards 20 × 20 cm.

Color	*Frontal irradiation*	*Lateral Irradiation*
Red	6 cm	2.5 cm
Green	4.5 cm	2 cm
Blue	3.5 – 4 cm	2 cm
Yellow	2.5 cm	1.5 cm
White	2 cm	0.8 cm

Under these conditions and applying facilitation by means of maximum contraction of the entire musculature, irradiation decreases considerably to about one fifth, and thus the red irradiation is only about 8 mm.

A little more irradiation is perceived using only one eye than using both eyes, but in these tests the differences are small and the most noticeable change is obtained by going from the inactive state to the facilitated state.

Subject T presents this anomaly to a much lesser degree and only perceives irradiation when looking at the object with the right eye, the one with worse vision. Irradiation intensity in the T case is approximately similar or somewhat lower than in the M case under facilitation. Subject T also sorts colors according to their irradiation, similar to M, from highest to lowest as: red, blue, green, medium gray, yellow and white. As he sees blue much more intense and saturated than subject M, he places it after red. As for black, he insists on placing it between green and medium grey. It should be noted that the irradiation appears only to the right side of the visual field, as it happens with green chromatopsia, since the right half is somewhat more amblyopic. In binocular vision, the irradiation practically disappears due to the mutual facilitation between the two eyes. As said, irradiation is perceived mainly in the right half of the visual field of the right eye, and the more peripheral the vision, the greater the irradiation, which causes that a completely flat red surface is perceived as quite concave on the right giving the sensation that it is raised by that side.

We already know about subject M that, from the chromatic series including white, he only perceives red, and all the other colors are perceived as greens of varying lightness: he sees blue as a pale green, yellow as a paler green, and finally, white as the palest green. Therefore, we can say that the lower the lightness of a color (i.e. the more saturated it is), the larger the color irradiation will be; hence the larger irradiation is obtained for red. Likewise, irradiation occurs in subject T for whom the most saturated colors (least pale) are red followed by blue. We must therefore admit as a general rule in these subjects that the spatial localization of the different colors is altered according to their lightness. According to Katz (1911/1930/1935), a factor such as the intensity with which attention

is awakened may be involved in the irradiation perceived; but according to what is said above, such a factor should be the intensity of color saturation and not its lightness. Also, when a normal subject looks at the spectrum, he already perceives a more aerial character at the more saturated ends of the spectrum, red and violet, whereas this character is minimal at yellow.

Color irradiation changes significantly depending on the stimulation conditions. It is enough to move away a piece of colored cardboard, for irradiation to increase progressively both frontally and at the edges, a fact already observed by Gelb (1920), although he mentions it briefly without giving it any more importance. It is clear that in this case there is a decrease in the intensity of the stimulus, thus the disorder of spatial localization becomes more acute, i.e. irradiation increases. This change can be observed both by moving the colored surface away from the subject or by diminishing the illumination of the colored surface while it remains at the same distance. In this last case, varying degrees of irradiation then arise successively: almost normal surface color vision is obtained under very strong illumination, color irradiation (flat color) is obtained in medium illumination, and aerial color with a very diffuse spatial localization is obtained in very weak illumination. In this last stage, it is no longer possible to distinguish the frontal from the lateral irradiation, since everything looks like a colored aerial mass much larger than the real colored surface. By diminishing the illumination even further, the color, which had already become fainter, disappears completely and is replaced by a kind of dull fog that fills the entire visible field, being much more extended than the preceding colored mass.

In this way, a joint variation in the intensity and extension of the color is seen when the stimulation intensity changes. When the colored surface is strongly illuminated, its color is perceived intensely and as a surface color, and when the illumination decreases, it appears more and more weak and with a flat character, and finally spatial or aerial, that is, as spherical. At this point, we can only consider the volume of the color, so it is not possible to distinguish any differentiation or spatial organization, the localization has been completely lost, and this happens when the color is about to disappear completely and be replaced with a simple colorless fog more or less dark. It is important to note that in the irradiation of flat colors, the lateral and frontal irradiation zones show a less intense color tone than the real colored surface.

Even in medium illumination, if the exposure time is very short (one second), a strong irradiation (i.e. spatial color) is obtained in the M case, and equally in the T case but to a lesser degree. If the exposure time is prolonged, the irradiation decreases significantly. Therefore, it would be possible to determine excitation (strength-duration) curves corresponding to the different levels of visuospatial localization: one curve for spatial color, another for flat color and another curve for surface color. Thus, we have different types of spatial localization: from the complete loss of localization in aerial (spatial) color to the total localization in surface color, passing through an intermediate state of diffuse or irradiated localization corresponding to flat color.

Under facilitation by maximum muscular effort and in ordinary illumination of colored cards, irradiation disappears only in the lightest colors: first in white, which even without facilitation presents very weak irradiation, and also in yellow, etc. Instead, a small

irradiation always remains in red, which only tends to disappear under strong illumination and facilitation. In average illumination and in the inactive state, when small objects are placed on a red paper, the frontal irradiation in which they are immersed makes them almost disappear. If facilitation is then applied, the irradiation decreases considerably and the objects become visible because the *thickness* of the color decreases.

Due to the different irradiation of different colors, changes in shapes or configurations can be perceived, depending also on the stimulation conditions. For example, let us take two partially overlapping squares, one red and one white (Fig. 46) with the white over the red. In weak illumination, subject M sees a full red square on the white one, that is, the corner of the red square completed by irradiation covers the corner of the white square that irradiates much less as shown in Fig. 46 a). If the illumination is high enough, the subject perceives then the real configuration and the white square appears complete [(Fig. 46 b)], covering the corner of the red square, since the weak irradiation of red in strong illumination does not achieve to complete the shape of the red square. These changes are also obtained by means of facilitation, without altering the illumination; thus, in weak illumination, if the subject is in an inactive state, the red square is seen as complete, and under facilitation it is seen incomplete and the white square complete.

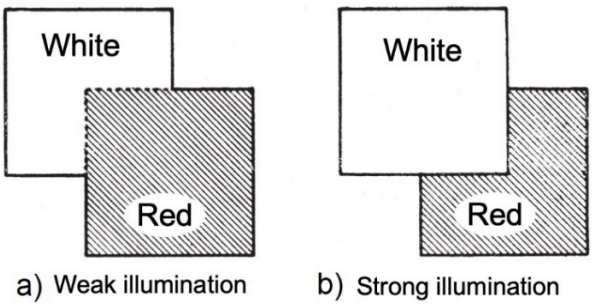


Figure 46. Completing the shape of the red square by irradiation (see text).

The best way to complete the shape under the indicated conditions is by combining a strong irradiation color with a very weak irradiation color, and provided that the area to be completed is not excessively large. Then, the stronger irradiation color tends to complete the shape of the square because this shape is more regular - or of greater meaning or pregnancy [8] according to Gestaltists - than the configuration based on right angles that results from covering the corner of the square.

Since irradiation alters spatial localization, it also modifies the perceived size of objects. By moving the object away or decreasing its illumination, the diameter of the test object tends to diminish as the lateral irradiation increases. This is because the irradiation has a weaker luminous intensity that allows it to be distinguished from the innermost real

[8] Pregnancy (Prägnant in German) or "good form" is a concept introduced by the Gestalists and refers to the quality of the visual forms that capture the attention of the observer because of their simplicity, balance, concision, accuracy or stability of their structure.

area. When illumination is quite low or the distance very long, the flat color is lost and evolves to spatial color, any distinct perception of the object disappears and there is only a diffuse irradiation over a much larger extension than that of the object.

10.2.3 Asynchrony between simple sensation and spatial localization

As already mentioned, pathological flat color vision is only the effect of an irradiated spatial localization caused by the failure of the normal localization. The latter can be achieved by intensifying the visual stimulus (e.g. by increasing light intensity). For this reason, light colors produce much less irradiation than more saturated colors (less whitish) under the same illumination conditions. For all colors, irradiation is weakened under strong illumination, and also by facilitation which reduces the asynchrony (or separation) between simple visual sensation and its spatial localization. Irradiation (diffuse spatial location) occurs in both vision and touch since the characteristics of this functional disturbance are the same for both sensory systems. In both systems there is a remarkable interval between minimal visual or tactile sensation and spatial localization. Weak stimuli that in a normal situation are always perfectly localized, are left without localization in our subjects, giving rise to gusts of tactile sensation without spatial character, or to aerial colors in vision. In more intense stimulation, a diffuse (irradiated) localization occurs, which is a rudimentary undefined spatial localization and occupies a certain area. Finally, in very intense stimuli, the normal defined and precise localization occurs. Irradiated localization in touch extends not only on surface but also in depth; the subject has the sensation that the stimulus reaches the deep layers of the skin, in the same way as in frontal irradiation of flat colors, which alters the perception of depth.

Spatial localization, being a more complex function that requires a greater physiological demand than simple qualitative sensation, is altered in our subjects in a much greater proportion, and is therefore delayed presenting a much higher intensity threshold. Then a disaggregation occurs, and alterations appear such as flat color vision, etc., alterations that we shall return to study quantitatively in a more precise way when dealing with tactile functions. Thus, flat colors result from a color-spatial asynchrony (disaggregation) leading to an abnormal interval. This disaggregation is governed by a mechanism common to all activities: excitability deficit but in greater proportion in the more complex ones.

We have also observed some weak irradiation in other brain-injured people with reduced visual field or other alterations in the visual field, the irradiation being especially towards the peripheral regions of the visual field, as in the T case. In a normal individual, traces of flat color can also be noticed in the outermost zones of the visual field. During the phase of recovery from the disorder in the two cases of Gelb, the irradiation disappeared first in the central zone, and much later, it was attenuated in the peripheral zones.

As a complement, it is interesting to mention that in a normal individual, pathological flat color vision appears under the effect of mescaline (Beringer 1923, 1927), a drug that significantly decreases brain excitability. In this case, colors seem swollen and penetrable, wrapping objects in an atmosphere of color. However, the transient conditions of

intoxication and the irregularities that occur are not as conducive to research as are brain-injured people, who show a more constant and uniform symptomatology

11 Visual form perception

11.1 Visual acuity

In the study of visual forms, we include visual acuity, visual perception of motion and perception of object shapes and visual configurations.

Visual acuity is a complex function of spatial discrimination of stimuli (minimum separable) and already includes aspects of shape perception. Visual acuity is extraordinarily altered in the two cases we are studying, especially in the M case. Due to the diffuse localization of visual stimuli by the irradiation mentioned above, it is easy to understand that spatial discrimination between stimuli is very difficult. A rather poor function can only be expected under intense illumination or by facilitation through muscular effort or other types of summation. In the usual clinical tests using optotypes, the visual acuity values obtained in subject M for different types of vision are shown in Table 12. Very different degrees of acuity are obtained depending on the types of vision. These types are determined by the facilitation due to muscular effort, by the joint action of the two eyes facilitating each other, and also by the small difference in vision between the two eyes. By combining the two types of facilitation, i.e. that of maximum muscular contraction and that of binocular vision, considerable increases in acuity are achieved, which can be as much as six times the value of the weakest eye in the inactive state.

Table 12. Visual acuity in both eyes of subject M in full sunlight.

Type of vision in subject M	*Visual Acuity*
Left eye, inactive state	1/25
Right eye, inactive state	1/16 -1/20
Left eye, facilitation by muscular effort	1/16 -1/20
Binocularly	1/10
Right eye, facilitation by muscular effort	1/8 -1/10
Binocularly and muscular effort	1/6 - (1/4 ?)

If the illumination is lower, but still sufficient to examine a normal individual, the acuity values in subject M decrease significantly, to more than half of the above acuities. Under intense illumination (as indicated in Table 12), it is still possible to obtain further improvement in acuity if, in addition to the binocular effect and maximum muscular

contraction, the subject also receives intense light over his own eyes. Then it is easy to reach acuity of 1/4, and if he is additionally told to make vigorous movements of his head, limbs, etc.,1/3 is reached. In this case, several types of facilitation (binocular vision, muscular effort, movements, intense light on both the test object and on both eyes) are combined. When determining visual acuity with the letter test or other optotypes, if the test is not seen with sharp vision, the test is perceived to be somewhat tilted due to the orientation disorder that leads to inverted or tilted vision. In sharp vision, the test object is seen in the correct position, but if the test object is smaller, it is perceived as both blurred and tilted, and so on for even smaller test objects. The T case behaves like the M case, but as the brain deficit is less intense, the various acuity values, shown in Table 13, are much more favorable.

Table 13. Visual acuity in both eyes of subject T in good illumination.

Type of vision in subject T	*Visual acuity*
Right eye	1/3 - 1/2
Left eye	1/2 - 2/3
Right eye, facilitation by muscular effort	2/3
Left eye, facilitation by muscular effort	2/3 – 1
Binocularly	2/3 - 1
Binocularly and muscular effort	1

Subject T behaves similarly to subject M, for example, with the right eye, the one with weaker vision, optotypes corresponding to visual acuity 1/3 are seen sharp and correctly oriented, whereas those corresponding to 1/2 can be recognized and read but are somewhat blurred and tilted. At the beginning of the examination of this subject, his visual acuity was 1/8 to 1/6 or even less; later, the disorders have regressed to some extent and his visual acuity has improved significantly. As for the Schneider case of Goldstein and Gelb (1918), acuity values are indicated as essentially variable from one examination to another, since three different ophthalmologists found different data; one found normal acuity, another found it was reduced and the third gave 1/10. It is probable that the last one was the most accurate, that value being between that of subject M and that of T. It must be noted that the acuity values in the Schneider case can be affected by his characteristic head contouring movements which, as we know, are merely central summation phenomena, contrary to the assumptions of the mentioned authors. If such movements are missing, the acuity can be very poor, a fact pointed out by the authors who consider the patient to suffer from apperceptive visual agnosia, i.e inability to perceive shapes.

In ordinary life, such wounded people behave with much better visual acuity than one would expect from clinical trials, especially the more severely affected subject M. We have already seen the big difference between using only one eye in an inactive state and using binocular vision along with facilitation by muscular effort. In fact, in binocular vision and good illumination, some muscular tension and small movements are sufficient to obtain an acuity within fairly usable limits. As is known, visual acuity in a normal

individual is already markedly influenced by illumination, growing proportionally to the logarithm of luminous intensity. The curves of acuity as a function of illumination for the two subjects we are studying have the same general characteristics as for a normal subject, but with very different values according to their different degree of brain alteration. We determine for central vision, the minimum separable as a function of illumination, and we obtain visual acuity by taking the reciprocal of the minimum separable.

In the curves of the minimum separable (Fig. 47), it can be seen the considerable difference between the values of the pathological cases and that of a normal subject, for a given intensity of illumination**.** The more pronounced the nervous excitability deficit, the greater the difference. The bending of the curves indicates the degree of functional variation; the more pronounced the brain disorder, the lesser the degree of functional variation.

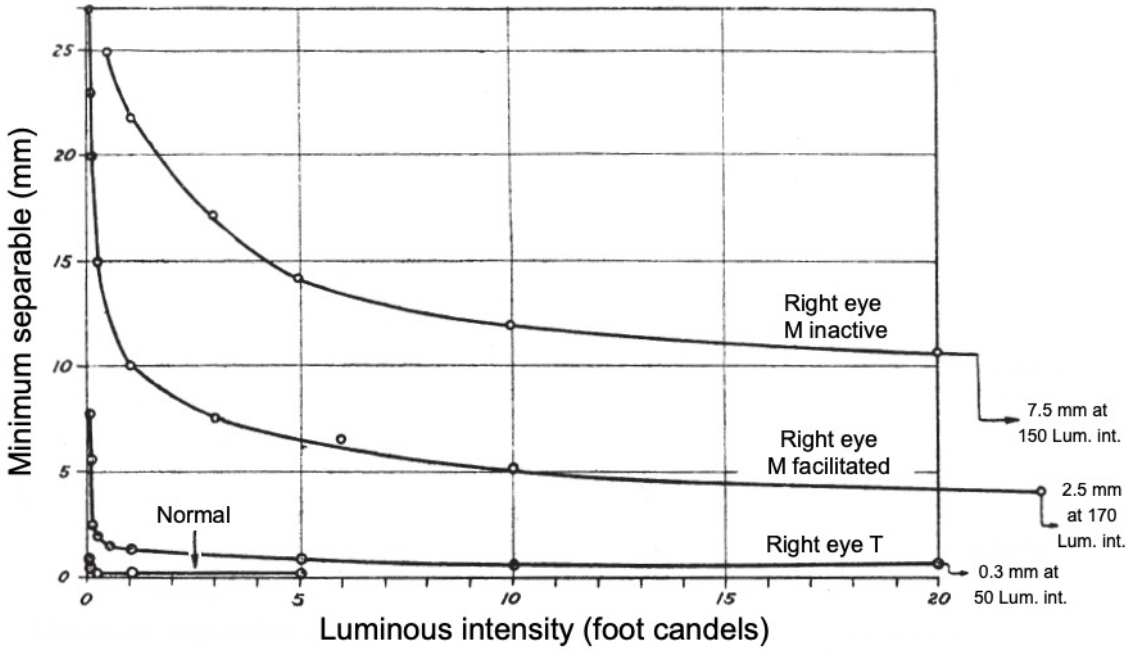


Figure 47. Minimum separable as a function of luminous intensity, in central vision of the right eye of M inactive, M under facilitation, T inactive, and a normal subject.

Taking the reciprocal of the minimum separable, the corresponding curves of acuity as a function of luminous intensity are obtained, shown in Fig. 48 where some corrections have been made to show all the curves in the same graph. It can be noticed in these acuity curves that, for the luminous intensity at which the normal subject reaches acuity 1, the pathological cases show extremely low acuities that only improve very slowly along the curve, and extremely high intensities are necessary to achieve a more favorable discrimination.

Finally, taking the logarithm of the illumination, more complete curves of the variation of visual acuity in the different cases are obtained (Fig. 49). For the right eye of subject M inactive, the acuity increases up to 1/25 for maximum illumination, and up to

1/12 applying facilitation by muscular effort. Subject T inactive, in strong illumination but much lower than in the previous case, reaches an acuity of 1/2. Also noteworthy is the different luminous intensity at the lowest values of acuity in the three pathological functional variants (M inactive, M under facilitation and T). The curves of Fig. 49 tend to become straight lines, and for the middle values of acuity it can be said that acuity grows proportionally to the logarithm of illumination.

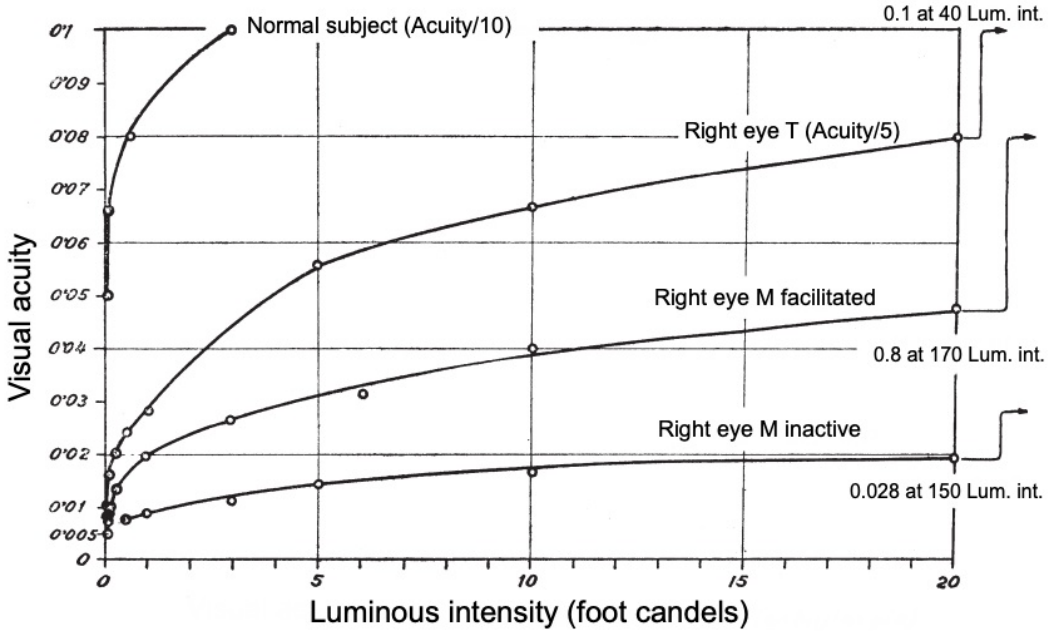


Figure 48. Visual acuity as a function of luminous intensity. Acuity is here the reciprocal of the minimum separable (see Fig. 47). The acuity of the normal subject has been divided by 10 and the acuity of subject T by 5 to represent them on the same graph.

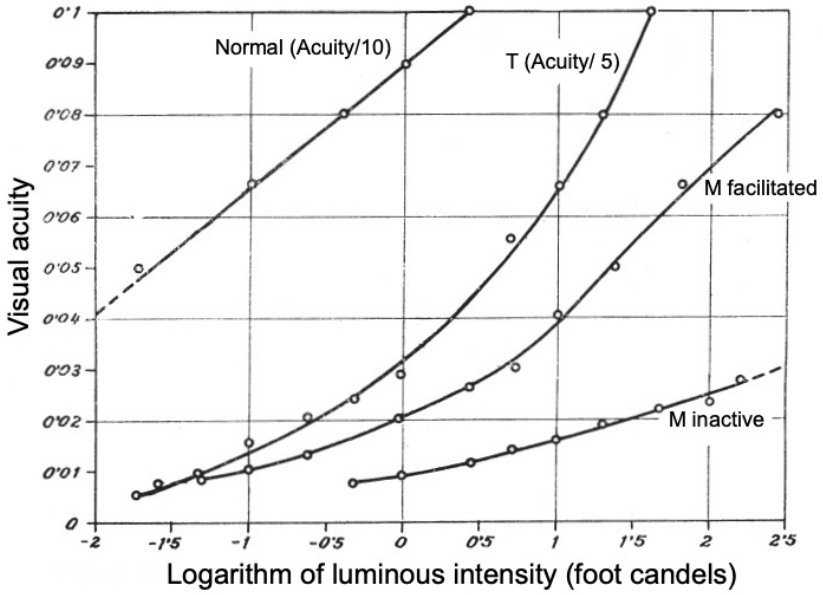


Figure 49. As in Fig. 48 but taking the logarithm of luminous intensity.

These curves give a clear account of the status of the acuity function. In a normal subject, the variation is rapid even for small light intensities. In the pathological cases, especially in M, the growth of acuity is very slow and the curve for M inactive begins at high intensities due to the deficit of excitability in the lower threshold and also in the differential threshold, the latter due to an enlargement of the normal interval by the asynchrony already discussed.

If visual acuity is determined not only for central vision but also along the meridian of the visual field, with constant illumination, the so-called Wertheim curve (Wertheim 1894) is obtained, which shows that the acuity deficit extends to the entire visual field, as was to be expected. In subject M, not only the acuity corresponding to foveal vision is very low, but an equally intense decrease also occurs in somewhat peripheral zones, the curve ending very soon due to the concentric reduction of the visual field. A diagram of the behavior of the Wertheim curve in correspondence with the concentric reduction and the electrical excitability of the retina in the case M inactive and under facilitation is shown in Fig. 50. This highlights the functional dependence of acuity and visual field on the state of brain excitability. Such functionality must be assessed from a physiological point of view, dismissing the idea of preformed anatomical structures.

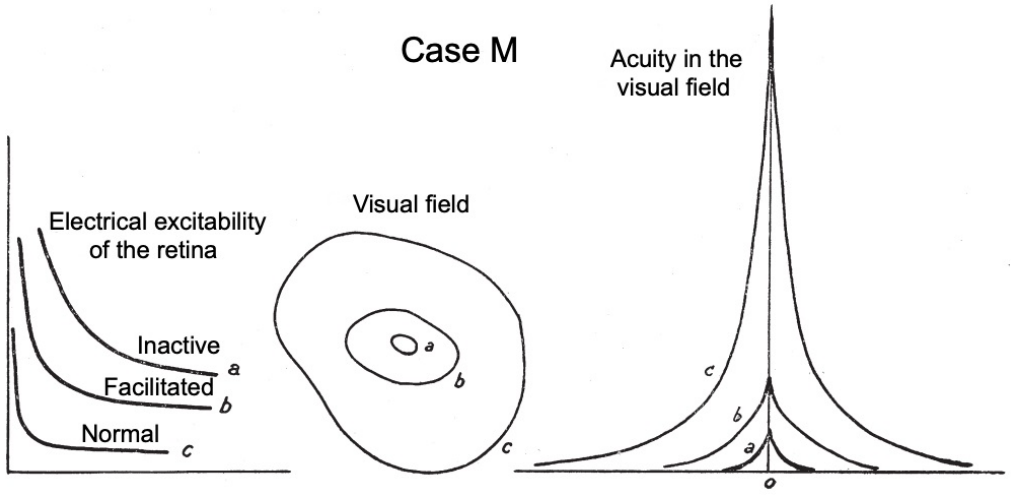


Figure 50. Schematic representation for the M case, compared to the normal one, of the functional correspondence between the visual field and the acuity in the field given by the Wertheim curves (right side), and the state of excitability of the retina (left side).

The mere fact that visual acuity in a normal individual depends so much on light intensity proves that acuity cannot simply be inferred from the anatomical arrangement of the sensitive elements in the retina (separation between cones, etc.). Helmholtz (1896) and later Hartridge (1918, 1922) suggested that visual acuity could be inextricably linked to intensity discrimination. Therefore, visual acuity must always be put in relation to the type of stimulation, or rather to the states of brain excitability.

It is understandable that in the above-mentioned tests and determinations, visual acuity (or minimum separable) is hampered in subject M by the pronounced spatial

irradiation described in previous pages. In fact, when this irradiation decreases under strong luminous intensity, acuity improves, i.e. the minimum separable decreases. The fact that visual acuity is reduced so considerably in these subjects, even in very intense illumination, is due to the character of the sensory disorder, which, being global, appears more pronounced in functions of greater differentiation. In short, there is an enlargement of the excitability intervals between the different acuity levels, leading to the exclusion of the highest levels (of highest acuity).

11.2 Alteration of visual motion perception

The loss of visual perception of motion was one of the first singular phenomena that subject M showed as a result of a careful examination, shortly after the finding of his inverted vision. It was precisely this alteration of visual perception of motion, already observed in 1938, that led us to search for some resemblance between M and Schneider. This type of disturbance is extremely rare, and cases of this type clearly detected to date would only be the Schneider case of Goldstein and Gelb (1918) and a much earlier case of Pötzl and Redlich (1911). The patient observed by the latter authors was a woman with an occipital lesion who, instead of perceiving the motion of an object, she perceived a series of objects; a sort of decomposition of the motion into successive, static images of the object along the path. In the Schneider case, the disorder appeared differently and the subject did not see moving objects, but perceived them either here or there, that is, at the departure and arrival points, lacking the sensation of displacement. When this singular alteration was found in subject M, he behaved very similarly to subject Schneider, but lacking at that time an appropriate understanding of the brain disorder, it was not possible to advance in the investigation of the phenomenon. In the two above-mentioned cases, the authors did not go further either. In the earliest case, the authors merely stated the disorder, and as for the Schneider case, the authors related the loss of visual perception of motion to apperceptive visual agnosia, searching for a primary basis for all this in the failure of the *transverse function* of Wertheimer (1912), whose proof of seeming motion was not obtained in this subject. This is perhaps the only time that Goldstein and Gelb got close to a physiological interpretation of the phenomena, although they did not go beyond suggesting it. In general, it can be said that the studies of these authors do not leave the field of psychology.

Subject M in binocular vision, at accommodation distance and in ordinary illumination does not perceive the gestural movements of a person in front of him such as winking, rapid opening and closing movements of the hands, etc. He does not perceive any change, which is understandable considering the brevity of these movements and his slow reaction time in all sensory processes. However, he can perceive these movements under facilitation by muscular effort, although normalization is not complete since, for example, very rapid winks may still be excluded from his perception

Subject T behaves much better in the visual perception of these types of movements, and it is necessary to resort to fast and very short movements to demonstrate that there is also a certain alteration in motion perception.

Visual perception of motion, like all other types of functions, is altered in such a way that it depends on the conditions of stimulation. For example, the motion of strongly illuminated moving stimuli can be perceived, whereas others of lower luminous intensity, which in a normal situation are perceived quite well, are totally excluded for subject M. And if in the latter case he perceives motion, the speed and length of the trajectory are altered. If a moving stimulus of suitable luminous intensity is observed from very close-up, its motion is perceived although only in the central region of the path. However, if it is observed from a slightly greater distance, any perception of motion disappears due to the strong decrease in light intensity according to the inverse of the square of the distance, besides the fact that the angle of vision is reduced.

All these variations are studied in a very simple way by means of a metronome, placing a small white disc of 1 cm in diameter at the end of the pendulum that moves according to the chosen frequency of oscillation. The regulation of the luminous intensity of the moving disc allows us to study quantitatively the excitability relationships for the perception of motion by subject M.

Under the indicated experimental conditions and lasting each pendulum oscillation 1 second, subject M in an inactive state and using only the right eye perceives the motion decomposed into various sensory phases of different excitation level, as shown in Fig. 51. The main phases from the simplest to the most complex are: Sensation of a motionless point in the middle of the oscillation zone; sensation of signs of motion only in the most central zone and with reverse direction of motion; finally, sensation of a larger path and normal direction of motion.

Since each oscillation lasts one second and the path is about 10 cm long, the average speed is 10 cm/s. To perceive a single oscillation (of 1 second) at a distance of 25 cm, very intense illumination would be necessary, and in general only a sensation of motionlessness is obtained even with high intensities, as shown by the lower curve in Fig. 51. The small disc is perceived static in the central zone of the path. Furthermore, it is devoid of any color, only a motionless light is perceived.

A second phase appears with signs of motion if the observation is prolonged. Thus, in less than two seconds (two oscillations), a sensation of motion appears at high luminous intensities, as shown in the intermediate curve of Fig. 51. If the illumination is reduced, it is necessary to increase the number of oscillations sufficiently to maintain the initial perception of motion. This intermediate curve of kinetic sensation is higher in the axis of light intensities than the previous one and is less concave, corresponding to a sensory phase of higher functional level. As for the concomitant color changes of the test object, when the slightest sign of motion appears, the white disc may still be colorless, but immediately afterwards it shows a dark greenish tint. The perceived motion in this phase is limited to the most central zone, showing a very short path and much greater speed than the actual motion, thus the speed is overestimated. Moreover, this motion presents the important peculiarity of being seen in the reverse direction. This is easy to verify since the speed of the metronome pendulum is slow enough and there is more than enough time

for the subject to verbally indicate the direction of motion in each oscillation. The reversal of motion is due to the special asynchrony in the brain mechanism of visual orientation, so that weak stimuli are seen in the opposite direction, and stronger ones in the correct direction. This will be discussed in subsequent chapters.

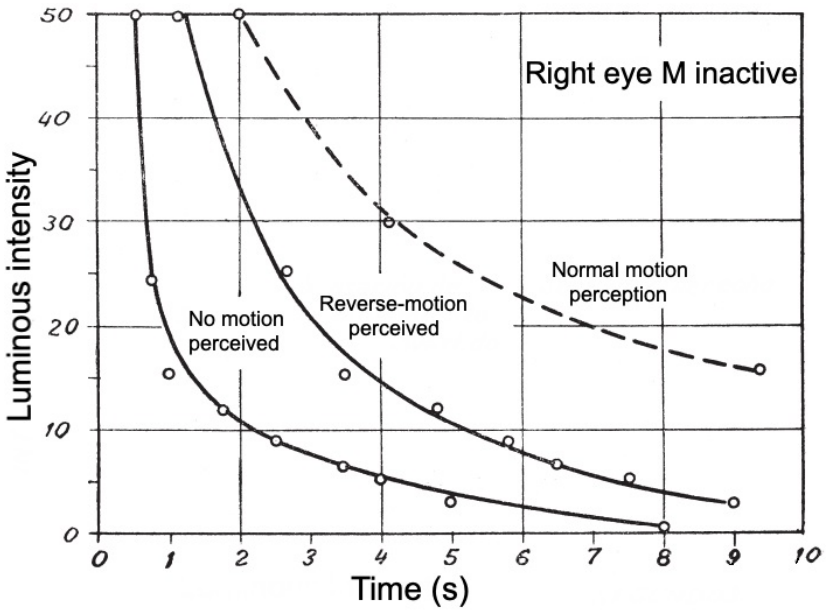


Figure 51. Strength-duration curve in the perception of motion by the right eye of M inactive. Test object: white disc 1 cm in diameter at the end of the metronome pendulum, at 60 beats per minute and at a distance of 25 cm from the observer. Lowest curve: only sensation of motionlessness in the middle of the path. Intermediate curve: very short motion with reverse direction. Dotted curve: larger and slower motion with normal direction.

Finally, it is still possible to obtain a third phase by appropriately increasing intensity and time of observation, which results in a motion perception closer to normal. In this phase, the perceived motion is of much longer path, slower, in the correct direction, and the color of the test object (the disc) becomes pale green with a certain amount of white. This last phase is the only one that resembles the motion perception by a normal individual, although it is still far from being identical. In fact, the amplitude of motion is smaller and the speed somewhat greater than for a normal subject. For a complete identification with a normal subject, it is necessary to apply facilitation, in which case the perceived motion reaches all the amplitude, becomes a little slower and the disc appears completely white.

These experiments indicate that visual perception of motion is extremely altered in the M case. However, motion can be perceived under special conditions such as high luminous intensity of the moving object, or also at lower intensities if the motion is observed for a sufficiently long time. For this reason, the motions that occur in ordinary life necessarily go unnoticed because they are below the threshold of necessary excitation since they are brief motions, and even in good illumination they are too brief to produce

any effect. It is therefore necessary to increase brain excitability by facilitation in order that motion can be perceived in some way.

The curves in Fig. 51 refer to vision in a single eye and in the inactive state, and similarly could be obtained for the facilitated state, in which case the separation (or asynchrony) between the phases would be much less. When the subject in the inactive state is in the first kinetic phase, it is sufficient to apply facilitation by muscular contraction to jump to the third phase or to a more normal stage. If the subject is in the second phase (slight sign of motion and inversion), facilitation action gets much more easily the change, i.e. re-inversion, path enlargement and speed reduction. This speed-reducing effect, which could be considered paradoxical, is not at all so if we consider the nature of the excitability disturbance. Because of the shorter perceived time, the motion seems faster. However, if perception is more favorable, either by simply increasing luminous intensity or by facilitation, the velocity decreases. Signs of this paradoxical effect are already present in a normal subject, thus for a constant speed motion, the perceived speed is higher when illumination decreases. Also in a normal subject, the same motion seems faster in peripheral vision than in central vision according to Exner (1886), which must be explained by a lower excitability in the peripheral regions of the visual field.

In the tests with metronome, in addition to facilitation by muscular effort, other weaker types of facilitation can be revealed, such as those produced by sensory stimulation of touch or hearing. Thus, when the subject is in the akinetic phase, a loud whistle next to his ear is sufficient for the motion to appear immediately, disappearing as soon as the sound stops. Likewise, by brushing the subject's back smoothly and with some speed, a similar effect on motion perception is obtained. The action of these facilitations is much less intense than that of strong contraction of the entire musculature, but in this type of experiment where there is little separation between the akinetic phase and that of signs of motion, these facilitations are sufficient to bridge the abnormal interval by means of central synchronization. Thus, an intersensory summation is obtained.

In relation to the luminous intensity of the kinetic stimulus, the effect of the different colors should now be considered. The same as discussed previously with regard to visual localization of colors in relation to their different spatial color irradiation according to their luminosity, is now applicable to visual perception of motion. The lighter a color, the better motion perception. For this reason, motion perception is easier with a white test object than with a red one. In this regard, the following test can be performed: a white test is placed on the metronome at a rhythm and distance suitable for reaching the threshold of minimum motion perception; motion is no longer perceived when viewed through a red glass, but is perceived again when the glass is removed.

As for subject T, since he presents a much smaller brain lesion and a correspondingly smaller alteration in excitability, he behaves normally with respect to motion perception in ordinary life, and only if illumination is very low, a deficit in his visual perception of motion can appear. In the metronome, very fast oscillations are necessary in order to stop them being perceived. Whereas a normal subject can see a 1/3-second oscillation very well, subject T fails completely when illumination is very low, using only the right eye (the worse eye) or the left eye. He may need three or four oscillations in monocular vision

or two oscillations in binocular vision to perceive motion. But this only happens in very low illuminations in which subject M would not even perceive a motionless stimulus. In the T case there are also changes in the perceived velocity according to the conditions of excitability, for example, in binocular vision and facilitation by muscular effort, the perceived motion is slower than that perceived only by the right eye and in an inactive state.

With regard to the Schneider case of Goldstein and Gelb (1918), the degree of his motion perception disorder is, as in all other functions, intermediate between that of the more severe M case and the milder T case, and must conform to all the conditions described for these cases. Thus, it is possible for him to perceive motion if the stimulation conditions meet certain requirements of sufficient intensity and exposure time. There is therefore no absolute loss of visual perception of motion as thought by the authors mentioned.

The origin of the disorder we are studying is the same as for other functions: because of asynchrony, activities of higher physiological demand are less accessible to ordinary stimuli and can only be awakened by more intense and prolonged excitation.

In the M case, for example, even in monocular vision and inactive state, a moving electric lamp observed at a short distance can be perfectly perceived even when the movements are rapid and of short duration. But apart from special cases like this, brief ordinary object motions under the usual much less intense illumination are discarded because of the extraordinary slowness of nervous reaction in this subject.

Stein (1928, 1940) already suggested that the loss of visual perception of motion must be related to an increase in sensory chronaxie, and we have experimental confirmation of this view in our cases. What in a normal subject is perceived as motion, is split into a series of stages in subject M: irradiated light, static localization, signs of rapid and inverted motion, and slower and larger motion. The greater the deficit of brain excitability, the more different (or asynchronous) are the excitability levels of these stages. As we shall see in the appropriate place, this alteration of the kinetic function occurs similarly in touch, both in joint movement and in the surface sensitivity when a stimulus moves over the skin. Likewise, a certain relationship could be established with the disorder of the perception of successive stimuli. The perception of motion not only implies perfect pinpoint localization, but also the ability to spatially discriminate between stimuli (minimum separable). It could then be possible to register a continuous series of very brief successive stimuli. This requires great sensory sensitivity; for this reason, motion perception disorder is one of the first and easiest to occur. It should be noted that motion perception is not an independent function as some authors have admitted, but, as is easy to understand, it is part of the process of discrimination and sensory organization, constituting one of its most complex stages. In the language of the Gestalt, the disorder we have studied would be explained by a destruction of the structure of the *field*, by the loss of coherence and the failure of the *transverse function* (Wertheimer 1912, Benussi 1914, Witasek 1910, Koffka 1919/1935, etc.). But this is a consideration from another point of view that leaves the physiological problem of excitability untouched and cannot directly address the quantitative characteristics of the disorder such as changes in strength-duration curves, acceleration of motion, reduction of trajectory, etc. In short, the

usual moving stimuli are not perceived because they are too fast and too weak in luminosity in relation to the excitability characteristics of the subject, especially subject M.

11.3 Visual perception of figure and object shapes

In view of what has been exposed in previous pages about visual acuity, one could infer in some way the behavior of our two subjects in visual perception of figures and objects. A very remarkable pathological feature that should be highlighted here is what we call *metamorphopsic pseudoagnosia*. By this name we refer to a special alteration of forms and configurations due to more favorable meanings that occur spontaneously because of the lability of the perception. Many examples can be mentioned about this type of disorder, but perhaps the simplest and most illustrative are found in the perception of letters. In the M case, it is frequent that a **C** is perceived as an **O** (shape totalization), a **J** as an **I** (simplification and symmetry), a large size **a** as two superimposed zeros, and an **S** as an **8**. It is not a simple alteration of visual acuity that changes shapes, but an instability in perception that tends to change the shapes into others of greater pregnancy (simpler and more meaningful) according to the laws of organization of the field of perception exposed by the Gestaltists. This type of alteration can considerably change the appearance of shapes and their meanings. For example, a calligraphic "A" large enough to be easily recognized (Fig. 52) is perceived by subject M as an "o" and an "A", as shown in Fig. 52. This disaggregation that occurs in this example is very typical and is observed quite regularly in all kinds of somewhat complex figures. Also noteworthy is the difficulty in perceiving sets in a unitary way.

Figure 52. Alteration of the visual perception of the figures in subject M. The calligraphic "A" (model) is perceived broken down into an "o" and an "A" as seen in the copy.

By using geometric shapes cut out from cardboard, it is observed that perception is guided by the most striking details such as angles, sharp points, corners, etc., and much less by sides and surfaces, so normal consistency and unity is not achieved. This is the reason for the distinction between geometric shapes often fails, and different shapes appear to be equal when they are perceived simultaneously. For example, an oval and a circle 3 cm in diameter, or a circle and a square, appear to be equal to subject M when shown to him at the same time, but he perceives them correctly when shown separately. The perception of shapes is carried out in a diffuse and unstable way, being subjected to many circumstances that significantly alter the perception of shapes; in addition, the

slowness in perception is considerable. This type of perception is common in the M case in the inactive state, in medium illumination and at the visual accommodation distance. Also subject T showed this kind of perception when he began to be examined in 1938 shortly after suffering the brain injury, but nowadays he does not show such a marked deficit and only by conveniently shortening the exposure time he makes the same mistakes as M. Subject M does not make mistakes only if he appeals to facilitation, which not only greatly reduces the reaction time but also makes the above-mentioned phenomena of alteration of shapes disappear, thus the distinction between shapes can be made correctly. The various errors in shape perception by subject M in an inactive state, which at least appear at the beginning of the test and disappear only with very prolonged attention, never appear under facilitation by muscular effort.

In the Schneider case of Goldstein and Gelb (1918), the perception of shapes was completely different if they were shown in the tachistoscope very quickly or under ordinary conditions. In the first case, vision was blurred and shapeless, only diffuse color spots, whereas in the second case the perception and recognition of shapes was quite feasible. This difference is based on the different exposure time of the stimulus, and since his reaction time is increased, there is not enough time in the tachystoscopic exposure for the shape sensation to develop, being reduced to the primary stage of shapeless spots. However, the mentioned authors think that there is no possibility in tachystoscopic vision for the patient to make his characteristic head movements and therefore shape recognition is not possible. The movements of the head have effect although not in the sense admitted by the authors but by the central summation action as we know. Without any kind of movement, it is sufficient to lengthen the observation time enough or to increase the illumination or the size of the object for normal perception to emerge. For example, subject M under facilitation by maximum muscular effort can well recognize figures of a convenient size with good illumination in a short time (even tachistoscopically in 1/5 second), whereas in the inactive state he would need up to 3 or 4 seconds. But in medium illumination, he may need much more than 6 or 7 seconds to recognize very simple shapes that a normal subject recognizes in about 1/10 second.

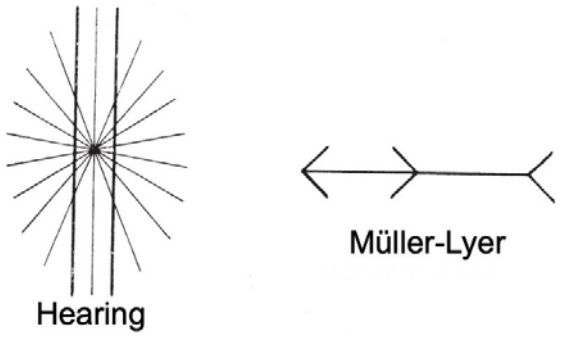


Figure 53. Geometrical-optical illusions of Hearing and Müller-Lyer, not perceived by subject M in an inactive state.

In relation to shape perception, the behavior of these subjects is of interest when they are faced with geometrical-optical illusions that occur in normal individuals and that here,

especially in subject M, hardly take place. Thus, in the well-known Müller-Lyer (1889) figure (Fig. 53), the optical illusion that the line segment with open ends seems longer, is very difficult to perceive, and only after a long time of very careful attention is it perceived, although not steadily. In other more complex figures such as the figure of Hering (1861), the illusion of curvature of the parallel lines never occurs in an inactive state, but is very well perceived by means of facilitation; the same is true for the above-mentioned illusion or other similar illusions. Geometrical-optical illusions do not manifest themselves in the inactive state because perception probably does not occur with sufficient unitary character or coherence, perhaps also because of the lack of precision in shape discrimination. In short, the perception of shapes without facilitation is always more or less diffuse or labile, there is not enough coherence and there is a tendency to alter the shapes.

Similar characteristics to those described for figure perception can be found in object perception. Object perception is very slow in our subjects, even with good illumination and an adequate size of the objects, because it is a complex process which develops less directly than in a normal individual. These subjects usually show a peculiar inability to perceive an object as a whole, and often have to resort to an indirect way of comprehension by means of an analytical-deductive procedure. The perception of an object is not carried out at once, but these subjects carry out a kind of *reading of the configuration (constellation)* of the object; that is to say, the perception is carried out by paying attention successively to the diverse specific aspects of the object in order to subsequently achieve its comprehension in a deductive way. Often the interpretation is only approximate when the objects are little known, but in general it is correct. Goldstein and Gelb (1918), in their case Schneider, have been the first to study this type of indirect perception which they put in relation to the difficulties of shape perception, but more accurately should be considered as the result of the inability of simultaneous complex recognition, as happens, for example, in Wolpert's simultaneous agnosia (Wolpert 1924).

Some examples of the mode of perception indicated are the following responses of subject M, inactive, facing different objects:

- Measuring tape rolled up: “Something messed up... that *should be* a tape measure”.
- Shell: “Something flattened and striped... *should be* the lid of a clam.”
- Natural sponge: “This… a bunch of wool …”. Questioned again: “…no, a sponge.”
- A white bean: “Something squashed, whitish, I don’t know what it is…” Insisting: “…a bean.”
- Apple: “A lemon”. Then he smells it and realizes it is an apple.

The understanding of the object can be quite late because, apart from the slowness of reaction, the subject first makes an examination of the visual configuration of the object and then attempts the interpretation, which is deduced either from a series of partial aspects or from some characteristic detail of the object. In objects of less frequent use the subject usually asks the question “what could this be?”. In special tests with slightly modified objects, both the diffuse perception and the difficulty of interpretation when characteristic aspects are missing are clearly demonstrated. For example:

- A table knife with the top half of the blade missing: He recognizes it immediately. He is asked about possible defects and answers: "It is okay and complete". Only when

he touches it he realizes that it is shorter than he thought; he says that with his sight he noticed that it was short, but not that short.

- Big key without teeth: (Puzzled) “I don’t know, if it is a key it would have something at the bottom.”

- Knife handle: “I don’t know either…a piece of white iron that I don’t know what it is.”

- The blade of the previous handle: “This…yes, a knife.” The previous handle is placed next to it and immediately says: “Ah! this, the handle.”

Apart from the slowness and indirect recognition, it can be said that objects are quite well understood, and only in special tests like the ones above, some difficulty arises. Instead, under facilitation by intense muscular contraction, the perception of objects is rapid and unitary, and modified objects are also correctly interpreted.

All these tests are performed in near vision and in very good illumination; the perception of the configuration is then reached in about three or four seconds, which means a very large increase in time compared to a normal individual. It is evident that in the examination of certain objects, although the configuration can be seen well, an additional time is spent in appreciating characteristics. But when the vision is less good because of the distance, or in near vision but with weak illumination, the perception of the object requires more time, going through a series of sensory stages. In such conditions, a pair of scissors needs about eight or nine seconds to be perceived; first, a dark spot appears which soon becomes smaller, then something elongated with a greenish tint is perceived, next a more defined shape such as "something long with two rings underneath" and the subject thinks: “it can't be a key, maybe a pair of scissors”. Given the rather long time needed for this development, the process can be followed quite easily in subject M, but if the exposure time of the object is shortened, the perception stops in the primary phases, which are insufficient to reach the interpretation of the object. Under the same conditions, subject T in monocular vision using the right eye needs only about two seconds, sometimes almost three, whereas in binocular vision only one second. Also in the first moments, objects are perceived as swollen and shapeless, and later more shaped. Thus, with low intensity of stimulation, the perception of objects develops slowly and goes through various phases due to asynchrony.

In subject M, who is the most impaired, very different types of object perception are easily obtained depending on whether only one or both eyes are used, and whether the state is inactive or facilitated by muscular effort. In general, in ordinary illumination, binocularly and inactive, or only with the right eye and with facilitation by muscular effort, perception shows some deficit; it is not very coherent and stable, and defects in objects go unnoticed or very attenuated. In contrast, in binocular vision and with facilitation, perception is very good and small objects like a dice are recognized in one second at most, and if they are bigger and easier, half a second is enough. However, if the illumination is reduced or the distance is increased, the difficulties of the other inferior types of vision appear. In ordinary life, the perception of objects, although a little slow, is not bad at all because binocular vision is used and there is always some facilitation by muscular effort that the subject consciously increases when necessary since the discovery of such phenomenon. But if summations are excluded, a great difficulty arises; thus, in

monocular vision and medium illumination and distance, a felt hat can easily be perceived as a beret. Subject M can see the time on a pocket watch at a maximum distance of 14 cm in good illumination, binocularly, and with facilitation by muscular effort; and although the thinnest lines of the Roman digits disappear, this is not a problem because he knows the location of the digits in relation to the watch handle, and if the hands seem too short because their thinner ends are invisible, this is not a great impediment either. Even with facilitation by muscular effort but in monocular vision, he can no longer perceive either the numbers or the hands of the clock. Therefore, to use the watch he should resort to all possible facilitation effects to overcome the situation. Naturally, in examples of this type, the not inconsiderable defect of visual acuity must be added to the agnosia disorder.

In summary, it can be said that visual perception of objects behaves in the same way as other functions, i.e. there is a deficit which can be very significant, but perception also depends on the conditions of stimulation, the state of central excitability, the type of facilitation, etc. In general, the interpretation of an object is almost always possible except in the case of special tests, although the process is slow, indirect and less consistent than in a normal individual. It is important to point out that the true capacity to interpret objects is properly revealed in drawings where, due to the necessary schematic simplifications, the difficulties of conception and understanding become very evident, which we will deal with later. Changes in perception of the shape of letters, for example, can already be considered as an alteration of alexic type, and lead us to the study of perception of drawings, symbols, etc. A rigid separation between objects and their abbreviated representations by means of drawings cannot be established, so we shall continue the study of the understanding of objects when dealing later with the cognitive schema. There we shall also address visual agnosia, which is not an isolated alteration but only a final and complex stage in the global and unitary disorder of the visual function.

Visual image orientation

12 Visual image orientation disorder

12.1 Precedents on the issue of visual image orientation

Given the importance of the orientation of the perceived visual image, especially because of the exceptional phenomena exhibited by our wounded patients, in particular subject M, a chapter is devoted to the study of this topic. The orientation of the perceived visual image is only one special aspect of spatial localization, and due to its functional complexity, there is a very remarkable disaggregation due to nervous asynchrony, which originates the phenomenon of inverted vision in the most disturbed cases, and diversely tilted in the slightest ones. The dynamic analysis regarding excitability conditions offers more possibilities here than in any other phenomenon because quantitative determinations can easily be performed. Also, different aspects of the visual functions analyzed in previous pages find application in the research on visual image orientation. A systematic and as complete as possible study of the orientation of the perceived visual image is therefore conducted.

The issue of visual image orientation has given rise to many discussions and works since long time ago. Porta (1589/1593) and Scheiner (1619) having discovered at the end of the Renaissance that the visual image was formed inverted on the retina, the question immediately arose as to how it was possible for visual perception to be right. Kepler (1604) addressed this issue, and later Descartes (1637) in his "Dioptrique" attempted an explanation of the fact by thinking that the inverted image on the retina is counteracted by the corresponding inversion of the optic nerve fibers in the brain, thereby making vision appear right. Ramón y Cajal (1898, 1899) also tried to explain the crossover of the optic chiasm and all the decussations of the pathways of the nervous system as an anatomical arrangement intended to compensate for the inversion on the retina. But we shall see that the physiological experiments of Stratton (1896, 1897) do not give weight to this anatomical criterion.

Continuing the development of the issue, we find in the sensualist philosophy of the 18th century a marked interest in the nascent psychology of senses as a basis for the theory of knowledge, and again the problem of right vision being the image inverted on the retina appears as a fundamental issue. Particularly noteworthy are Berkeley (1709/1910) and Condillac (1754/1821), who address this problem in relation to the theories of spatial

perception in which either touch or vision intervene predominantly, according to various authors. Essentially, it is thought that from the coordination of these two senses should be derived the perception of space and orientation in it. Especially Condillac (1754/1821), studying the formation of spatial orientation using his well-known simile of the statue that gradually acquires the different senses, points out that sight relies on touch to achieve the primary orientations. In this way, the inversion on the retina does not mean any difficulty for the right vision, since as long as the eyes have not been instructed by touch, there is neither up nor down for them.

The issue becomes even more significant within the physiology of senses established in the 19th century. Nativists and empiricists, represented respectively by Müller (1826) and Helmholtz (1896), dispute the solution of this issue. According to the latter author, even the adult eye needs constant information from experience to maintain agreement between visual and tactile perceptions. The debated question of why objects appear right while their retinal images are inverted seems to be solved within such a criterion. The sense of touch is able by itself to give us complete notions about space, even without the sense of sight; the behavior of those born blind is enough to convince us. Moreover, the sense of gravity which determines up and down is not immediately obtained by sight but exclusively by touch.

Finally, the issue acquired an experimental character at the end of the 19th century thanks to the important experiments of Stratton (1896, 1897) which show that the orientation of the visual image is only the result of the functional relationship between visual and tactile (haptic) stimuli on our own body. Thus, it is possible to see just as well with an inverted image on the retina as with a right image on it, since there is a correlation and adaptation based on the upright position of our own body. Stratton performed the experiments on himself, closing one eye and placing in front of the other eye a combination of lenses that gave an image inversion and therefore the image was right on the retina. The experiment lasted two and a half days and finally he was able to achieve a more or less perfect right (correct) vision despite the fact that the image of the retina was not inverted in relation to the object. In other experiments, the duration was much longer, and he described in detail a number of very interesting phenomena. When he removed the lenses, a muddled scene appeared before his eyes which lasted several hours until normality was restored.

Throughout this historical development of the issue, it was established that the orientation of the visual image must result from a correlation between visual and tactile sensations, the latter being the point of reference. Thus, the anatomical conditions, in particular the inversion in the retina, have no meaning since all is decided by the visual-tactile correlation. However, the pathological manifestation of inverted vision leads to a new general situation. The question of whether the retina could by itself determine the orientation of the image is sometimes overlooked, sometimes left in suspense, and only very rarely assumed as possible by some isolated author, but we find the specific answer to that question in the brain lesions that cause inverted or diversely tilted vision. Of course it is possible to affirm that inverted vision is due to the local sign of the retina because of an alteration in the brain mechanism of the visual-tactile correlation. Thus, the old question of the inverted image on the retina becomes topical again.

From the above indications, it should be noted that the issue of visual image orientation has so far been a study of the domain of classical psychologists and also of some physiologists of senses. Such an important issue for brain pathology is still foreign to it today, and not because it could be claimed that visual orientation disorders have not appeared in brain-injured patients. On our part, we can state that it is an extremely common alteration which, properly examined, can easily be found, if not the most exceptional complete inversion at least image tilt to varying degrees. This reveals the disturbance of the nervous mechanism of orientation of the perceived visual image and the role played by the inverted image on the retina. References on inverted vision in brain disorders are already found in Pick (1908 a), Kolb (1907), Phleps (1908), etc., and in other more modern observations by Gerstmann (1926), Wilders (1928), Halpern (1930), Pötzl (1943), etc. All the indications of these authors are very brief, and it is characteristic that in none of the cases known before ours cases, the alteration in the orientation of the visual image has been objectively observed, because the alteration had occurred sporadically in cortical epileptic seizures, angiospastic crisis, etc. This is why the authors limited themselves to recording the phenomenon, obtaining the information from the anamnesis of the patients or from their spontaneous statements. There is therefore a lack of any kind of test or experiment aimed to elucidate the disorder. From time to time hypothetical considerations were made about the mechanism of the disorder trying to include it in the mechanism of agnosia. In some cases, the existence of tumors or other types of lesions in the parieto-occipital region was verified by autopsy. Despite the singularity of this disorder, it has remained so unnoticed or ignored that it is not mentioned in the main works on brain pathology such as the two by Monakow (1914 a) and the recent one by Kleist (1934), nor in the various monographs on visual agnosia (Stauffenberg 1914, Poppelreuter 1917, 1923, Pötzl 1943, Quensel 1931, Lange 1936, etc.), although it can be assumed from some descriptions, that patients diagnosed with visual agnosia of spatial orientation would be affected by a visual image orientation disorder that went unnoticed.

However, we must point out, as an exception and very interesting physiologically, the following experiment conducted by Hoff (1929). A patient who presented a large cranial laceration due to a war wound in the parieto-occipital region, presented cortical epileptic seizures during which, without loss of consciousness, objects appeared to be tilted about 30º. When the patient was seizure free, a similar tilt occurred by cooling the brain through the cranial laceration. No details were provided about the concomitant visual disturbances (e.g. shape, etc.), the author limiting himself to localize the mechanism of visual image orientation in the mentioned brain region.

In the two cases we are dealing with, especially in case M, we shall expose for the first time in an objective way the complex phenomena of disaggregation in the orientation of visual image according to tests and experiments performed during several years, which constitute a crucial experiment to decide on the mechanism of visual image orientation in humans.

12.2 Inverted vision in diverse degree: M, T and other cases

In the summer of 1938, the phenomenon of inverted vision in the M case was discovered by chance, a few months after the subject suffered his brain injury and while he was still convalescing. He did not seem to be very surprised by the finding, and explained that since long time ago, he was seeing occasionally tilted and even inverted objects. He seemed to have seen men upside down working on a scaffold; without giving much importance to the fact, he used to say: “these are things that sometimes come into my sight”. Otherwise, the disorder did not make his ordinary life at all difficult; he was able to walk down the street and orient himself perfectly without suffering any accident. This is understandable if one considers the phenomena of summation that usually occur spontaneously: binocular vision (mutual eye facilitation) and standing or walking which acts as a moderate but very efficient facilitation by muscular effort.

In addition, if the subject only pays attention to close or large objects, which appear sharper, perception is correct even without facilitation by muscular effort. Objects that are seen well, appear correctly oriented, so the subject cannot find any divergence with tactile information when handling them. However, if an object is far away and its motion is perceived as reversed, the perception of its form is very poor or completely blurred and only the reverse direction of motion can be perceived, and since it is far away, orientation by the sense of touch is not possible and no discordance with this sense is manifested. For all these reasons, in ordinary life the phenomenon easily goes unnoticed by the subject, and when it occurs in unexpected situations it is so fleeting that it does not alarm the subject who gives more importance to the healing of his wounds, to his general state, to headaches, etc.

In August 1938, a considerable amount of new data was obtained in a short period of time during a very laborious and sometimes not at all easy research work. We were completely ignorant about the origin and mechanism of the disorder, and finding ourselves at every step in the midst of an endless change of phenomena, experimentation and observation were done rather randomly. At last, simple rules were established to produce inverted vision: objects appeared correctly oriented when seen distinctly because they were close, appeared tilted when they were far away and vision was worse, and appeared inverted when they were even further away and vision was very poor. Of two very different sized objects placed at the same distance, the smaller one was more tilted. Also, if the exposure time of the object was very short, it was perceived as strongly tilted or inverted, even in near and distinct vision. These conditions of stimulation that allowed us to obtain inverted vision with ease and security in subject M, were not properly valued until much later, since the attention was initially focused on finding new phenomena close to the orientation disorder, such as recognizing numbers and letters equally well in any orientation, and other complex alterations of spatial orientation. One year later, this subject was examined again in detail, the excitability conditions were specified and the dynamic action phenomena were found. When a sufficiently distant object was perceived as inverted, it was found that by applying facilitation through muscular effort, re-inversion was possible. Since that time, visual orientation disorder and all other disorders

have been systematically investigated through a large number of experiments, both in the M and T case.

A more precise examination shows that inverted vision is not complete in subject M, since in the eye with worse vision (left eye) the maximum inversion that can be reached is about 170º, and in the right eye it is 135º, the subject being in an inactive state and examining each eye separately. The difference up to 180° is sufficiently small so that vision in certain circumstances can be considered inverted for practical purposes. Other maximum tilt limits are obtained through different facilitations (bi effect, muscular contraction, etc.), due to the correction of asynchrony and subsequent improvement of the orientation disorder. In this way, a same individual can present alterations of different intensity in the image orientation, according to his brain excitability state (his physiological level).

Because the brain lesion in subject T is less severe, his physiological level is less altered, as we have already seen in various visual functions. In this subject, we find a maximum tilt of only about 25º in the eye with worse vision, and different limits of less asynchrony are also obtained by applying different types of facilitation. Conversely, the degree of asynchrony can be increased either by brain cooling through the cranial wound or more simply by the effect of alcoholic drinks; in both situations brain excitability is reduced and consequently the degree of asynchrony is increased. At the beginning of the examination of this second subject in 1938, the tilt of the image was somewhat greater but not reaching 90º. However, during his first cortical epileptic seizures due to his cranial scar, his vision seemed to be strongly altered, and even after the seizures had stopped, visual image orientation was very disturbed, preferring to stay in bed until the disorder disappeared. In his own words, the position of objects "appeared very messy". It also seems that he had presented this altered state when he regained consciousness a few days after being injured. It is very likely that in such a situation this subject has presented the phenomenon of inverted vision or at least close to it, and that later, after a rather rapid recovery, it has been reduced to a small tilt that now remains stable and can be revealed by minimal stimulation and other conditions, as in subject M. Subject T also showed until 1939, the peculiar and important phenomenon of loss of orthogonal function (see below) in the same way as it is presented today by subject M in an inactive state and even with good vision. Subject T was able to read the newspaper equally well whether it was in a normal position or upside down, without noticing any difference and affirming that the letters were always in a normal position. This disorder disappeared probably since 1940, and a further detailed examination in 1942 did not reveal the slightest alteration in this respect.

In the Schneider case of Goldstein and Gelb (1918), there is no mention of inversion or rotation of the visual image, but it should certainly be somewhat tilted under conditions of minimal vision. This tilt could be evaluated in at least approximately 80º, perhaps more, based on several data on concentric reduction of the visual field and other disturbances.

We can also mention other cases found by us, some previous to this study on brain dynamics in the M and T cases, and others after it. In this last group there are two brain-injured subjects; one of them, already carefully examined by us in 1938, has a small

shrapnel brain injury in the right occipital region, two or three centimeters from the midline. He presents a somewhat *asymmetric* type of brain disorder; therefore, he is not a pure central syndrome like M, T and Schneider, although he clearly shows dynamic alterations of a central type (general repercussion, summation, asynchrony, etc.). The importance of this case and the other (see below) will be discussed in the last part of this study. For the moment, we only indicate that the visual field of this first subject shows an intermediate alteration between hemianopsia and concentric reduction, which could be more precisely termed *asymmetric concentric reduction*. Limiting ourselves to the visual system, he presents, besides disorders in all functions (colors, luminosity, acuity, etc.) including an extremely evident green chromatopsia already mentioned, a maximum tilt limit of the visual image in the eye with worse vision of about 45º, much more than in the T case. This significant alteration in image orientation was recently discovered by us and has gone completely unnoticed by him for about five years, as well as other alterations, despite he being a professionally qualified individual who performs well in his activities. The other case is that of a student who was examined long after being wounded in the left parieto-temporal region and who also shows an asymmetric brain disorder, as the previous case but with a different location of the lesion. In the former case, the lesion is near the right occipital pole; in the latter, the lesion is near the left superior parietal lobe. Due to the dynamic phenomena of repercussion, which also occur in this subject, the rather anterior parieto-temporal lesion has caused a general disturbance that affects, although weakly, the visual function. His vision seems completely normal at first, and only sometimes he gets tired easily. But when the subject is duly examined, he shows a deficit, more evident in the right eye (contralateral to the lesion) in which a tilted vision of 5º to 7º can be detected. This subject must have had a more intense repercussion shortly after he was wounded; when questioned about this, he said that while he was hospitalized, he was surprised at seeing the baseboard strip of the room quite tilted instead of horizontal.

As for the cases we found prior to our brain dynamics research, we can cite numerous cases. It is remarkable that among all the cases studied so far, including M and T, only one wounded man with an anatomical occipital lesion and visual disorders very similar to those of the above-mentioned subject with asymmetric concentric reduction, spontaneously complained of seeing things tilted. Indeed, it was enough to tilt an elongated object in the opposite direction for him to see it vertically. At that time (1938) we did not know how to interpret this disorder and therefore neither did we know how to determine the maximum tilt. Since the compensating opposite tilt had to be about 40° under ordinary conditions, much higher limits were certainly possible with minimal stimulation. Other cases with some tilted vision were found when various tests were carried out on wounded people with either an occipital or parietal convexity injury, but we did not determine the degree of disorder either, limiting ourselves in the protocols to recording the tilted vision together with other phenomena of a different nature. At least 6 cases are in these conditions and surely some of them with vision close to the inversion as in the M case. In addition, many other cases must have gone unnoticed to us.

As an end to this section, we should draw attention to the following points. Firstly, it is clear that cases with tilted vision should be considered as frustrated types of inverted

vision or rudimentary forms of inversion. Even in subject M, an exact 180º inversion is not reached although his vision can practically be considered inverted under adequate stimulation conditions. The degree of asynchrony determines the maximum tilt limit, and the disorder in all cases lies only in the disaggregation of the brain's mechanism of localization or visual-tactile orientation. If the perceived orientation is somewhat tilted, such a mechanism is more or less asynchronous, and the retinal factor (with inverted image localization) manifests itself to a variable degree. In short, different tilts are quantitative aspects of the same alteration.

Secondly, the alteration of the orientation of the visual image does not represent an autonomous syndrome, as is also the case with any other type of disorder, since a functional disorder is always for the whole of brain activity. Within all visual functions it is not possible to consider the function of visual image orientation independent of all others, and the intensity of the orientation disorder runs parallel with the disorder of the other visual activities. It is also very important to point out that the orientation of the visual image is disturbed from anywhere in the sensory brain. In fact, we have detected image tilts of different degrees in patients with either right or left parieto-occipital lesions, lesions in the occipital pole or far from the occipital region, such as lesions in the very anterior parieto-temporal region. Thus, there is no need to search for any kind of localization, an issue already partly addressed.

Finally, it can be stated that tilted vision is a very common disorder which has to be conveniently searched for. Any brain injury that affects visual functions in some way will immediately affect the orientation of the visual image by causing at least very small tilts. Tilt measurement can even be useful as a simple and sensitive method to determine the intensity of the visual system disturbance.

12.3 General features of the visual image orientation disorder

The mentioned visual image orientation disorder and especially the phenomenon of inverted vision will be studied in case M, using case T as a complement to the different tests. The first experiment consists of the tilt and inversion that occurs when an object, which is correctly seen at a short distance, is progressively moved away. This is nothing more than the fundamental experiment of asynchrony, in which the sensory level (perceived orientation) depends on the intensity of stimulation (visual angle and light reflected by the object). This change in perceived orientation as a function of the stimulus intensity occurs in conjunction with changes in other visual functions such as color and shape; thus, the alteration of the visual system is global, which is important to bear in mind. If we use an upright white cardboard arrow about 10 cm high and 1.5 cm wide (3 cm at the head), it can be seen practically upright in very near vision and in very good illumination. In these conditions, the perception of its shape is good and correct, and as for its color, although there are some green hues, most of the arrow looks white. But if the arrow moves away, always in the same upright position and with its head upwards, a tilt is produced and at the same time the shape becomes blurred and the white of the arrow tends to disappear while green dominates more and more. This usually occurs when the

perceived tilt is about 90º. Moving the arrow further away, all these characteristics are quickly accentuated and the arrow finally looks almost inverted, dark green and very altered in shape, like something elongated where the arrow head is distinguished only by a small bulk little different from the rest of the shape. Then the maximum inversion occurs, which does not exceed about 140º in the right eye, whereas it can reach 170º in the left eye. By moving the arrow a bit further away, all visual structure disappears and the preceding perception becomes a dark, rounded and diffuse colorless spot. Consequently, as a general empirical rule it can be stated that in sharp vision, the perceived orientation is normal, and the more blurry the image becomes, the more pronounced the alteration of the perceived orientation. In terms of excitability it means that intense stimulation results in normal orientation, and weak stimulation results in altered orientation.

It is clear that the degree of asynchrony and therefore the degree of image tilt depends on the severity of the brain disorder, and in all cases, the maximum tilt limit must be determined under minimum stimulation, i.e. when the perceived shape is just at the point of being lost.

In the perception of orientation (and also in any other type of perception), the following three factors must be borne in mind: *stimulus*, *receptor* and *central* nervous condition. In the stimulus, it is necessary to consider mainly its intensity and duration; the receptor influences the sensation according to its state (light adaptation degree) and by the site of the visual field (central or peripheral vision), and finally, the nervous centers show different physiological level (different level of excitability and asynchrony) according to the summation effects to which they are subjected. As for the stimulus, the excitation produced by the retinal image of an object depends on its size and luminosity. Thus, two arrows of different size and at the same distance will have different tilts; the larger one should be moved away to equal their tilts. In addition, since subject M's left eye has somewhat less functional capability than the right eye, an arrow at a given distance will be perceived as more tilted if seen by the left eye than if seen by the right eye. Stimulus duration should also be considered. Even in very near vision and very good illumination, if the exposure time is shortened, the sensation does not develop sufficiently and a tilt appears; the shorter the exposure time, the more pronounced the tilt. Together with the tilt, there is blurring of the shape, color alteration, etc., since the entire visual function is reduced. It should be noted that, due to the slow reaction time, even at high stimulation intensities, a time of several seconds is required to achieve completely a right vision. A certain amount of stimulus (combination of intensity and duration) is therefore needed to perceive a normal orientation of the visual image. This is also true for the receptor, which intervenes through the degree of adaptation to light and the stimulated zone of the visual field. An object correctly perceived in central vision can appear tilted in peripheral vision; the more peripheral the vision, the more tilted the object appears. Therefore, to maintain correct orientation, the intensity of stimulation must be increased in peripheral vision. As for the central nervous state, its action depends on the summation effects that reduce asynchrony. If subject M in the inactive state and in monocular vision perceives the arrow at the limit of inversion, a strong contraction of all his musculature is enough to cause a significant re-inversion that brings the arrow to an almost normal

position. A complete re-inversion from the maximum limit of inversion never occurs because the indicated facilitation does not erase completely the asynchrony; however, the re-inversion is considerable, about 120º. Nonetheless, by adding other types of facilitation such as binocular vision, the re-inversion can be further improved so that almost full re-inversion can be reached. By adequately reducing the stimulation until an imperfect vision is obtained, a tilt can be achieved again even in binocular vision and under facilitation by muscular contraction, but in this case the maximum tilt is about 30º, whereas only using the left eye and in the inactive state the tilt is 170º.

Also through iterative stimulation, a certain re-inversion of the image is achieved from maximum tilt in monocular vision and inactive state. This can easily be done by quickly covering and uncovering the white arrow with a black card. However, in the various experiments described below, only summative facilitations allowing accurate determinations will be used.

The great asynchrony exhibited in visual image orientation and the great ease to measure image tilt offer an opportunity to clearly show the effect of weak tactile and acoustic sensory facilitations on visual perception. A loud whistle next to the ear of subject M causes the tilted vision to straighten out by about 15º, so that he perceives a small turn with the sound stimulus, and an opposite turn when the summative excitement (the sound) ceases. A similar effect is obtained with tactile stimulation, for example, by brushing the subject's back softly and quickly, without producing pain so that it does not cause defensive muscular contraction that could mask the experiment.

Finally, we must pay attention to the type of turn in the perceived image orientation. A rotation in the frontal plane occurs, which is the only way to preserve the visual image, which changes its orientation by rotating around the central point of fixation in central vision. In the T case, in addition to the rotation in the frontal plane, the upper part of the object appears to be slightly tilted towards the subject when the rotation is at its maximum (30º to the right in the right eye) because that part of the image is in a weaker region of the visual field, and due to spatial irradiation (flat color), that part appears to be raised towards the subject. It should be remembered that when studying the vision of flat colors in the T case, it was indicated that a colored surface situated frontally is perceived by this subject with frontal irradiation only in the right half of the visual field, so this part seems closer. For both M and T subjects and in central vision, when the intensity of the stimulus decreases, the rotation of the perceived visual image is clockwise in the right eye and counter-clockwise in the left eye. However, when the object is situated peripherally in the visual field, the sense of rotation is different for each half of the field, so that in the two right halves the image rotates in the same sense, and in the two left halves it rotates in the opposite sense, thus obtaining two homonymous hemianopsic layouts with a mutually opposite rotation. This gives rise to the special phenomenon of *binocular duplication of orientation* of the image when the image is not normal due to the stimulation conditions. A double image of a single object is then seen, the two images positioned in an X-shape, since the tilt perceived by each eye is different. In addition, because vision in each eye is impaired differently, we have that in subject M for example, the image in the left eye is more blurred and more tilted than in the right eye.

As for the method followed to determine the alterations in visual image orientation, it is very simple. The characteristics of the stimulus (luminous intensity, duration, etc.) can be easily and directly measured, and for the quantitative evaluation of the image orientation (or sensory level) produced by a certain stimulus, the compensation effect is used, i.e. rotation of the test arrow in the opposite sense until the subject achieves correct perception. By measuring then the degrees of the compensatory tilt on a graduated circle, we know exactly the rotation experienced by the perceived image. In this way it is easy to get an experimental curve with as many points as desired, obtaining very complete determinations.

13 Dynamics of visual image orientation

13.1 Fundamental experiment on visual image orientation. Stimulus dependence

The multiple excitability relationships that arise in the tests on the alteration of visual image orientation can be classified into three main groups: perceived orientation as a function of stimulus, as a function of the central nervous state and as a function of the state of the receptor. Of special interest is the degree of the disorder according to the different types of vision which in fact have their origin in the state of the nervous centers. We shall begin with experiments dealing with perceived orientation as a function of stimulus, the other two factors (central nervous state and receptor) being constant.

The perceived orientation (sensory level) as a function of the stimulation is no more than the fundamental experiment we exposed in the general part of this work (Part I) when dealing with the dynamic analysis of sensory functions. There are three fundamental types of tests related respectively to recruitment, to the asynchronous set of disaggregated functions and to sensory level development.

An experience close to conditions of ordinary life and the first that was detected in subject M is the tilt and inversion of the perceived image by increasing the distance from the test object. The test object is the above-mentioned white cardboard arrow. In order to obtain a greater degree of asynchrony and to better evaluate visual function, monocular vision is used, choosing now the right eye of subject M because it is the eye usually studied in this subject, although his left eye presents a somewhat more intense alteration.

Fig. 54 shows the curves of tilt degrees (sensory level) as a function of the intensity of stimulation given by the visual angle subtended by the arrow at different distances from the observer's eye (angle obtained from the trigonometric tangent determined by the distance and height of the arrow), in a uniformly illuminated space (outdoors with light intensity of 125 foot candles). To be more precise, it should also be considered that when the arrow moves away, the luminosity reflected by it decreases rapidly as the inverse of the square of the arrow-observer distance. In this test, the duration of the stimulus is not a variable since it is considered indefinite, i. e. long enough so that in each determination the sensation is developed as much as possible. Moving the arrow away, the sense of rotation of the image is clockwise, and counterclockwise when the arrow approaches. In addition, with a small visual angle that causes a strong tilt, there is blurred vision of the arrow, green chromatopsia, etc., as indicated on previous pages.

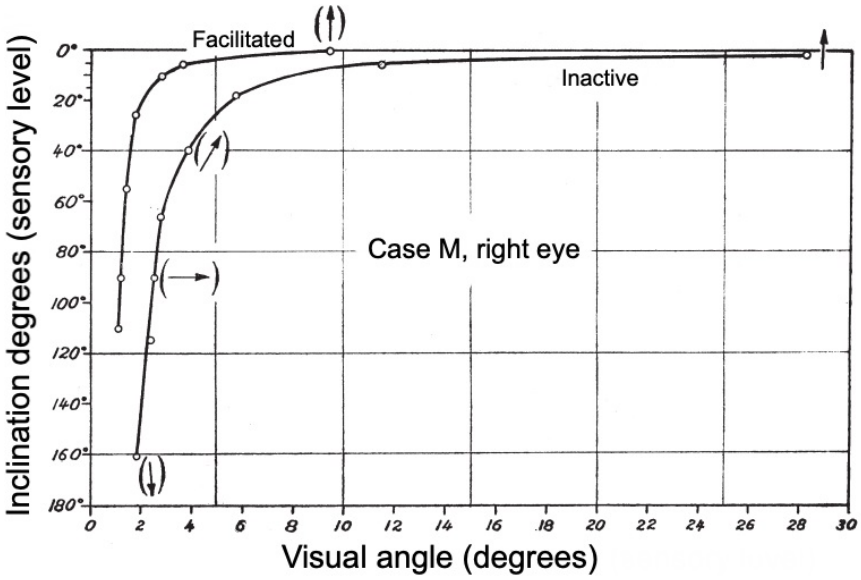


Figure 54. Perceived orientation of a 10 cm upright test arrow as a function of the subtended visual angle, for the right eye of subject M, in inactive state and under facilitation by muscular effort.

By comparing the two curves represented for the right eye of M, in inactive state and under facilitation by maximum muscular effort, the corresponding functional differences are easily appreciated. Both curves are of the same type but with different specific characteristics. The facilitation curve has a more pronounced curvature than that of the inactive state and develops within much lower values of visual angle, i.e. there is a saving in stimulus intensity. Subject M inactive at 3 meters perceives a tilt of 160º, but under facilitation the tilt is 25º at 3 meters, and 100º at 5 meters. Also, the different degree of asynchrony makes the maximum tilt limit much less under facilitation, approximately 50º less. It is observed in the curves that starting from the inactive state and low stimulus intensity corresponding to a tilt of 160º, if facilitation is applied, a very significant, although not complete, re-inversion is achieved; there are still about 20 degrees left, i. e. a correction of about 140º is achieved. It is important to note that it is very difficult to achieve completely correct arrow orientation in the inactive state and using only one eye. Even with very high intensities, a small tilt of about 5º remains, especially using the left eye which is the worse eye. Careful determinations also show the difficulty of achieving absolutely normal orientation using the right eye; even in very near vision (greater visual angle), the curve remains somewhat distant from the 0º value, as shown in Fig. 54.

The type of the curves shown corresponds approximately to Fechner's law (Fechner 1860), i.e. sensory growth is proportional to the logarithm of stimulus intensity, with the specific quantitative differences of the two extreme states of brain excitability in the M case (inactive and under facilitation). Excitability relationships are studied more accurately by evaluating the intensity of the stimulation more precisely, as we shall do in all the tests that follow, with a constant visual angle and varying the illumination of the test arrow.

To this end, we used the experimental setup shown in Fig. 55. On a table there is a holder with a white arrow on a black background surrounded by a circle marked in

degrees, and another holder with a luxometer whose photocell is at the same height as the center of the arrow. There is also an electric lamp with a screen illuminating both the arrow and the luxometer equally. Luminous intensity, regulated by a resistance, is measured by the luxometer, and the perceived tilt of the arrow is measured by the compensatory opposite rotation so that the subject perceives it completely vertical and pointing upwards. The examined subjects are placed at a distance of 25 to 50 cm from the arrow, depending on the type of experiment. Thus, while the subtended visual angle remains fixed, the sensory level of visual image orientation (degrees of tilt) depends solely on the light reflected by the arrow.

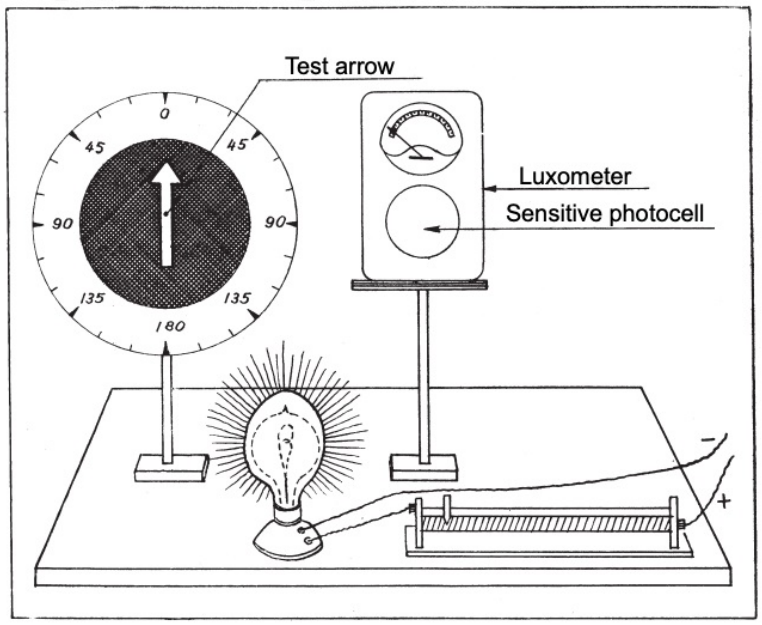


Figure 55. Experimental setup used in the test on visual image orientation as a function of light intensity. The illumination received by the arrow is measured by the luxometer, and the tilt perceived by the subject is measured by the degrees of rotation of the arrow in opposite sense so that it is perceived as vertical and pointing upwards.

By means of the indicated experimental setup, we have performed in our two subjects the fundamental experiment on asynchrony in its three main modalities: recruitment, asynchronous bundle of out-of-phase sensory levels and development in time of visual image orientation.

13.1.1 Recruitment of sensory levels as a function of the stimulus

First we shall expose the results of subject M who is particularly suitable for a more extensive analysis due to his greater asynchrony. A comparison is always made between his inactive state and the facilitated state by muscular effort, as shown in Fig. 56 where the two curves corresponding to these states are displayed. Both curves are of the same type and only differ quantitatively because they correspond to different physiological

levels. In the facilitation curve, not only the required intensity of the stimulus is reduced, but due to less asynchrony of the nervous elements, the curvature is more pronounced and the curve is significantly higher than that of the inactive state for the same eye, thus indicating a much smaller tilt limit of the visual image. With a luminous intensity corresponding to the tilt limit in the inactive state (about 145º), a decrease in tilt degrees of about 100º to 110º is possible by means of facilitation; hence, about 30º or more are always left to reach normal orientation.

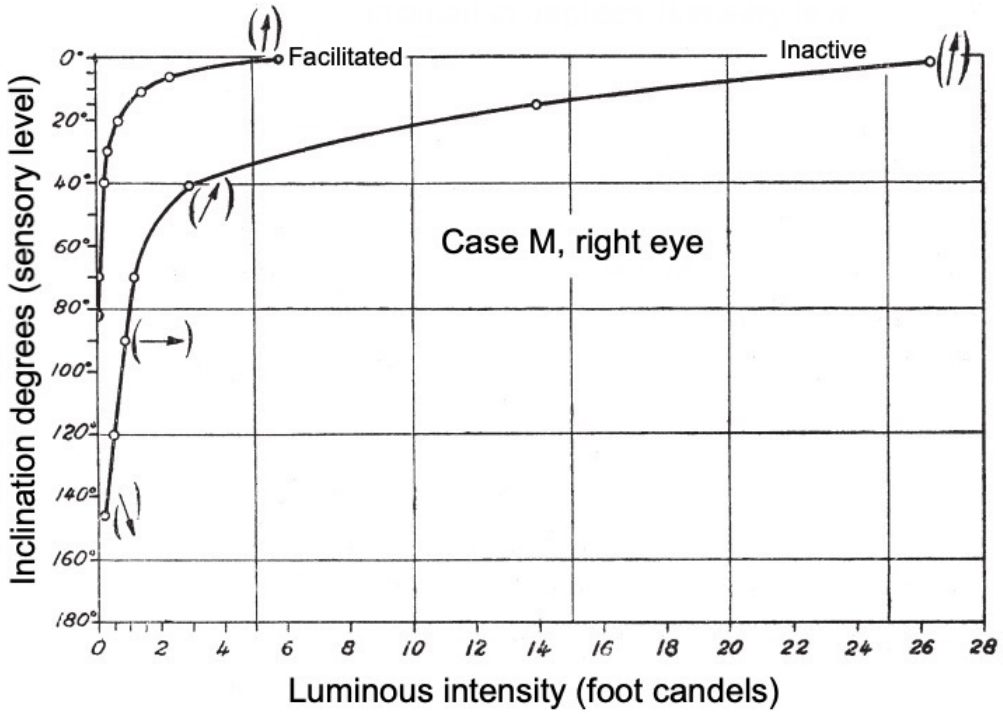


Figure 56. Recruitment of sensory levels: visual image orientation as a function of stimulus intensity (test arrow illumination) for the right eye of subject M, in the inactive state and under facilitation by muscular effort. Note the reduction in the required stimulus and the marked decrease of the maximum tilt limit when M is under facilitation. The test arrow is 40 cm from the observer

We have already said when dealing with the perceived orientation as a function of the visual angle subtended by the arrow, that a completely normal orientation without the slightest deviation is difficult to achieve in the inactive state, even with large stimuli. Even in the right eye, the one with better vision, it seems that there are always about three degrees left. This can only be determined by very meticulous examinations, since patients, especially the M case in the inactive state, only respond with sufficient precision when insisted upon; otherwise a deviation of up to 5° is neglected, and then it is difficult to determine the values at the ends of the curves. It can be seen in the curves that under facilitation and illumination of about 6 foot candles, 0º of deviation is reached, however, in the inactive state and with an illumination intensity 5 times greater, the arrow does not arrive at the exactly vertical position, the superior end of the curve being slightly below 0º.

In these tests we cannot establish a comparison with a normal subject, as we have done for other functions studied previously, since the referred orientation function responds to an all-or-nothing effect in a normal subject, so that a minimal stimulus does not produce the slightest deviation in the perceived orientation. It is only possible to compare the different pathological cases with each other, as we shall do further on. For the moment, we shall limit ourselves to a comparison between the two extreme states of excitability, inactive and facilitated, for the same eye.

If a colored arrow, e.g. red, is used instead of a white arrow, under identical stimulation conditions (visual angle and illumination) the red arrow is tilted about 10 degrees more. The white arrow, although it is seen as a very pale green, reflects more light than the red one and therefore the tilt is smaller because the stimulus is greater. This is the same effect as the one already exposed when dealing with the effect of color on motion perception using a metronome.

As for the changes in shape and color that occur along with the change in orientation of the perceived image, it is convenient to give more details resulting from the illumination test we are dealing with. These details are shown in Table 14.

Table 14. Correlation between orientation, shape (visual acuity) and color of the arrow perceived by the right eye of subject M in the inactive state. Experiment corresponding to Fig. 56.

Orient. (degrees)	*Shape (acuity)*	*Color*
0º	Distinct vision of the sharp ends of the arrowhead	Mostly white, very little green
10º	Sharp tips of the arrow head are reduced	White and green equally
30º	Slightly blurred	Green dominates over white
40º	The arrowhead triangle is not well-defined	Green dominates even more
70º	Idem but more deficient	Still traces of white
90º	Very blurry arrowhead	Completely green
130º	Smaller arrow, very fuzzy shape	Dark green
145º	All shape structure just about to be erased	Dark green limit

Inversion ↓ — **Irradiation** ↓ — **Chromatopsia** ↓

In general terms, it can be said that the shape and color of the arrow appear already altered when it is perceived with a tilt of 10º, and the alteration is quite marked around 45º. In this regard, it should be noted that when visual acuity is tested with optotypes, those of small size are perceived as tilted, and when the deviation is about 30º to 40º, they are already at the limit of distinction of their shape. The perception is extremely altered at 90º, and from here on, it worsens very quickly, as can be seen in the recruitment curves and in Table 14. At the limit of inversion, shape structure is extremely rudimentary,

appears as a faint hint, and the same happens with color perception. This sensory level corresponds in the M case to a perception of motion only as hints and in the opposite direction. Thus, maximal inversion, shape rudiments, minimal color emergence and threshold perception of motion correspond to the same functional level.

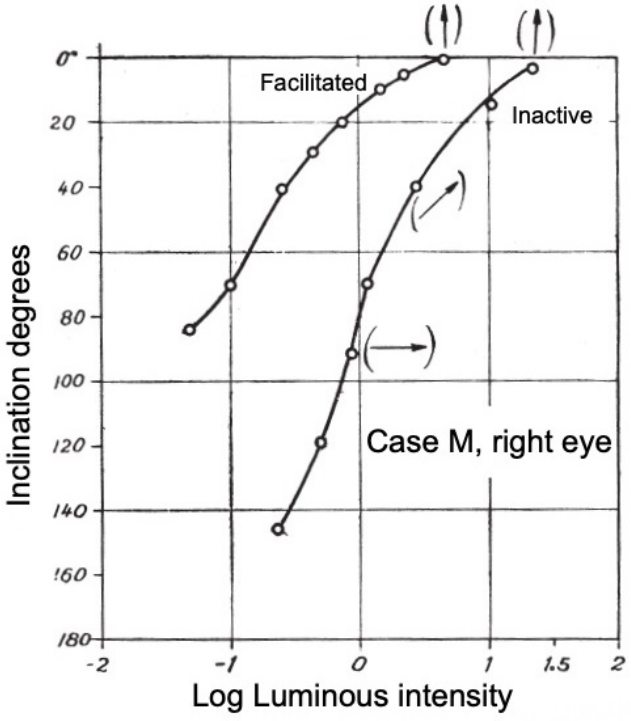


Figure 57. The same curves as in Fig. 56 but represented versus the logarithm of luminous intensity. The sensory level of the perceived orientation of the visual image increases about proportionally to the logarithm of the stimulus intensity. This is Fechner's law, which is fulfilled for most of the curve.

The sensory growth of the visual image orientation as a function of the intensity of the stimulus follows Fechner's logarithmic law (Fechner 1860). It is sufficient to take the logarithm of the illumination in the graphs of Fig. 56 to transform them into those of Fig. 57, which show a characteristic sigmoidal shape expressing the degrees of image tilt as a function of the logarithm of the stimulus intensity. Fechner's law is fulfilled except for the extreme values (very low and very high intensities), since most of the S-shaped curve resembles considerably a straight line. The recruitment of nervous elements as the basis for sensory growth is carried out in this type of alteration according to the normal general law of sensory variation, but in our cases the excitability values are very different from those of a normal subject. In the case of facilitation, the mentioned logarithmic relationship is fulfilled in the same way as in the inactive state, although at different intensities because the facilitated state corresponds to another value of brain excitability.

As regards the determination of the curves in this type of experiment, it is necessary for the subject examined to adapt to darkness first, and it is also necessary to proceed with sufficient slowness in the determinations since the stimulus is supposed to act indefinitely or for a sufficient time. It is also very important that the distance of the arrow is fixed, and above all, that the subject's head is upright and immobile, otherwise the orientation of the visual image is easily altered for reasons that we shall mention later on. Likewise,

any small dazzle or sudden change to very different illumination must be carefully avoided. If these conditions are fulfilled, the determinations are very accurate and reliable.

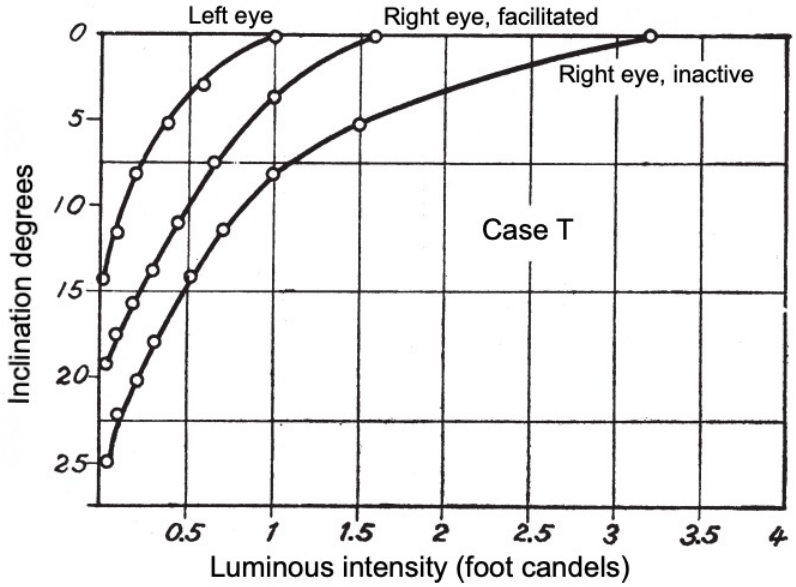


Figure 58. Orientation of the visual image as a function of illumination of the test arrow, for subject T, left eye, in the inactive state, and right eye in the inactive and facilitated state, under the same experimental conditions as for subject M (Fig. 54).

Regarding subject T, we already know that this individual presents much less asynchrony and therefore the orientation disorder of the visual image only consists of small tilts with respect to the correct orientation. Fig. 58 shows various types of alteration of the image orientation in subject T: for the right eye (the worse eye), in inactive state and under facilitation, and for the left eye in inactive state. The tests are carried out under the same experimental conditions as in the M case; therefore, the results of both cases can be directly compared. For this purpose, Fig. 59 shows various types of curves, including the one for the eye with worse vision in T inactive and the one for the right eye in M inactive. The great difference between the two subjects can easily be appreciated. Given the lesser brain disturbance in subject T, sensory asynchrony in visual image orientation is only obtained with very low stimuli, and the maximum tilt value is small. This value is about 25º in the right eye in an inactive state, and 18º with facilitation by muscular effort. In the left eye, which behaves somewhat better, the tilt limit is about 15º, and the effect of facilitation here is much smaller. Also in subject T there is an overall alteration of the visual function as that described for subject M, although much more attenuated. Like in M, the maximum tilt limit corresponds to the shape alteration limit, and a little before this limit and only in the right eye, fleeting greenish spots appear on the right side of the arrowhead, which disappear after a few seconds. These signs of chromatopsia appear mainly when the illumination is reduced.

Subject T can exhibit much more severe alterations as a consequence of epileptic seizures, alcohol ingestion or cooling of the cerebral cortex with ethyl chloride through his cranial wound.

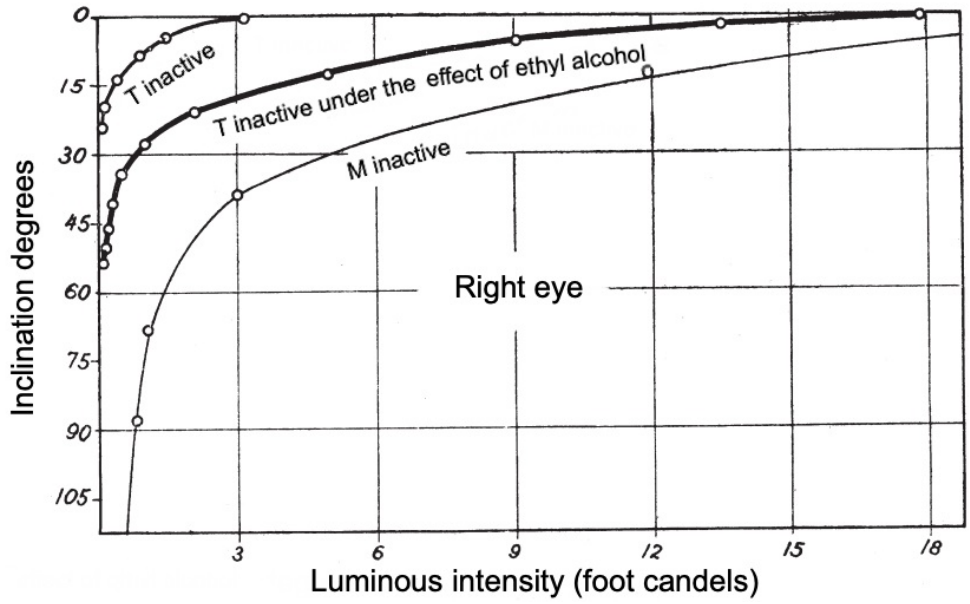


Figure 59. Orientation of the visual image as a function of illumination of the test arrow, for the right eye of subject T in the inactive state under the moderate effect of ethyl alcohol, to be compared with the highest curve (same eye but alcohol-free) and with the lowest curve (right eye of subject M in the inactive state).

The effect of alcohol is remarkable; it is known that it increases nervous chronaxie, so it produces in this subject a transitory intensification of all his brain disorders when he falls into a state of alcoholic intoxication. In such a state and in binocular vision, he says he perceives objects as much more tilted and diffuse, and everything a little darker, with a tendency for green color to dominate. If the state of alcoholic intoxication is acute, this alteration disappears in about one hour, and in less time if he drinks a cup of strong coffee. In order to examine the effects of alcohol, we have studied in this subject the effect of moderate doses by means of the following test: a small glass of sherry is dispensed to him and the change in the maximum tilt limit is measured. After five minutes, this limit has already increased considerably, reaching about 50º, whereas alcohol-free it was 25º. After ten minutes, the maximum effect seems to be reached, with the tilt being about 52º - 58º, which slowly decreases until the recovery of the initial state in about half an hour. During this ethylic effect, greenish chromatopsia increases, becoming less fleeting, although without reaching in any way that of the M case. Fig. 59 shows the curve of visual image tilt under the maximum effect of alcohol at a moderate dose. In comparison with the curve for the same eye before the ethyl test, it can be seen that not only is the maximum tilt limit more than doubled, but the curve has a flatter course and completely normal vision requires very high luminous intensities compared to that required in the ordinary state of subject T.

From the result of this moderate dose of alcohol, the effect of intense drunkenness, which subject T has suffered more than once, can be predicted. But the effect, no matter how excessive, always disappears completely. A similar effect to that observed with

alcohol is produced by cooling the cerebral cortex with ethyl chloride through the loss of cranial bone that this subject presents. In this case a moderate functional incapacity is produced that is not easy to determine with precision because the cooling becomes unbearable for the subject, with some clouding of the sensorium and of all waking state activity. The test arrow is perceived to be tilted more than 60º, and the whole vision becomes very dark and greenish. This experimental method of cooling the nervous centers, introduced by Trendelenburg (1910), offers many possibilities in experimental animals, the function being fully recovered when cooling ceases. However, this type of test in humans must be carried out with caution and for a short time because it can produce a rather accentuated general clouding of all kinds of mental activities (darkening of vision, clumsiness of language, etc.), although it is harmless when used properly, and recovery occurs in a few minutes without leaving behind the slightest trace.

Of greater interest is the effect produced by epileptic accesses. Our patient reported that just after epileptic seizures, he suffers from very poor vision, especially in the right eye, perceiving a very large increase in the tilt of objects. These cortical epilepsy seizures, consequence of the cranial scar, appear from time to time, two or three a year, and are favored by states of alcoholic intoxication. In 1944, we examined our patient two days after he suffered a severe epileptic seizure that resulted in facial contusions as he fell on his face. Although he had improved considerably in the days before the seizure, we were able to verify extremely intense disturbances that he never presented in his usual state. Thus, the tilt limit in his right eye was 120º whereas it was 25º in his usual state, and the tilt limit in his left eye was 70º whereas it was 14º in his usual state. Facilitation capability was also greatly increased, and he was able to go from 120º of perceived tilt to 60º through muscular contraction, using his right eye. At the same time, there was an analogous change in the visual acuity (from 1/8 to 1/6 with facilitation), in the very pronounced and stable green chromatopsia, in the concentric reduction of the visual field (he goes from a field reaching only 14º in the right eye with 0.5 cm test to a field reaching 22º with facilitation), etc. Tactile and auditory functions also showed greater alterations and significant changes under facilitation. In summary, when subject T was examined two days after a strong epileptic seizure, he showed an intermediate functional level between that of M inactive and M under facilitation by strong muscular effort; therefore, it can be admitted that just after the seizure, his brain alteration must have been probably as pronounced or more than that of M inactive. These alterations tend to disappear slowly, and after one week of having the seizure, he returns to his usual state.

13.1.2 Asynchronous bundle of out-of-phase (disaggregated) functions

The strength-duration curves for the right eye of subject M in the inactive state, for different sensory levels of visual image orientation are shown in Fig. 60. The tests are performed with the same experimental setup used in the previous experiments, determining for a given tilt (sensory level), the necessary duration of the stimulus according to the intensity of illumination of the test arrow. Due to the very slow reaction time of subject M, even the minimum exposure times of the arrow with high illumination

intensities can easily be measured with a chronometer, without the need for a tachistoscopic display.

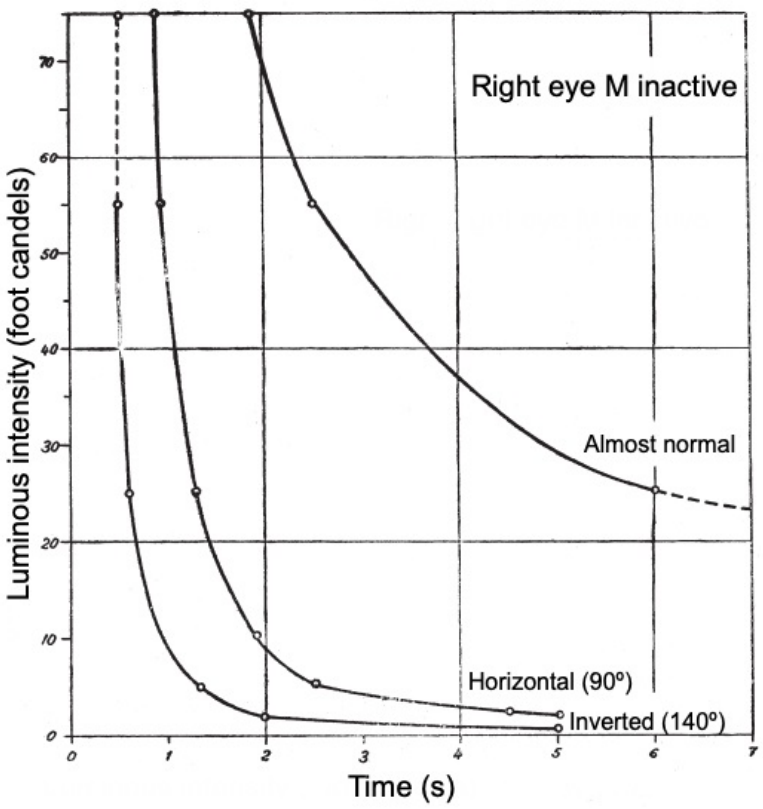


Figure 60. Strength-duration curves for three different sensory levels corresponding to the perceived orientations of the visual image in the right eye of subject M inactive. Note the high rheobase threshold of the almost correct vision and the lower curvature of the corresponding curve. (Experiment with the test arrow used in previous examinations).

Three different orientations of the perceived image are tested: almost normal vision (about 3º image tilt), maximum tilt (140º) and an easily recognizable intermediate situation which is the horizontal orientation (90º tilt). The result is three curves with different characteristics (different rheobase threshold and curvature). The curve for almost normal vision, being much higher than the other two curves and showing less concavity, indicates a functional level of great physiological demand, i.e. a high threshold and a slow reaction time, so its alteration is the first one to appear. In cases with mild lesion, as in subject T, it is the only alteration that occurs. The other two curves corresponding to pronounced image tilts are much more similar to each other although there is a noticeable difference between them in tilt degrees. We have already seen in the recruitment curves in Fig. 56 that the increment in stimulus intensity required to go from maximum inversion to horizontal orientation is relatively small, whereas it increases considerably to go from 90º to the right (correct) orientation, because the reinversion increases proportionally to the logarithm of illumination of the test arrow. Therefore, the two lower curves in Fig. 60 correspond to more easily excitable levels in time and intensity, so only in cases of intense brain alteration do these disorders in the orientation

of the visual image occur. This bundle of asynchronous curves corresponding to out-of-phase functional levels (disaggregated functions) has no precedent in normal subjects, for whom we could only determine a single curve for the correctly oriented image, which would be well below the lowest curve of subject M, and with a much shorter useful time (half a second maximum).

Under facilitation and certain types of excitability for vision, we would obtain, for both subject M and subject T, bundles of curves with less asynchrony, which would show a shorter useful time and a lower rheobase threshold. The separation between the curves in the rheobase level between the correct (or almost correct) orientation and the maximum tilt, is a measure of the asynchrony (or degree of functional delay), and of course, is the difference observed in Fig. 56 between the intensity of the stimulus for the inversion limit and the corresponding intensity for correct vision.

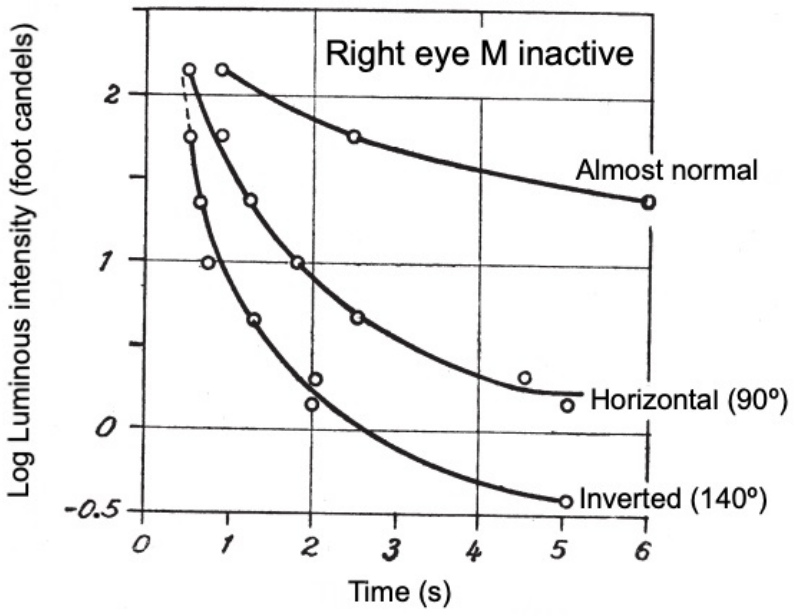


Figure 61. The same curves as in Fig. 60 but taking the logarithm of the luminous intensity of the stimulus.

Taking the logarithm of the luminous intensity of the stimulus, the asynchronous curves in Fig. 60 become those shown in Fig. 61. The rheobase levels in these new curves correspond to values of the recruitment curve for M inactive in Fig. 57, where the sensory level (perceived orientation) increases almost proportionally to the logarithm of luminous intensity.

13.1.3 Development in time of visual image orientation.

Finally, by establishing a correspondence between degrees of perceived tilt and exposure time of the stimulus for a constant luminous intensity, a curve is obtained for the development in time of the orientation of the perceived image, thus completing the

modalities of the fundamental experiment on sensory asynchrony in visual image orientation.

Due to the considerable slow reaction time in subject M (and also to a lesser degree in subject T) in monocular vision and inactive state, when any type of mid-intensity stimulation acts for a certain time, both subject M and subject T can perceive the development in time of the sensation since it goes through different stages until a final steady sensory level is reached. It is verified that subject M takes about six or seven seconds to correctly perceive a sufficiently illuminated vertical test arrow. But before reaching this final stage, the arrow is perceived as fuzzy and somewhat tilted in the first moments, and a rotation of the image occurs as the image of the arrow improves over a few seconds. Normally, subject M can only perceive a rotation of about 80° or perhaps somewhat less, since the rotation is extremely fast at first and only a change in little tilted orientations, which evolve much more slowly, can be easily perceived. By studying the evolution in time of the perceived orientation, with the appropriate device, this evolution can be conveniently fragmented and thus the complete evolution can be determined quite accurately.

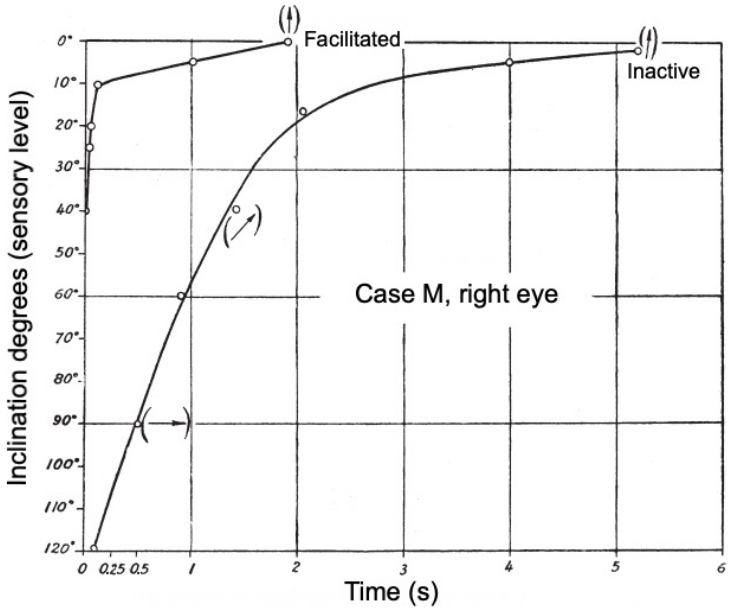


Figure 62. Time evolution of the orientation (inclination degrees) of the perceived image of the vertical test arrow, for the right eye of subject M, in an inactive state, and under facilitation by muscular effort. Note the different evolution speed and the different degree of asynchrony in these two states.

Without resorting to complicated methods, we already know that an object is perceived correctly if its stimulation intensity (size and lighting) as well as the exposure time are sufficient. However, it is perceived to be quite tilted if this time is reduced to less than one second or half a second. In order to obtain the time evolution curves of the perceived orientation, shown in Figs. 62 and 63, the duration of the stimulus is determined by means of a tachistoscopic display adjustable to the required time, and the degrees of inclination of the image by means of the compensation method, i.e. by rotating the arrow

in the opposite direction until it is perceived vertically pointing upwards. The distance and the illumination of the arrow remain constant during the determination of the entire curve of evolution in time of the sensation. In these tests we have used slightly higher luminous intensity than that of the rheobase level for correctly oriented vision, and this is the reason why subject M inactive reaches practically correct vision in less than six or seven seconds; and similarly for the other cases depicted. In the curves, we can easily appreciate the very different evolution speed and the different degree of asynchrony according to brain excitability in each case.

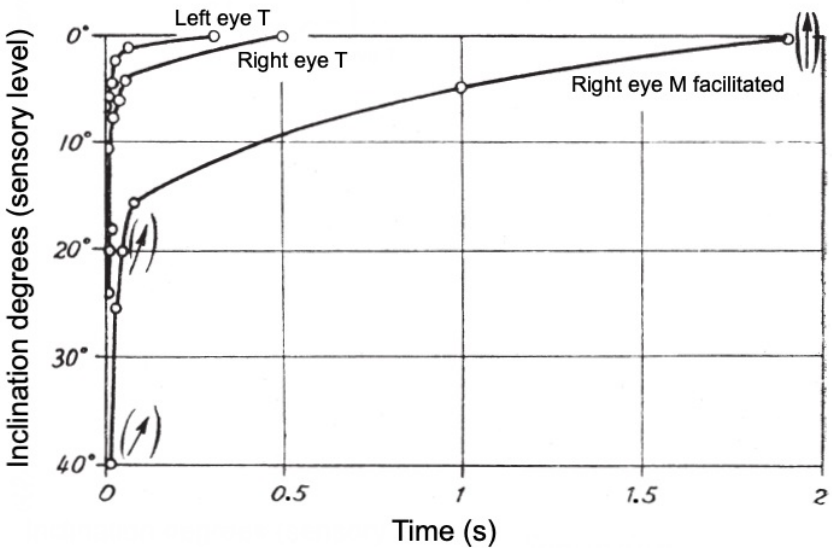


Figure 63. Time evolution of the orientation (inclination degrees) of the perceived image of the vertical test arrow, for subject T inactive, both eyes, and for the right eye of subject M under facilitation (for comparison). Note the different evolution speed and the different degree of asynchrony in each case.

The time evolution curve is approximately of the same type as that of sensory growth by increasing stimulus intensity (Fig. 56), although less precision is obtained here due to a greater experimental difficulty.

13.2 Visual image orientation depending on “types of vision”

We shall see that with only these two subjects, M and T, a large number of brain excitability levels can be obtained by combining the effect of different types of facilitation.

Binocular effect in these subjects results in very interesting phenomena. When both eyes are used, a mutual facilitation in visual excitability occurs, improving in each eye the orientation of the image. The image becomes tilted only when the stimulus is less intense, and reaches a much smaller tilt limit, although not as small as under facilitation by muscular effort. Also, because the image is tilted in opposite directions in each eye, when the intensity of the stimulus is decreased by reducing the illumination of the arrow

or simply moving it away, a binocular duplication occurs and two crossed X-shaped arrows appear, each arrow tilted to a different degree due to the different sensitivity in each eye. The following describes different stages of perception in subject M under different conditions.

1) Binocularly, in the inactive state, and illumination adjusted to obtain 0º of deviation (upright arrow): if he closes his right eye, the arrow tilts 28º to the left (tilt corresponding to the left eye only). But if he closes his left eye, the arrow tilts 13º to the right (tilt corresponding to the right eye only).

2) By suitably decreasing the illumination, the following image duplication is obtained: a somewhat diffuse arrow at 30º corresponding to the left eye and another still vertical one corresponding to the right eye. If he closes his right eye, the arrow in his left eye tilts even more.

3) By further reducing the illumination so that the image seen by the left eye is at 90º, the image seen by the right eye is oblique. In addition, the image seen by the left eye is smaller, more diffuse and darker.

4) By reducing the illumination even further, the arrow seen by the left eye disappears at 122º whereas the right eye sees it at 100º. Thus, single vision is again obtained instead of double vision.

Any type of facilitation modifies the image orientation disorder, changing the aspect of the corresponding curves and reducing the tilt limit as seen in previous figures.

Fig. 64 shows different curves for the image orientation perceived by subject M according to the kind of facilitation used. In this figure, a considerable change in the sensory function can be appreciated when comparing very different *types of vision*, such as that of the left eye in the inactive state, and that of the right eye under binocular effect and facilitation by muscular effort.

As we already know, facilitation exerts an action on the nervous centers by modifying the central state and thus making up for the deficit caused by the destruction of brain mass. When muscular effort is combined with binocular effect, the action exerted is considerable and the visual defect is significantly corrected. Therefore, it is understandable that the usual behavior of subject M in ordinary life, using binocular vision and performing some muscular activity such as walking and other movements, results in a quite reduced image orientation disorder compared to what is obtained in monocular tests in the inactive state.

Figure 64 shows how the curves tend to approach 0º of deviation as more facilitations are applied, so lower and lower stimulus intensities are needed to reveal deviations from the vertical line, and at the same time the maximum tilt that can be achieved is much smaller. In addition, the curves tend to have a more pronounced curvature, which means that the variation of image orientation as a function of stimulus (differential sensitivity) tends to be more pronounced. In short, asynchrony is largely corrected by the action of summative facilitations that bring the excitabilities of the central nervous elements involved in image orientation closer, i.e. more synchronous, and the disorder is thus greatly reduced.

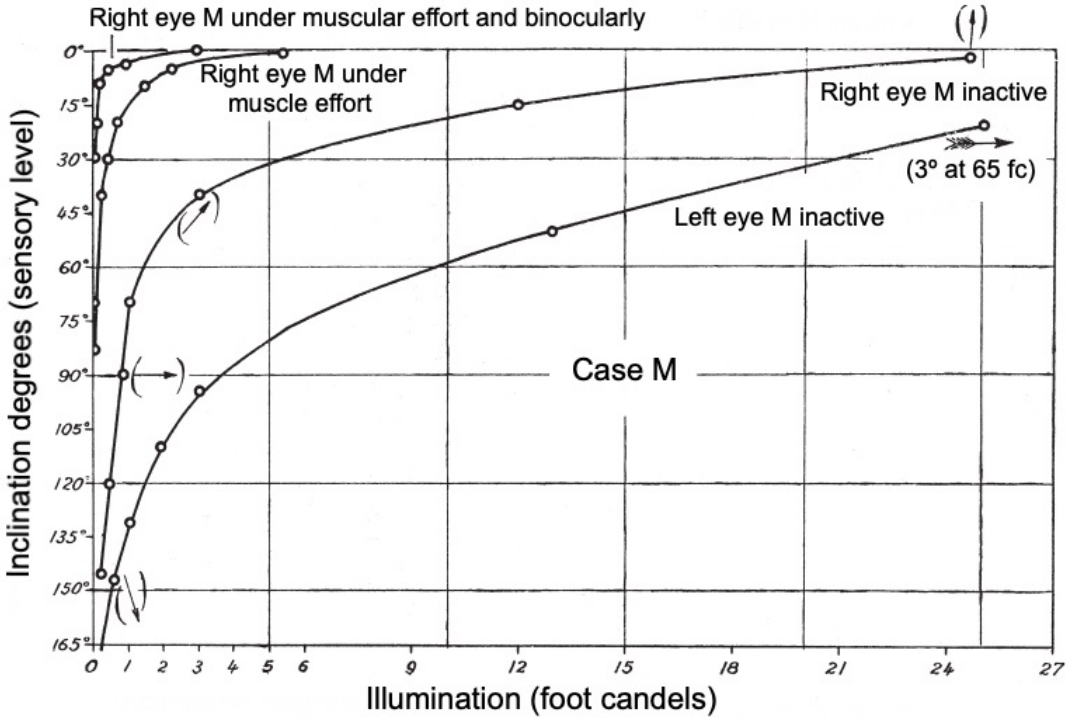


Figure 64. Orientation of the image perceived by subject M as a function of luminous intensity of the stimulus, for different *types of vision* in the right eye according to different types of facilitation, and for the left eye in the inactive state. Note the different slopes and different maximum tilt limits. The more facilitations are applied, the greater the reduction of the stimulus to produce correct vision, and the smaller the tilt limit.

In Fig. 58, above the curve for the most favorable state of subject M, there are still three curves for the T case. Hence, with only two subjects, we obtain a series of functional types that allow us to investigate the perception of visual image orientation in very different states. It is possible to obtain, for each subject, eight types of vision. The two eyes having a somewhat different functionality, different curves are already obtained monocularly in the inactive state; in addition, we must also consider facilitation by muscular effort, by binocular effect and the combination of both facilitations for each eye. Therefore, with both subjects, 16 types of vision are obtained with the corresponding different curves.

From the data of the curves shown in Figs. 58 and 64, different degrees of inclination are obtained with respect to the best vision taken as a reference. Thus, by adjusting light intensity so that in the left eye of subject T the exact upward orientation of the arrow is reached, other types of vision provide the tilts shown in Table 15.

Thus, with the same intensity of stimulus (illumination of the arrow at a fixed distance) required for the left eye of subject T to see the arrow completely vertical upwards, the left eye of subject M perceives the arrow at an angle of 135º, i.e. close to full inversion.

Table 15. Inclination degrees for different types of vision in comparison with the best vision taken as a reference (see the text). OS = left eye, OD = right eye.

Type of vision	*Inclination*
T inactive, OS	0º (reference)
T facilitated by muscular effort, OD	3º to the right
T inactive, OD	6º to the right
M facilitated by muscular effort and binocular effect, OD	6º to the right
M facilitated by muscular effort, OD	16º to the right
M inactive, OD	75º to the right
M inactive, OS	135º to the left

Although we do not provide all the types of curves of perceived orientation that can be obtained in these two subjects, we shall indicate the maximum tilt limit of the image according to the types of vision in each subject. The tilt limits in the eight types of vision provided by the M case are shown in Fig. 65. These data are not the inclinations shown in Table 15, since a different stimulus intensity is needed in Fig. 65 for each type of vision. Thus, as more favorable types of excitability are considered, the maximum tilt becomes smaller and corresponds to a lower stimulus intensity, as can be appreciated in the curves shown in Fig. 64. As seen in Fig. 65, even subject M in the inactive state with his worse vision eye does not achieve a complete 180º inversion, although it is quite close and can be practically admitted as inverted vision.

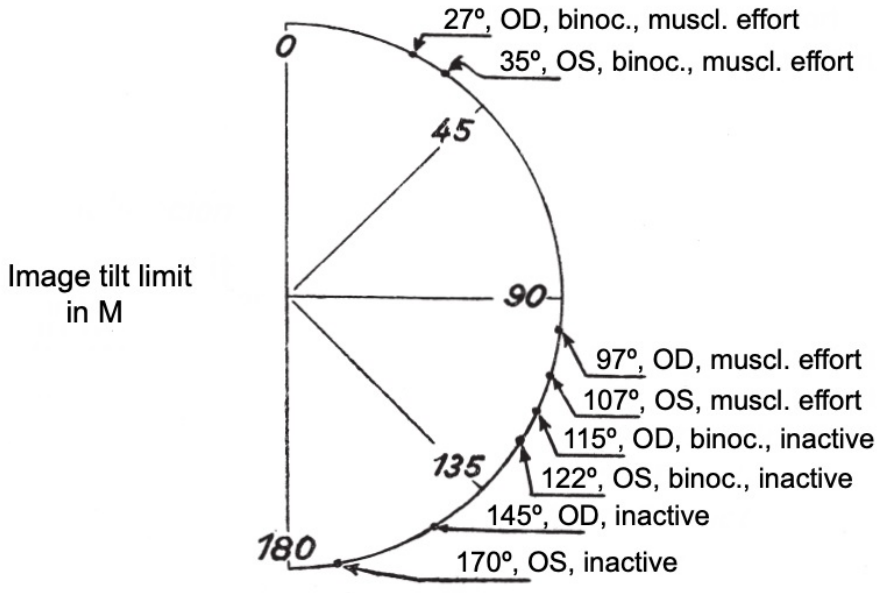


Figure 65. Image tilt limit according to the *types of vision* in subject M, corresponding to different excitability states obtained by means of diverse facilitations. OD = right eye; OS = left eye; binoc. = binocularly; muscl. effort =muscular effort.

It can also be appreciated that the action of the binocular effect is weaker than muscular effort, and by the combination of both facilitations, the functional state is largely

improved, showing a very reduced tilt limit in comparison with the other states. It should be recalled that beyond the maximum tilt limit, the arrow loses every vestige of shape, even the aspect of something elongated, being impossible to appreciate its orientation since the arrow becomes a diffuse rounded spot.

The types of vision that can be determined in the T case are shown in Fig. 66, which refer to the ordinary state of this subject, i.e. free from postictal effects of epileptic seizures, toxic residuals, etc. The types of better vision, that is, left eye facilitated by binocular effect and left eye facilitated by both binocular effect and muscular effort are not indicated since they do not seem to present any image tilt. As regards the tilt limit of the image, it can be seen that the types of vision in the M case are lower than those in the T case. Nevertheless, the most favorable state in M can be equated with the most deficient type in case T. Thus, OD of M facilitated by muscular effort and binocular effect, with 27º maximum image tilt, is practically equivalent to OD of T inactive, with 25º maximum image tilt.

Therefore, by applying all the facilitations, the M case becomes equivalent to the most deficient state of vision in the T case. This means not only an equalization in the orientation function, but also in all other visual functions (color, acuity, motion, etc.), as well as a same chronaxie value in the electrical excitation of the retina. However, although the mentioned types can be considered equal as far as functional level is concerned, a small difference is found in accurate determinations, which is negligible in ordinary life.

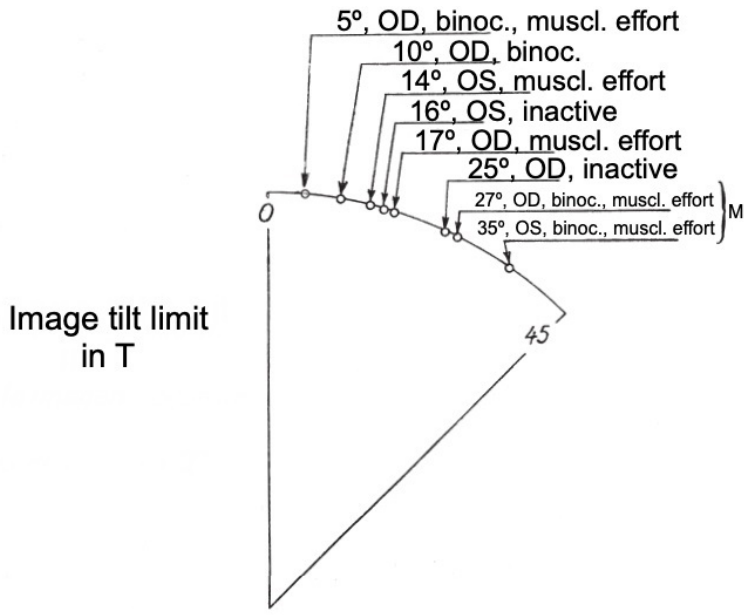


Figure 66. Image tilt limit according to the *types of vision* in subject T, corresponding to different excitability states obtained with diverse facilitations. Compare with the more favorable types in subject M. OD = right eye; OS = left eye; binoc. = binocularly; muscl. effort = muscular effort.

We have already indicated that in the T case, excitability is greatly altered by the action of alcohol drinks, leading to an increase in image tilt. Excitability is also particularly altered by the functional exclusion produced by epileptic seizures whose

residual effects last a few days. In this situation, subject T may show a very similar state to that of subject M, and many types of vision are the same in both subjects.

13.3 Synchronization by diverse facilitations. Variation in the central nervous state

As was said, the diverse degrees of alteration corresponding to the different types of vision are due to the action of facilitations through the effect of synchronization. Now we shall deal with the evolution of synchronization as a function of the intensity of the facilitation applied. First, we shall study the action of muscular effort on the re-inversion of the image. For the M case inactive, using the right eye and illumination such that the arrow is perceived with maximum inversion, the degrees of re-inversion (or sensory growth) of the orientation of the arrow are determined in relation to the muscular effort made by the subject by holding different weights. This is carried out in the same way as in previous experiments on saving voltage in electrical excitation of the retina, or on enlargement of the visual field by means of muscular effort.

Figure 67 shows the result obtained, which is a type of curve similar to that of perceived image orientation as a function of stimulus intensity. However, the conditions are quite different here since the stimulus (illumination and distance of the arrow) remains invariable, and the re-inversion (or sensory growth of the orientation) occurs by a central nervous modification produced by muscular effort. This facilitation works by increasing brain sensitivity (brain excitability) and bringing the asynchronous levels closer together. Hence, by increasing muscular effort, the asynchronous levels, lowering their thresholds, enter successively within the sphere of action of the invariable stimulus used in the experiment.

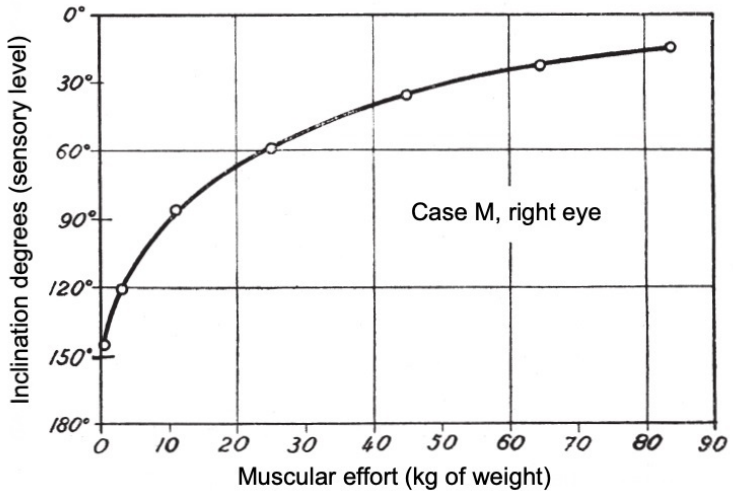


Figure 67. Synchronization (re-inversion of the image) in the M case, right eye, as a function of facilitation by muscular effort measured in kg of weight held by subject M.

As we know, a reduction in image tilt entails a parallel improvement of visual function in other aspects such as colors, shape, etc. The points of the curve here

represented constitute the average of several determinations, since this test is less stable than the preceding ones. An efficient re-inversion is observed with small weights, and it becomes less and less efficient with increasing weights, without reaching, even with extremely large efforts (holding 85 kg), the full re-inversion, since there are still about 15º to reach the vertical up orientation. But we see that by means of great muscular efforts, a very important re-inversion is achieved, namely, from 150º-145º to 15º, thus reaching a somewhat greater reduction than that indicated in other facilitation curves in previous figures. For example, in Fig. 56, the maximum image tilt in the inactive state becomes about 35º- 40º by means of the ordinary facilitation by muscular contraction, that is, there is a re-inversion of about 105º-110º, whereas the efforts of holding large weights make possible a re-inversion of 130º or more. These end positions are always difficult to determine, but we shall for the moment stick to the data indicated. In any case, starting from the maximum tilt, muscular effort does not attain to completely erase the asynchrony and to restore the function *ad integrum*, although here, in the case of visual image orientation, greater effects seem to be obtained by this type of facilitation than in other experiments such as that of saving voltage (Fig.11), or enlargement of the visual field (Fig. 45), etc.

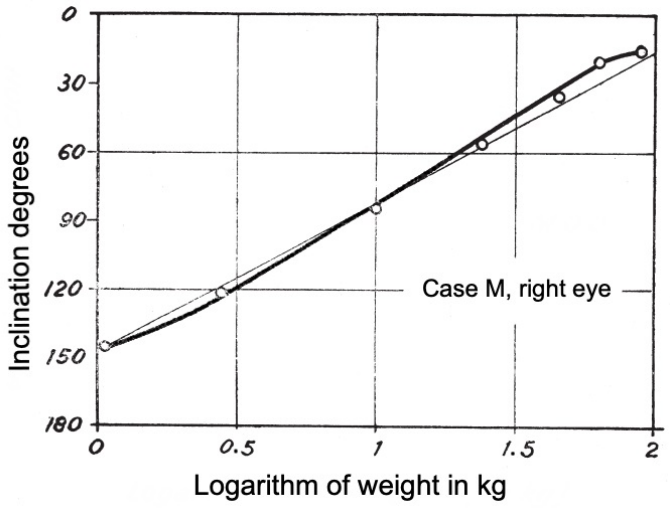


Figure 68. Re-inversion as a function of the logarithm of muscular effort (transformation of the curve of Fig. 67). Note that synchronization is proportional to the logarithm of muscular effort.

Taking the logarithm of the sustained weights, the curve of Fig. 67 becomes a rudimentary `S´ which approximates a straight line (Fig. 68), in the same way as in the case of orientation as a function of stimulus intensity (Figs. 56 and 57). This enables us to establish that the sensory level grows directly proportional to the logarithm of the facilitating muscular effort, due to the synchronization effect. Therefore, the growth of the sensory level as a function of stimulus intensity or as a function of the summative muscular effort is produced in both cases in the same way, although by a different mechanism. In the first case, there is a recruitment of levels by peripheral action, whereas

in the second case there is a synchronization of levels by a central effect of summation which increases and unifies brain excitability.

Another very interesting form of facilitation is the effect of one eye on the other one. When dealing with the different types of vision, we have already studied the double image obtained binocularly (each image has a different tilt) and the correction of image tilt. Thus, by adjusting the illumination of the arrow so that it is perceived pointing up by subject M inactive and binocularly, if he closes his right eye, the arrow tilts 28º to the left, which is the tilt corresponding to the left eye only. These tests have led us to determine the effect on the image seen by only one eye when the other one is not looking at the arrow and is illuminated directly and individually. Indeed, a very remarkable effect is produced. If, for example, the right eye looks at the upright arrow which, due to its weak illumination, is perceived quite tilted, and the left eye, isolated from the sight of the arrow, receives a luminous beam of sufficient intensity, a significant re-inversion of about 75º-80º of the image of the arrow is perceived by the right eye. Monitoring suitably the illumination of the facilitating eye, the curve of the orientation of the image perceived by the right eye as a function of the light on the left eye (which has been duly separated from the arrow by a screen) is easily obtained with high precision. The illumination of the arrow remains constant. The curve obtained, shown in Fig. 69, has the same form as the previous curve obtained with facilitation by muscular effort, shown in Fig. 67, except that in the present case of ocular facilitation the re-inversion is much smaller. This similarity between the curves is very important since it ensures and confirms the action mechanism of muscular effort, whose measurement is less easy and less stable than the measurement of luminous facilitation, performed with great ease and regularity.

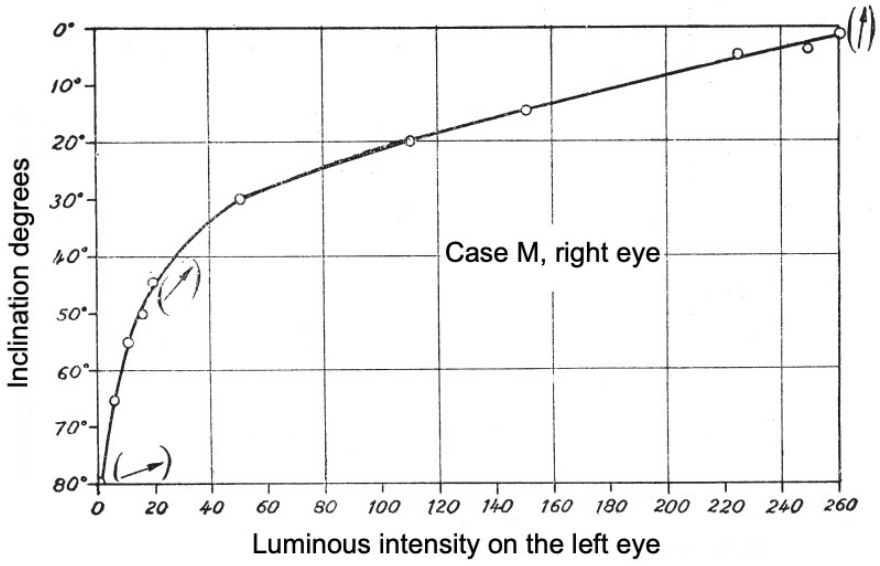


Figure 69. Orientation of the image perceived by the right eye of subject M, as a function of the luminous stimulation on the left eye isolated from the sight of the arrow, the illumination of the arrow being constant. The vertical up arrow is perceived as rotated 80º at the beginning of the test.

Taking the logarithm of the illumination applied over the facilitating eye, the curve in Fig. 70 shows the same aspect as in the previous case with muscular effort. Thus, the action of the two types of facilitation are identical. It should be noted that in order to achieve a full re-inversion by means of this type of ocular facilitation, the illumination of the facilitating eye must be increased considerably becoming much higher than the one needed to have normal vision only in the right eye. Also, it must be realized that the left eye does not receive any information of shape from the test arrow, but it is stimulated only by light. Experiments to study the effect of the various types of stimulation still need to be conducted and will be the subject of further studies. For the moment, we shall restrict ourselves to the referred experience, which proves how facilitation action of one eye on the other one, under the indicated conditions, produces a synchronizing central summation of the same type as that originated by facilitation by muscular effort.

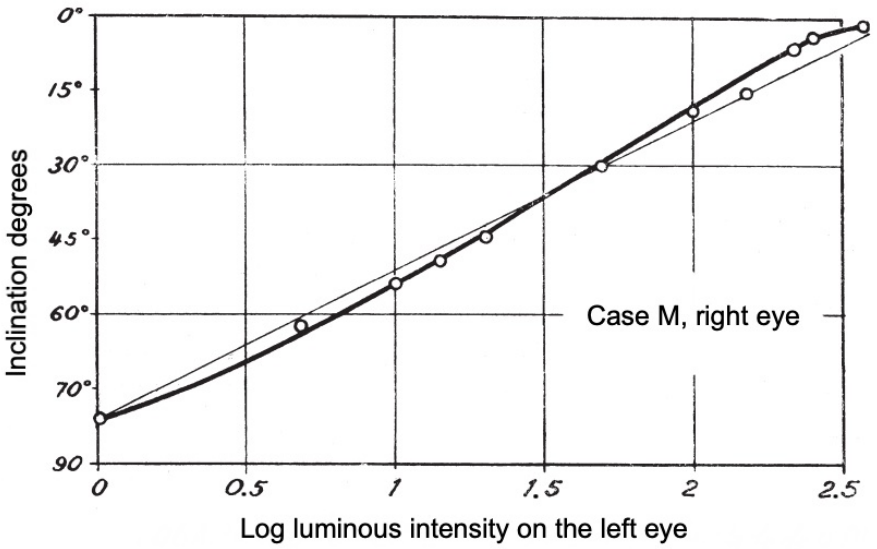


Figure 70. Same conditions as in the previous Fig. 69 but taking the logarithm of the illumination received by the facilitating eye (the one not looking at the arrow), isolated from the right eye. The sensory level (or synchronization) is proportional to the logarithm of the illumination on the facilitating eye.

Visual perception can still improve remarkably by other means since visual function is activated not only by the action of one eye on the other eye, but also by the light that bathes the eye being examined. In other words, in addition to the indirect facilitation of one eye on the other eye, the direct facilitation by the action of the light that surrounds and bathes the examined eye must be taken into account, irrespective of the light coming from the test arrow. If the arrow is illuminated so that it is seen somewhat tilted by the right eye of subject M inactive, it turns out that when this right eye is directly illuminated without changing the illumination of the arrow, a certain degree of re-inversion is achieved, which varies in the way we already know by the previous experiments. However, there is a point in which, there being a great difference between the direct

illumination on the eye and that of the arrow, re-inversion ceases and tilt reappears, increasing progressively in conformity with the mentioned difference. This new tilt under strong illumination of the examined eye is due to the fact that the eye is dazzled, and the arrow, which continues under much lower illumination, is seen increasingly darker and indistinct. This type of experience teaches us that, even in monocular vision, in order to determine the functional state correctly, it is necessary to pay attention not only to the luminous intensity of the test object, but also to the light that surrounds the eye being examined. Thus, it was already mentioned that visual acuity varies depending on whether the tests are performed indoors under medium illumination and strong brightness of the optotypes, or outdoors in full sunlight. In the latter case, the light surrounding the eye should be added to the light coming from the optotypes, in which case visual acuity would be much better.

In short, eye functionality in these subjects (M and T) depends on stimulation intensity. This intensity includes not only the one of the perceived object (size, luminosity, etc.) but also the intensity of the diffuse light surrounding and exciting the eye, contributing to the final result of the perception. Up to a certain point, because the surrounding luminosity does not correspond to the test object, the effect of such luminosity can be considered as a facilitation effect, that is, a nervous stimulation foreign to the sensory stimulation from the test object. Likewise it occurs in these subjects that a tactile stimulus is facilitated by another adjacent stimulus.

Concerning the general characteristics of synchronization by any type of facilitation, it was already said that re-inversion as a function of facilitation follows a behavior similar to re-inversion as a function of stimulus intensity; that is, the orientation perceived is directly proportional to the logarithm of facilitation or stimulus intensity, respectively. That said, facilitation cannot be considered only as a foreign stimulus that is added to the stimulus itself of the examined sensation, since facilitation is characterized by its central effect; good proof of this is the diminution of the maximum tilt limit of visual image orientation. We also know that subject M in binocular vision and with muscular effort resembles considerably subject T in monocular vision and inactive (Fig. 66). Thus, the loss of brain mass (central mass) is compensated by facilitation, so that case M, having a considerable central deficit, can be equated to case T, having a small central deficit. Likewise, we know from the study carried out on general excitability, that facilitation reduces the chronaxie and rheobase values, i.e., excitability is increased and iteration capability is reduced. This increase in excitability brings asynchronous levels closer together, i.e., there is a synchronization of the abnormally independent partial functions; however, such synchronization is never complete because the deficit in brain excitability cannot be fully corrected.

13.4 Visual image orientation depending on the state of the receptor

We have already exposed the variations in visual image orientation due to changes in stimulus intensity or changes in the central nervous state (i.e., by facilitation). We shall now deal with the influence of a third factor, namely the state of the retinal receptor, in which two main types of variation must be distinguished: topographic and light adaptation.

The different experiments analyzed above have been carried out using central vision. However, the arrow, correctly perceived in central vision due to stimulation conditions, is perceived as tilted as soon as it is placed peripherally in the visual field. The tilt increases to the limit of inversion as the position of the arrow is increasingly peripheral. This change is of the same type as the one shown by other functions, such as colors and visual acuity. Because of the fast decrease in excitability from the center to the periphery of the visual field, the same stimulus that in central vision produces normal perception, peripherally produces an increasingly tilted image. This is due to an increase in the excitation threshold towards the peripheral field that makes that the invariable stimulus becomes undervalued, and consequently, the response of the nervous centers is reduced.

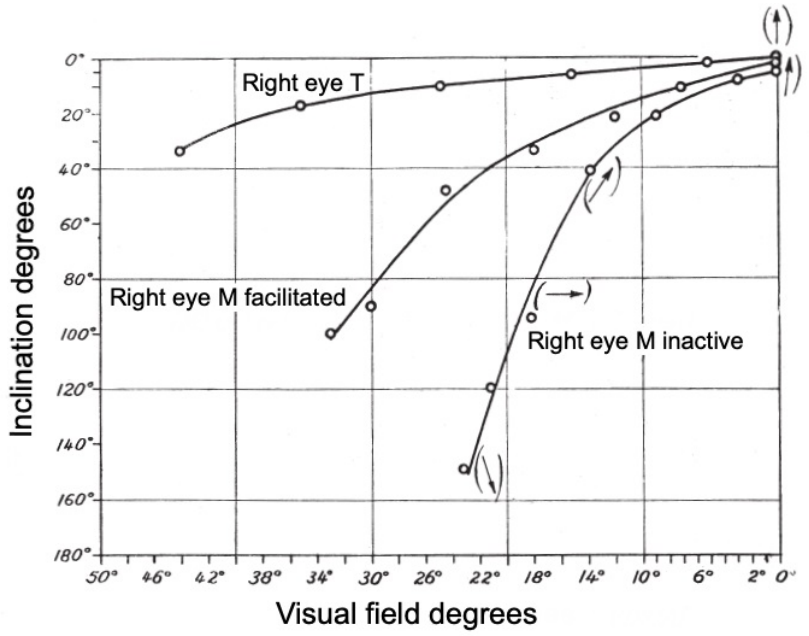


Figure 71. Orientation perceived of a vertical up test arrow, as a function of visual field degrees on the perimeter, for the right eye of subject T, subject M inactive and under facilitation by strong muscular effort. Note the different slope of the curves, the limit of vision on the perimeter and the maximum value of the tilt perceived.

Figure 71 shows the curves obtained for the right eye in the two extreme states of subject M, inactive and under facilitation by strong muscular effort, and in subject T. In order to obtain the curves, it is sufficient to provide enough illumination to the test arrow so that it is perceived as upright as possible in central vision. Then, by sliding the arrow towards the periphery, the image tilt degrees are determined along the horizontal meridian, the illumination of the arrow and perimeter being constant. Thus, three different

curves result, corresponding to different types of brain excitability. Each curve shows different slope, different maximum tilt of the image, and different site of the perimeter where it is reached. Due to the conditions of illumination and size of the arrow used, subject M in inactive state reaches up to about 25º of visual field. Under facilitation by muscular effort, the enlargement of the visual field is much less pronounced than by employing the test object of 0.5 cm in diameter, as we saw when studying the concentric reduction of the visual field. Here, in the inactive state, the arrow turns about 150º when it is at 25º on the perimeter, whereas under facilitation, the maximum tilt is about 100º when it is at 35º on the perimeter. For the T case, the maximum tilt is only about 30º when the arrow is at about 45º- 48º on the perimeter. All these tilt limits are the same as the ones obtained in central vision by reducing suitably the stimulation intensity. If in the M case, instead of the right eye, the left eye is used in the same illumination conditions (not indicated in Fig. 71), the tilt limit is about 170º when the arrow is at about 20º on the perimeter. According to what we already know, when the arrow is moved along the perimeter and is perceived as tilted, its shape changes, becoming blurry, and its color, initially white, gets increasingly greener. When the limit of maximum tilt is reached for each case, the perceived shape has worsened so much that, when the arrow is moved to an even more peripheral zone on the perimeter, its whole structure disappears, and nothing but a shapeless spot is perceived without the least element of directionality. The different slopes of the curves are of the same type as those we have seen in the experiments on orientation perceived as a function of stimulus intensity or stimulus duration, and are in relation with the other characteristics of the physiological level of each of the cases examined. As it can be seen in Fig. 71, only subject T reaches in central vision (0º on the perimeter) completely normal perception without the least deviation, whereas in subject M there is still a deviation of 1º or 2º under facilitation, and 5º to 7º in inactive state, in the same manner as indicated in other types of experiments on perceived orientation.

It follows from these tests that in order to keep normal visual perception it would be necessary to increase the illumination when the arrow becomes more peripheral in the visual field. If the stimulus intensity is constant, as in the curves shown in Fig. 71, the topographic variation of excitability on the retina is the cause of the stimulus being registered diminished towards the periphery, leading to partial reactions in visual orientation function, given the asynchrony of the nervous centers. Therefore, it can be said that these centers need to receive a constant amount of stimulation to bring about the most upright up (normal) image orientation possible in the entire visual field, within the circumstances of excitability in each individual. Not only the stimulus factor must be taken into account but also the type of receptor, that is, its excitation threshold or sensitivity to pick up the stimulus. For this reason, the same stimulus will cause very different reactions depending on the excitability of the receptor that picks it up, as it was shown in the experiments performed. In a normal subject, no alterations in visual image orientation are expected although excitability in the peripheral visual field is much lower than in the central field. This is because in the normal individual there is no asynchrony in the centers so that a minimum luminous stimulus already provides a correct perception of the orientation. Thus, in a normal subject there is an all-or-nothing response, whereas

in pathological cases there is a partial reaction more or less complete depending on the kind of stimulation.

Identical considerations must be made regarding the influence of another type of variation in the receptor: light adaptation. Already without using special tests, it can be verified that if subject M is looking at a vertical up arrow illuminated more than enough to be seen correctly, and then the illumination is slightly reduced suddenly, a tilt is produced; although after 30 to 60 seconds the primitive position is recovered because of adaptation to the new intensity of illumination, still enough to achieve the correct orientation of the image.

The influence of light adaptation (change in sensitivity of the receptor) on the functional level of visual image orientation is studied more accurately in the following experiment. First, a suitable illumination is provided to the vertical up test arrow so that it is seen as correctly as possible by the right eye of subject M in the inactive state, at a certain distance. Next, the eye is dazzled with a lamp of 250 foot candles for two minutes. Subsequently, the perceived tilt of the arrow is determined during the time taken to adapt to the intensity of the light from the arrow (much less than that of the dazzle). Immediately after the dazzle, the luminosity of the test arrow turns out to be too low for the high threshold established and the arrow is not perceived at all; however, in a few minutes, the adaptation process starts to recover the situation previous to the dazzle, and the arrow tends to take shape, going through different degrees of perceived tilt, from the maximum tilt to the initial orientation at the beginning of the experiment.

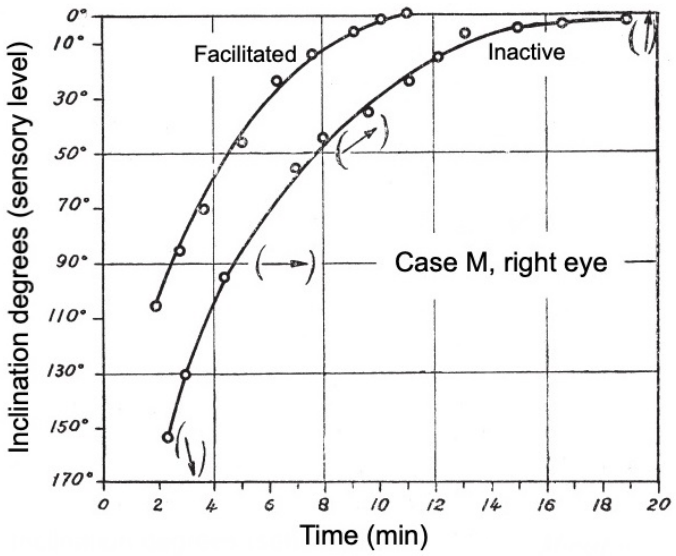


Figure 72. Recovery of the orientation of the image after a strong dazzle in the right eye of subject M. As light adaptation to lower intensities is developing, the image of the vertical up test arrow is straightening until reaching the orientation previous to the dazzle.

Figure 72 shows the two recovery curves corresponding to the inactive state and the facilitated state by strong muscular effort. In the latter state, the recovery is made in about ten minutes, and in the inactive state in about twenty minutes, without reaching in this

case a vertical up direction as perfect as with facilitation. In addition, the recovery under facilitation begins earlier than in the inactive state. The maximum tilt limit at the beginning of the recovery is different in the inactive state and in the facilitated state, as in previous tests.

Therefore, even in central vision, changes in the receptor according to the state of adaptation to light must be taken into account. When there is a change in the excitation threshold, i.e., in the receptor sensitivity, the same stimulus is detected differently and consequently causes very different responses in the nervous centers.

The recovery of the initial vertical up position prior to the dazzle is related to the light adaptation curve studied in the M case on very previous pages. However, in the case under discussion, we are not dealing with adaptation to the minimum threshold of light, but to a gradual increase in the visibility of the arrow (illuminated with a fixed intensity), as a result of the process of accommodation to light. At the beginning of the recovery, nothing is seen, but after about one minute and a half under facilitation, the perceived arrow has a diffuse shape and maximum tilt corresponding to that state. In the inactive state, that situation occurs a bit later, after two minutes and a half, and with larger tilt than in the facilitated state. As adaptation increases, so does the re-inversion, which takes place mainly in the phase of rapid adaptation (see Fig. 21). This phase lasts three to five minutes in the normal subject, whereas in the M case inactive it increases up to ten or fifteen minutes, and over twenty minutes must elapse to reach an adaptation level that a normal subject achieves in four minutes. Under facilitation by strong muscular effort, intermediate values between those of the inactive state and a normal subject, somewhat closer to the former, are obtained. In any case, although the recovery (re-inversion) occurs in the phase of rapid adaptation, subject M takes a long time to reach the state previous to the dazzle due to the slowness of his nervous processes.

As adaptation increases, the stimulation produced by the arrow has a greater effect; the arrow looks brighter, less green, more defined in its shape and less tilted. This is because the stimulus reaches the nervous centers with greater intensity.

13.5 Various complementary tests

There are still some tests that should be exposed as a complement to those performed on the dynamics of visual image orientation, although we shall now restrict ourselves to simple descriptions without indication of measurements and experimental curves.

a) Of interest for the behavior in ordinary life is the following test on drawing and perceived orientation. Subject M in inactive state, using his right eye, draws a vertical line with a pencil on white paper, at the accommodation distance and in medium illumination. At the beginning he does not see the line which is 1 cm long and less than 1 mm thick. If the line is made bigger, making it thicker and 0.5 cm longer, it is perceived, although tilted about 60º. Subsequently, if the line is intensified by drawing it about fifteen times larger (both in width and length) it is finally seen vertical, that is, in its real aspect. Therefore, the perceived orientation of the line depends on its intensity and size.

When the line begins to be seen, it is already perceived as much less tilted than the maximum inversion. This is due to the fact that the progression from the tilt limit to 60º occurs with a very small difference in stimulus intensity, as can be seen in the curves of orientation perceived as a function of the stimulus, e.g., in Fig. 56. But if the mentioned drawing is done very carefully, enlarging it very slightly and enhancing it very weakly, it is verified that the line can be perceived when it shows a tilt of about 140º, which is the inversion limit in the right eye for the inactive state, as we know. Later, with an insignificant enhancement of the line, the tilt goes easily to 90º and to 70º. Instead, it is remarkable the considerable enlargement needed (about fifteen times) for the initial line to go from the maximum tilt to the normal orientation (vertical), and even then, there is still a weak tilt, since a normal orientation is never fully achieved monocularly and in the inactive state. A warning of practical importance in orientation tests is that, if one does not proceed with appropriate meticulousness, the perceived arrow easily goes from a tilt close to 90º to the disappearance of its shape and orientation, thus leaving the maximum tilt undetermined. As said, subject M presents this incomplete asynchrony at the beginning of his visual perception, i.e., without tachistoscopic perception during the development in time of the sensation of visual image orientation. Thus, the time elapsed between the maximum tilt and a tilt of about 50º is so minimal that these positions cannot be distinguished from each other. So, when M is looking at an object, at the first instant he already perceives it to be tilted at less than 90º. Shortly thereafter, the image goes to the normal position.

b) Other remarks we should make refer to changes in the visually perceived direction of motion. When visual perception of motion was studied by metronome tests in subject M, we saw that the first oscillation perceived (only signs of motion), in addition to being very short in relation to the real trajectory, it is seen in reversed direction of motion. Thus, if the pendulum of the metronome moves from left to right covering about 10 cm, the perceived motion is from right to left and only in the most central region, covering only 2 - 3 cm. As an example, when in ordinary life vehicles or persons are seen moving in inverted direction by subject M, they are perceived ill-defined, and in the best of cases, only by the size of the blurred spot he can guess what it is all about. It is clear that for the inversion of motion, it is essential that vision be very reduced, presenting the same circumstances we already know for the inversion of an object according to its capability of visual stimulation. Therefore, depending on visual conditions, the apparent direction of a same motion can be different. It will be inverted in very bad vision, but it will be seen transverse to the real direction (tilt of 90º) if vision is somewhat better, and finally, if vision is still better, only a little deviation from the real direction of motion will be perceived.

In the perception of motion direction, the different types of vision should be considered, as in the perception of the orientation of objects. The result is very different depending on the type of vision used. In subject T, the inversion of the direction of motion never occurs since the maximum tilt in his worse eye and inactive state is only about 25º, and therefore, under conditions of minimal vision, motion is perceived with only a small deviation in relation to the real direction. It must be realized that in order to obtain a reduced vision in the perception of motion, it is sufficient to accelerate it properly, since

given the slow reaction of these subjects, minimal vision is easily reached for very rapid motions.

Proceeding in this manner, it is shown that in the M case, binocularly and under facilitation by muscular effort, inversion never takes place, and only a very small deviation from the real direction of the moving object occurs. If the motion speed is increased, the moving object is no longer visible, without reaching a greater deviation. In monocular vision and under the same facilitation and initial conditions, a very important tilt can be perceived, greater than 90º. In binocular vision and the inactive state, the tilt is very pronounced, close to inversion, and in ordinary life (vehicles, persons, etc.), it can practically be taken as a reversal of the direction of motion. Finally, in monocular vision and the inactive state (minimum stimulation), the tilt comes up to the maximum; however, in careful determinations, it is verified that it is not a 180º inversion, but a bit less in accordance with the tilt limits we have indicated when studying the types of vision. In short, since subject M proceeds with some approximation, it turns out that even with minimal stimulation, in binocular vision and with facilitation by muscular effort, he admits that there is no deviation. Strong tilt occurs in monocular vision under the same facilitation; and for the rest of cases, binocular inactive and monocular inactive, inversion appears although a little less in the first case.

In all these tests, in order to determine the maximum tilt for each type of vision, it is essential that the translation of the moving object can be seen, even if it only corresponds to the most central part of the trajectory and there are only signs of motion.

In motion directions fully or partially perpendicular to the frontal plane, inversion can also occur under conditions of minimum vision, monocularly and in the inactive state, so that a motion towards the subject turns into distancing from him, i.e. a motion in depth. The above-mentioned tests deal with motions in the frontal plane, but also in any other plane (e.g., the sagittal plane as indicated), the direction of motion can be reversed, since it is always the same process, i.e., visual inversion of the trajectory of the moving object, due to an asynchrony in the brain mechanism of visual image orientation.

c) A very different issue is the lability or instability of the orientation of the visual image regardless of stimulation conditions. We have already mentioned that when the test arrow is seen clearly because of adequate conditions of luminosity and distance, it appears correctly oriented, without any (or negligible) deviation, however, such orientation is less stable than one might think at first glance. It is true that if we do not add any other experimental factor, the orientation of the arrow is maintained inalterable, but using certain contrivances we shall see that the orientation can be modified considerably. In this regard, we have prepared the following test for subject M. It consists of placing in front of the eye that is looking at the test arrow, a cardboard with a slot in the middle of which the arrow can be seen. When this slot is rotated in any direction, the perceived arrow follows the rotation of the slot, either clockwise or counterclockwise. It is not that the arrow takes exactly the same orientation as the slot, but rather it is dragged by the rotation of the slot, which demonstrates the instability of the orientation of the arrow even under optimal visual conditions. In this way, a rotation of the arrow of about 90º can be achieved if its initial position without slot is very close to the vertical up direction. The tilt can be much greater if the arrow was already seen quite tilted before using the slot; in this case,

the arrow always takes the orientation given to the slot. But with the exception of this latter case, there is only some deviation induced by the rotation of the slot, the orientation of the arrow being lagged behind the orientation of the slot. Under facilitation, the influence of the slot decreases considerably, and if initially the arrow is correctly perceived, the rotation of the slot only induces a deviation of the arrow of very few degrees.

With regard the interpretation of the deviation induced by the rotation of the slot, we must first consider the instability or malleability of the orientation of visual image as a result of asynchrony, which makes orientation dependent on the balance between the haptic factor (tactile sensations due to body movements) inducing correct orientation, and the retinal or inverted factor. When the normal mechanism is broken, visual image orientation is drifting, and depending on the stimulation conditions, either correct orientation or inverted one is established, the latter being guided by the retinal factor. Such instability causes that any external factor intervening in the visual sensory field can change the orientation of the visual image, given its lack of firmness. Thus, if the slot regulates the field vision according to a certain orientation, this will influence the perceived orientation of the object. The more labile and unstable the structure of the sensory field, the greater the effect of the slot, therefore the effect will be greater in the inactive state than under facilitation.

Another modification in the orientation by influence of a similar mechanism over the visual field is presented in the test of tubular vision. If for the right eye of subject M, inactive, the visual conditions are such that the vertical up arrow is seen with a tilt of about 50º, and then he looks at the arrow through a tube 9 mm in diameter and 13 cm in length, the perception of the arrow improves somewhat, it is seen less tilted, diminishing from 50º to 32º. It is known that looking through a very narrow tube significantly improves vision in a normal subject. Colors appear more intensely saturated and the details of a drawing become sharper and more distinct.

In the M case, the tubular effect should be interpreted as produced by concentration of the functional capability of the visual field in a very small central zone, which would produce an accumulation or enhancement of activity, thus increasing the sensitivity of the receptor and therefore improving the orientation of the arrow.

d) Finally, it is necessary to point out some considerations about the influence of the own body of the subject on the mechanism of visual image orientation, although this topic will be discussed later when dealing with egocentric and allocentric spatial orientation in visual schema. When performing all the experiments concerning visual image orientation under very different conditions or modalities, care has been taken that the subject examined keeps his head completely vertical (immobile in his natural position) when looking at the test arrow. This detail is not unimportant since the slightest lateral tilt of the head produces a deviation in the perceived position of the arrow. This phenomenon has given rise, especially in subject T, to a remarkable tic or small movement of the head that the subject makes instinctively, without becoming aware of it. When the image of an object tilts towards the right side, the subject moves his head towards the left, so that the image appears straightened out, and even fully upright in many instances. This tic was very characteristic at the beginning of the study of this

subject, and its meaning was not easy to elucidate since he used to make this movement involuntarily for all types of perceptions: visual, tactile, auditory, etc. This is due, on the one hand, to the fact that a tilt to the left of the head and even of the trunk counteracts the deviation of the image orientation to the right, and on the other hand, it is also due to the fact that this movement or tic executed with some abruptness constitutes a summative facilitation. This fact has made that the tic be associated unconsciously to all kind of perceptions, although it has nothing specific regarding facilitation and can be replaced with any type of muscular shake. Not only in this subject T but in other two brain-injured people different from those studied here and with similar disturbances, we were able to verify the compensatory tilt of the head, although for different circumstances, it has not given rise to the tic that subject T presented for a long time. People with brain injuries who do not have a significant visual orientation disorder can correct any image deviation by compensatory tilt of the head and even the trunk. When they look at an object, they usually try to adapt the position of the head in relation to the orientation of the image perceived. To this end, they perform a series of trial and error movements until they find a position that allows them the best possible vision and orientation. One of our patients (not M or T) not only tilted the head to the side opposite to the deviation of the visual image but he also bent at the waist, curving strongly to one side to achieve an optimal straightening of the image orientation. But when the orientation disorder is relatively small, as in the T case, and the deviation can be counteracted by a small lateral tilt of the head, an associative mechanism is easily established involuntarily, which ends up accompanying every kind of sensory perception, although in many cases it acts only as facilitation by muscular effort. In this way a tic arises, somewhat different from the contouring movements described by Goldstein and Gelb (1918) in their Schneider case. In the cases above described, the tic acts by the mentioned double action of counteracting the tilt of the visual image and by central summation.

The phenomenon of counteracting the deviation by the opposite tilt of the head reveals the disaggregation of the brain mechanism of visual orientation. In a normal subject, head tilt does not cause any deviation in the orientation of the visual image, except in very special conditions, for example a bright line seen in the dark, i.e., the Aubert phenomenon (Aubert, 1865). It can be said that these brain-injured patients present this phenomenon in any situation without the need for experiments in the dark.

Hence, the maintenance of the natural upright position of the head in visual orientation experiments is of utmost importance for correct determinations. It should still be noted that the re-inversion of the visual image by lateral deviation of the head is very diverse according to the intensity of the brain disorder. Thus, in subject M inactive, it appears at its maximum, greatly diminishing its effect by facilitation. For subject T inactive, it is much less than for subject M under facilitation, and is more intense using his right eye than his left eye.

14 Theory of visual image orientation

14.1 Functional complex of visual image orientation

We have already indicated at the beginning of this study that the orientation of the visual image can be considered a spatial localization; it is a complex function that easily succumbs and disintegrates because of the dynamic conditions that originate asynchrony of the nervous elements of the cerebral cortex. The phenomena resulting from such disintegration or asynchrony depend on the sensory factors taking part in the formation of the functional complex of visual image orientation.

Here we limit ourselves to the most essential aspects in order to obtain a rational explanation of the experiments described above. It is known that the issue of visual image orientation has given rise to innumerable discussions and multiple studies, mostly because of the inverted image on the retina. Opinions of every kind are to be found even of those, like Mach (1886/1906), who think that this type of controversy makes no sense, since they consider the issue of why the image is seen correctly oriented in spite of the inversion on the retina, as a pseudo-problem, and it does not deserve the endless discussions that it has given rise to. According to Mach, the essential fact is that all sensory systems of spatial character, no matter how different, are related by a common functional link, that is, the movements used for orientation. Thus, the same spatial orientation is established for all kinds of sensory systems since they jointly participate in spatial perception.

Some classical authors already mention the visual-tactile functional complex that is involved in visual image orientation, and for those with an empirical conception, visual orientation seems to depend entirely on tactile support, the visual factor playing no role. Visual orientation is then conditioned by touch, understood in a wide sense (movements, static sensations, etc.). That interpretation is adopted by authors of the 18th century, for example Condillac (1754/1821), and those of the late 19th century such as Helmholtz (1896). Likewise, the important experiment of Stratton (1896, 1897) seems to support this view; it does not matter if the image formed on the retina has one direction or another, since in both cases the adequate correspondence between visual and tactile sensations of movement is acquired through experience, and the result will always be the same.

In such theories, visual image orientation is established entirely by the tactile component, so that the visual factor, by itself, is overridden as regards orientation. This means to discard fully and definitely the question of retinal inversion. Nevertheless, since opinions of all kinds are not lacking, we could also mention, as a curious detail, in favor of the visual factor (visual image orientation provided by the retina), the opinion of Buffon (1880), who imagined that newborns, lacking associations or adequate references,

would have inverted vision because they would use the information from the retina for spatial localization. Leaving the appraisal of this hypothesis aside, it happens that the issue of visual image orientation and the old associated problem of the inverted image on the retina acquire a new perspective in view of the phenomena about pathological inverted vision studied here. In this way, the retinal factor turns back to recover its meaning because it is able by itself to decide visual image orientation, since the inversion in our brain-injured patients has to be put in relation to the inverse localization of the spatial retinal local sign.

The theory of the functional visual-tactile complex, according to which visual image orientation is determined by the second factor (touch), is still acceptable on condition of recognizing that the visual factor can also determine the orientation, a condition that has been generally denied by all supporters of the commonly accepted theory of the mechanism of visual image orientation. In the pathological cases with inverted vision, something similar to the first phase of adaptation in the Stratton experiment occurs, that is, incongruent orientations for sight and touch coexist until adaptation is acquired again and correspondence between sight and touch is recovered. However, in our pathological cases the genesis is very different; sight and touch are in opposite orientation because the retinal factor gains autonomy and conditions by itself spatial orientation of visual sensations. The emergence of this autonomy is due to brain asynchrony, which breaks the functional link between the visual factor and tactile factor, but if the stimulation intensity becomes much higher, and the whole brain mechanism of the visual-tactile complex goes into action, then the correspondence between the orientations is obtained again and the orientation determined by the retinal factor is excluded.

14.2 Disaggregation and degradation in visual image orientation

As we know, nervous asynchrony gives rise to the fundamental experiment of functional time lag and dynamic reduction. Functional complexes become disaggregated and simplified, and in the case of visual image orientation, the orientation of the image is no longer adapted to tactile orientation, body movements, etc. Consequently, we could say that visual image orientation is as if it were drifting, with no other rule of orientation than that provided by the visual system itself, thus manifesting the retinal inversion. The inverted orientation is therefore the result of a dynamic reduction of the activity of the visual-haptic complex, in such a way that the simplification (or reduction) of the mechanism of visual orientation excludes the secondary or less immediate factor (tactile factor). The simplification of complex functions, which means their *disaggregation* and *degradation*, is the rule in cerebral alterations, and now in the case of visual image orientation, we again find similar conditions to those of green chromatopsia in the perception of white. In this last case, due to a considerable difference between the excitabilities of the primary colors (these ones needed to generate white by their joint stimulation), white cannot be obtained, and a pale green appears in its place. Analogously, in the case of visual image orientation we are facing a difference between the excitability

for normal and inverted (or variably tilted) image orientation; and instead of the unitary function that reacts according to all-or-nothing, a wide interval is established between the factors that make up the functional complex. At the lower end of the interval is the isolated visual factor that originates inverted vision, and at the upper end (highest level) is the correct vision caused by the unitary action of the brain mechanism in visual image orientation, i.e., by the tactile support. Depending on the intensity of stimulation, the respective sensation level is reached, and either visual inversion (retinal action) or normal vision (tactile or haptic action) is obtained. This pathological interval creates a new situation that has no antecedent in the sensory activity of a normal subject, thus appearing visual inversion in the same way as green chromatopsia in the case of colors and as replacement of white.

The magnitude of the abnormal interval depends on the degree of asynchrony, which is not always so pronounced as to cause a complete disaggregation of the visual-tactile complex. Even in the M case, a complete inversion of 180º is not exactly reached, and if the cerebral deficit is offset by various central summations, the limit of inversion is reduced, appearing only moderate tilts. Also, we know that depending on stimulation conditions, it is possible to gradually go from the inversion to the correct image orientation, following specific sensory laws. Thus, a certain balance is always stablished between the two modes of orientation, correct and inverted, depending on the intensity of the visual stimulus. If a deviation occurs, even if small, it means a failure of the haptic factor (which enables the correct orientation) and at the same time a certain autonomy of the retinal factor. If there is complete inversion, it means that only the retinal factor is acting, the tactile or para-visual influence being totally excluded.

In cases of severe disorders, such as M, even without total exclusion of the tactile factor since inversion is somewhat incomplete, the orientation of the visual image does not easily go from one extreme to the other in the interval mentioned above, even if stimulus intensity is greatly increased, and there are always a few degrees left to reach a completely correct orientation. Asynchrony is so pronounced in this case that even with intense stimuli it is not possible to recruit all the nervous elements of the tactile factor. The same happens in the case of white, which does not appear completely clean of a green hue, even under the most intense illumination. However, with facilitation, the magnitude of the interval (or asynchrony) is much less, and then it is feasible to achieve a normal orientation by an adequate stimulus intensity.

Since visual activity is altered in its totality and the diverse functions present a same asynchronous disorder so that sensory manifestations change remarkably according to stimulation, alterations in visual image orientation do not remain isolated from alterations in other visual activities that appear simultaneously. Let us recall that when the image is tilted, the greater the tilt, the greater the alteration of color and shape.

The empirical rule that correct orientation can be expected in distinct vision already indicates a solidary activity, and also easily leads to the idea that distinct vision and correct visual image orientation are approximately isochronous functions, i.e. of the same excitability. Thus, in our subjects, as soon as chromatic irradiation destroys or disaggregates image details of the test arrow, a deviation of its orientation appears.

Disaggregation and degradation of sensory activity do not represent a sensory chaos, but they manifest themselves according to the dynamic reduction mechanism, that is, sensory activities appear in a particular sequence according to their new characteristics of nervous excitability; those of greater complexity being the most difficult to be stimulated. The general law of Fechner (sensory perception proportional to the logarithm of stimulus intensity) (Fechner 1860), is still valid along the new abnormal interval that includes such sensory activities. Therefore, there is no sensory chaos or annulment of fundamental laws, but another order with the same laws, in other words, a nervous organization at a different scale from the normal one.

As for the nosological meaning of inverted vision and other similar alterations, they do not represent independent entities or syndromes, and only represent multiple aspects of the general disturbance suffered by visual functions due to a single lesion. Moreover, the lesion not even needs to be located in the striate or extrastriate visual cortex, since because of the dynamic repercussion due to the joint activity of the cerebral sensory cortex, a parietal lesion, for example, easily alters visual activity, and along with it visual image orientation. It should be noted that we have observed alterations in the orientation of the visual image in both left and right brain lesions.

For the moment, without pretending to have conducted a complete study on the orientation of the visual image, we put an end to this issue.

Schema in visual perception

15 Schema in visual forms

15.1 Cognitive visual schema

The cognitive visual schema corresponds to the highest degree of organic differentiation of sensory structures by accomplishing a stylized and more properly characteristic perception of objects and sets of them. Cognitive schema makes it possible intelligent behavior, that is, abstraction. The function ‘schema´ does not imply any singular activity; it is derived directly from the shape structure of which it constitutes a special feature.

Although it is now impossible to determine experimental curves of the phenomena, given the nature of this topic, it is feasible to perform certain dynamic variations by means of facilitation that allow this topic to be studied in depth, in the same way as in the previous study of visual perception of shapes.

As we have observed when studying the perception of objects, these are almost always correctly interpreted by subject M provided he counts on sufficient time and favorable conditions for vision. However, his perception under unfavorable conditions does not occur in an immediate or unitary way, but by means of successive partial aspects, and in many instances, the correct interpretation is reached mainly thanks to characteristic details (for example, the handle to recognize a key) without getting a clear idea of the whole object examined. If agnosia can often be overcome by indirect understanding tricks in the case of physical objects, the situation is very different when it concerns the understanding of schematic drawings of these objects.

It is well known that patients with visual agnosia fail much more frequently in understanding drawings of objects than in understanding those same physical objects when they look directly at them, in which case hardly any significant defects are committed. It means, using the nomenclature of brain nosology, that visual *asymbolia* occurs more easily than visual *agnosia* of objects. The distinction between the different behaviors when subjects are faced with physical objects and schematic drawings is of utmost importance for the correct understanding of visual agnosia, using this term in a general sense. Comprehension of physical objects and comprehension of schematic drawings are successive phases of sensory organization which are altered simultaneously in agnosia, and only by means of a meticulous examination and adequate tests it is possible to clearly discover the agnosia for physical objects. By contrast, the defect in schemas is much more easily revealed in view of their shortened or summarized character

and the imaginative effort required for their understanding. Schemas are originated directly from physical objects through a stylization process that gives rise to "*strong forms or figures*" in the language used by the Gestaltists. The demonstration that such stylization exists and it is not pure explanatory phantasy can be found not only in certain experiences of simplification of forms such as in tachistoscopic vision or when they are represented in the imagination, but also in the fact that representation through drawing would be impossible without it.

The disaggregation and diffuse conception that we have already observed when previously studying the perception of forms and objects, is much more pronounced and with very remarkable features in schemas, as it results from the following tests with simplified drawings to be recognized by subject M and subject T.

When M is faced with the drawings of Fig. 73, in binocular vision and good illumination, the following results are obtained.

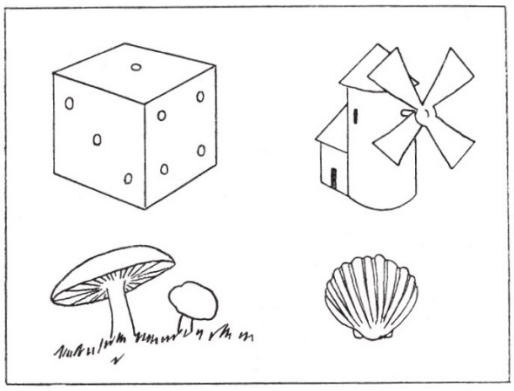


Figure 73. Simplified drawings to test visual schema. In general, they are well recognized by subject M under facilitation by muscular effort, whereas failure is the rule in the inactive state (see the text).

With respect to the die, M in inactive state does not know, he declines to interpret the figure after having looked at it intensely. Finally, he believes it is about a house with windows, but he cannot be sure. M under facilitation by muscular effort takes just one second to answer "it´s a die".

With regard to the windmill, M inactive only perceives a house, and when asked about the sails, he finds it very hard to catch sight of them. First, he only sees the two sails on the right side. He confuses the other two sails with the drawing of the house. They are perceived *in a different way*. At last, he succeeds in recognizing all of them independently from the rest of the figure, but he does not get to see anything but "a house and a cross". The meaning of the whole escapes him. Under facilitation by muscular effort he realizes after a short time that it is a windmill.

As for the mushrooms, M inactive perceives them as trees, but under facilitation the interpretation is correct.

As for the sea shell, M inactive at first does not know, a bit later he takes it for a fan. Under facilitation he says “a clam”.

When subject T is faced with these same drawings shows a certain deficit; nonetheless, using only his right eye, the one with worse vision, he interprets correctly the die and the shell, although the latter perceives it at first as something shapeless. As for the mushrooms, he believes they are just “a flowerpot”, and as for the windmill, he makes a misinterpretation of the same type as that made by subject M. Initially, he only perceives a house, but realizing that his interpretation is not quite right, he tries new exams of the drawing. The sails have gone unnoticed to him, partly confused with the figure of the house, as in the M case, and only after much attention, when asked about their presence, he answers: “It is a sentry box and a cross” (he has been a soldier for a long time). He also thinks of a church and a castle, and long after he understands it is a windmill. All these difficulties do not appear in binocular vision, the interpretation being then quick and correct.

Figure 74. Simplified drawings to test visual schema. In the M case, although an interpretation close to the correct one is achieved under facilitation by muscular effort, mistakes still persist in all the drawings (see the text).

When M is faced with the drawings of Fig. 74, in binocular vision and good illumination, the following results are obtained.

As for the candle, M inactive finds very difficult to know what it might be, even after a long time; it seems to him it might be a tower or a tree (cypress?) and he finally opts for a chimney. Under facilitation by muscular effort, his perception does not improve much, and although he does not reach the correct interpretation, his understanding of the drawing is enriched to a certain degree; thus, he thinks of a smoking chimney or a fountain for street decoration.

As for the weight, M inactive says “a bottle”, and under facilitation, “a glass jar”. Facilitation does not correct the defect although the response seems a bit more appropriate than in the inactive state.

As for the cheese, M inactive says “something …”, and he cannot say anything else. But under facilitation he says “it's not a thing, it looks like a die, but it’s not sure”.

As for the whistle, M inactive thinks it looks like a shoe or also a bottle. Under facilitation he says “something like a bottle, but it’s not sure”.

In these other figures, it is verified that even in binocular vision and under facilitation by maximum muscular effort, the correct interpretation of the schematic figures remains impossible for subject M, although more or less plausible and approximate interpretaions are achieved. The perception is more accurate with facilitation than in the inactive state, but in any case, the difference in perception between these two states is not as clear as in the drawings of Fig. 73, which are however of the same type of simplification.

However, subject T, even using only his right eye, recognizes the four drawings of Fig. 74 with relative ease.

With regard the drawings shown in Fig. 75, when subject M is faced with these figures, in binocular vision and in good illumination, the following results are obtained.

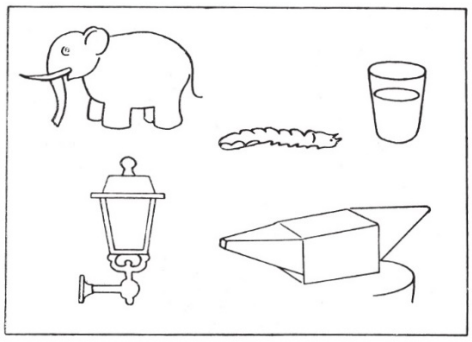


Figure 75. Drawings to test visual schema. Remarkable illusions or figure deformations are perceived by M, disappearing under facilitation by muscular effort (see the text).

As for the elephant, M inactive says “a dog”, but under facilitation says “elephant” without any difficulty. In the inactive state, he did not see the trunk and the head seemed to him to be prolonged towards the tusks. Under facilitation, this illusion disappears and in addition he perceives the tail well.

As for the street lamp, M inactive says “a pitcher”, and under facilitation “a street lamp”. In the inactive state he does not pay attention to the device holding the lamp, which changes its meaning, but under facilitation the attention is equally distributed throughout the figure.

As for the caterpillar, M inactive does not know and says “perhaps it’s a spike”, but under facilitation says “worm”; he distinguishes well the head and even the horns.

As for the glass with water, M inactive says “two glasses” (one inside the other!), and under facilitation “glass with water”. The change in his interpretation is remarkable.

As for the anvil, M inactive has no idea, even after a long time. Under facilitation, he has sometimes failed, renouncing to any interpretation, but occasionally has achieved the correct interpretation. It seems that he does not pay enough attention to the pointed ends and concentrates on the central block.

Regarding the T case, he makes similar mistakes, also due to paying or distributing the attention heterogeneously on the drawing. Such mistakes are more easily made in

monocular vision, but much less so in binocular vision and provided there are good conditions of luminosity and distance. For example, in monocular vision he takes the elephant for a wild boar, whereas in binocular vision he quickly realizes that it is an elephant. It seems that in the first attempt the trunk went unnoticed to him, hence he got the optical illusion of perceiving a boar. In the case of the glass, using only the right eye he makes the same mistake as the previous subject by thinking that there were two glasses (he says "one glass…and a smaller one inside"), whereas with the two eyes he says "glass with liquid". In the case of the caterpillar, he only sees "one line… two lines with sharp point making curve…", even binocularly. Other figures, such as the street lamp and the anvil, although he generally interprets them correctly, has sometimes failed in tests on different dates, thus, he has taken the anvil occasionally for a Civil Guard three-cornered hat, and the street lamp for a watchtower, which means not having paid attention to the support of the lamp which is what gives its character. Sometimes he has also thought that it is a jar; all confirming that his perception has been fragmentary. However, when insisting, he always comes to understand the figure correctly ("of this… that illuminate the streets…, a street lamp"). There are, then, undoubtedly disturbances of the same type as in the M case, that is, disaggregation and fuzzy understanding, although attenuated.

That the deficit in the understanding of such schematic drawings is due to the peculiar alteration in the organization of forms and not simply to visual acuity defects – although these must be considered – is evidently confirmed by the behavior of these subjects when faced with overlapping drawings, as in the figure of Poppelreuter (Poppelreuter 1917) shown in Fig. 76.

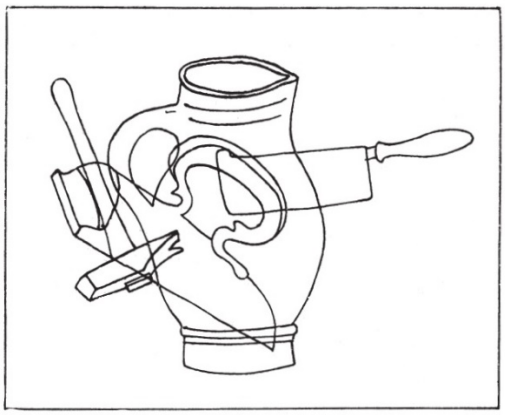


Figure 76. Figure of Poppelreuter (Poppelreuter 1917) to test visual agnosia (see the text).

Subject M sees only many lines in that figure at first, and desists from interpreting it. By insisting and encouraging him to pay more attention, his perception in the inactive state, in binocular vision and with good illumination is as follows. After quite a long while of looking and searching, it seems to him that there is a jug. He is told there are more things to find, and after a long time, he only gets to perceive part of the cleaver that he takes for a saw. He thinks that the rest of the figures are drawings or ornaments of the jug. These tests, and especially the figure of overlapped drawings, bother him greatly when

he fails and he complains strongly. Paying much attention and patience, sometimes he has been able to find, after many minutes, other objects in the figure, such as the cleaver and the hammer, but not the iron which appears particularly difficult for him. In any case, he loses many details that he does not properly understand, e.g., he considers the handle of the jug as something different and independent of the jug because he had not perceived it in the first recognition of the jug.

Under facilitation by muscular effort, his perception becomes firmer and he finds successively the various objects. First the jug with its handle, then the cleaver or handsaw or sawblade, he cannot be sure which of the three would be the correct answer. Occasionally, in the inactive state, he takes the cleaver handle for the jug handle, but this never occurs in the facilitated state. Next, he usually finds the hammer, which he perceives only exceptionally before the cleaver, and afterwards some difficulty appears, even under facilitation, to find the iron, and after a long time he says "another thing with a handle… an iron". It is necessary to indicate that initially, when he has shown resistance to interpret the figure because it seemed very difficult to him, the objects to be looked for (hammer, cleaver, iron) were named to encourage him to look for them, which has undoubtedly facilitated his task; otherwise it is possible that they had remained unrecognized.

Subject T also, faced with this figure, presents serious difficulties of understanding; on one occasion he saw only "a drum with sticks, everything mixed up", without attempting to interpret anything more. On another occasion, he perceived a jug with two or three handles (the ones sticking out). It is remarkable and quite characteristic in subject T as well as in the previous one (M), the naturally way they respond and the seemingly certainty of having been right. When subject T is asked about the other drawings on the figure, he says, after some thought, that the jug is cracked. All this occurs commonly in monocular vision, which as we know corresponds to a functional level slightly higher than that of subject M in binocular vision and under facilitation by muscular effort. In binocular vision perception improves, but still he hesitates long before interpreting the overlapped drawings of the figure. In addition, he makes many lateral movements of his head and moves or turns the drawings as if trying to find favorable positions for the interpretation, apart from the fact that, as we have said, the movements or shakes of his head favor visual function by summation. Finally, he finds the hammer and then the cleaver, but fails with the iron, and only by telling him his name and that he must find it, does he find it after some time of searching.

This last test of the overlapped drawings shows even more clearly than the previous figures that, in fact, the mistakes in the recognition of schematic drawings are due to true misunderstandings or illusions in the appreciation of the configuration because, given the instability of the perception, there is a tendency to formation of figurations that respond to a simpler state of equilibrium in the sensory field.

The data shown on the behavior of these two subjects in the interpretation of schematic drawings are only the most characteristic extract among the numerous tests carried out during the several years that they have been examined. Therefore, the answers shown have not been obtained at random, but represent a very clear and constant indication of the agnosic disorder in these subjects. From the above tests, it follows that

agnosia in the M case is revealed very easily, not yielding completely even with facilitation; for the T case, the behavior is better than that of M under facilitation, but there is still a significant deficit.

Regarding the Schneider case of Goldstein and Gelb (1918), the authors point out that when the patient inspects the figure he indicates the various characters but does not understand the figure in its entirety and only interprets partial aspects, which segregated from the whole acquire a new meaning. These authors, adopting the viewpoint of the Gestaltists, highlight the confusion and instability of the background and shape in the figure in such a way that new aspects appear that the normal subject would never be able to see. In more recent studies, Gelb (1937) attributed most of the alteration to a fragmentation of shapes as well as to a reduction of pregnancy (meaning) in their organization. All these circumstances also occur in our two brain-injured patients and in some others we have studied with visual agnosia. The interpretation on the alleged motor recognition of the Schneider case, which must be discarded although it continues to be erroneously admitted in subsequent publications, affects the theoretical interpretation but does not change the intrinsic phenomena of recognition of schematic figures, albeit the very important issue about the modifications in nervous excitability is left aside.

Considering all the results of the previous tests, it must be admitted that the wrong interpretation is mainly due to a change in the configuration, i.e., to a metamorphopsia which leads to a metamorphopsic pseudo-agnosia, as we have indicated when dealing with shape perception in previous sections. One might think that the subjects invent the answers at random, but this would be a very superficial appreciation, and nothing, not even remotely, supports it when the nature of the various answers and above all the set of answers is carefully examined. A remarkable feature we have already pointed out is the attitude of conviction with which they assume certain faulty or somewhat approximate interpretations. This suggests that they have indeed perceived a certain illusion in the figure, which for a normal subject would be impossible, but in them easily takes place given the instability of the sensory organization. Therefore, there is no fortuitous invention, but an *altered view* of the configuration of the drawing. In other words, the patient usually answers in accordance with what he is capable of perceiving, and if it looks like an unfounded invention, it is due to the fact that figures lend themselves to be seen under new aspects. However, the alteration in understanding schematic drawings does not only lie in the perceived distortions; there is also an imaginative difficulty, that is, a deficit in the construction of the mental representation on the basis of highly stylized or simplified drawings, which would constitute the intrinsic disorder of schema. In any case, a rigid separation should not be made between perceptual alteration (alteration of what has been seen) and agnosia in the strict sense of the word (constructional deficit). Both contribute equally to the failure in understanding schematic drawings, and although they are of a somewhat different type, they are stages that condition each other in the organization of shapes in the sensory field.

By examining more closely the circumstances involved in this disorder, it can be noted that some factors influence each other and contribute jointly to determine the alteration. Thus, we find *illusions* or deformations of the figure (an “a” is seen as two superimposed zeros, the elephant is perceived as deformed, etc.), *disaggregation* or

fragmentation of the whole (the case of the windmill broken down into a house and a cross, the case of the street lamp, etc.), and also *inability to construct* or imagine an adequate figuration on the basis of schematic lines (example of the cheese, candlestick, etc.). All these factors can occur separately, depending on the kind of drawing, or simultaneously as in the figure of Poppelreuter wherein the overlapping drawings increase the instability of perception and understanding, causing both illusions (jug with several handles) and disaggregation (cracked or decorated jug), and also difficulty for appropriate imaginative construction. In short, perception is diffuse and labile, so the equilibrium of shapes is altered, and consequently their phenomenic aspect, as it occurs in visual-geometric illusions in normal subjects, although here it is due to other reasons. There is also constructive inability, e.g. difficulty in imagining the relief in flat drawings without shadows, deficit in the assessment of the whole, etc. Thus, there is both a reduced sensory activity in perception and in the imaginative construction, and both are expression of the same fact, *the reduction of pregnancy (meaning) in the organization of shapes*. Mere perception and mental construction are both continuation and reflection of each other and, ultimately, aspects of a same process whose basis lies in the functional reduction of the sensory field. A radical separation or distinction between both would then be rather contrived. The facilitation phenomenon, which in the M case modifies in a very important way the functional level, improves clearly the perception of schemes due to the reorganization that is established in the sensory field, this one broadly understood as the set of sensory organization processes.

15.2 Visual behavior

An alteration in the cognitive schema distorts to a high degree the intelligent behavior of these subjects, since this kind of activity, intelligence, is solidary of the structure of the sensory field, and following the Gestaltists, there is no possible distinction between sensory and intellectual functions, to the point that it might be admitted that “intelligence is a spontaneous mode of organization”. Thus, the general alterations we have found in the conceptualization of schematic figures, again participate in various alterations of higher visual behavior giving rise to what is commonly considered a visual intelligence disorder. Such alterations are produced, for example, in the recognition of situations or scenes in drawings, in the construction of drawings and puzzles, etc., and very especially in the abstract behavior regarding colors, i.e. everything related to color agnosia. We shall deal with these topics succinctly, by restricting ourselves to the essential aspects.

A characteristic example of agnosia in the M case, taken from ordinary life, is the recognition of banknotes. These are recognized not by their value numbers printed on them but by the general format, size and ornamentation aspect. Thus, he knows that those of small size have a certain value, the largest and longest ones have another value, those with a more square shape have a different value, etc. In many cases, the figures illustrating them are recognized more or less correctly, but a typical difficulty is to find the numbers that indicate the value of the bill since they are in the middle of all kinds of arabesques which seriously alter their recognition. Thus, for some types of bills, it seems impossible

he finds out the numbers in spite of being visible in a variety of ways. When he tries to find those numbers, he seems to be in a situation similar to that of a normal subject in the face of an ambiguous image (Vexierbild by German authors) within which there is another hidden figure different from the one seen at first. This difficulty appears even in the best functional visual conditions, that is, in good illumination, at as near vision as necessary, binocularly and under facilitation by muscular effort. But these conditions do not prevent the defect to subsist, and the numbers are almost impossible to be perceived. When, in view of this result, he is shown the drawing containing the number 5 on certain five-pesetas bills (see Fig. 77), the situation does not improve. He only sees many lines, and by assuring him there is a 5 and he should see it, no matter how hard he tries, he does not see anything other than a 3. Thus he perceives only the strongest shading of the number 5 which, in fact, resembles remarkably a 3 if one disregards some fragments. The rest of the drawing of the number 5 remains mixed up with the background, without acquiring individuality, making impossible for him to determine its shape, no matter how much help be given to him. He affirms that he only sees the indicated number 3. In short, in this test, the subject even in his optimal functional state cannot distinguish or segregate the figure of the number from the ornamental background; he can only isolate the more solid shadow which adopts a different meaning. Therefore, there is a marked organizational deficit in perception, due to its instability, and especially its diffuse character.

Figure 77. Bill of five pesetas. Subject M binocularly and with facilitation by muscular effort cannot see the `5´, but only a `3´ corresponding to part of the shading of the `5´.

These same facts are demonstrated in many other examples such as in incomplete drawings of the face of a person. In the inactive state, the defect goes easily unnoticed, and even if the mouth or the nose is missing, he recognizes the figure without noticing any defect, due to a diffuse perception. However, by means of facilitation by muscular effort, the defect is immediately pointed out. Also, the negative and the positive image of a same photographic portrait of a person are taken as identical in the usual state of the patient. He also does not distinguish between a photography taken from a living person and a photography taken from a sculpture, he only recognizes that in both cases there is a person. In photographs of big size and pictures easily visible by the subject, he only perceives the general characteristic and always responds “one person”, but he hardly indicates gender or age. For that purpose, he is guided by details, long hair in women, moustache and tie in men, etc. For this reason, in an old picture showing a man with long

hair, he thinks the person is a woman, even though he has been made aware that he has a moustache, but he is not able to solve the problem and has no interest in giving further explanations. All these difficulties disappear automatically with facilitation by muscular effort, and all the peculiarities of portraits, drawings, prints, etc. are perceived. In this case the perception becomes more defined and firm, as we know from other tests. However, in certain tests, some difficulty arises even under reinforcement, both in subject M and subject T. Thus, these subjects generally fail when trying to distinguish between very simple drawings that are a little different, because they seem to them visually the same.

Much greater difficulty is shown in the well-known ambiguous images (drawings with double meaning), i.e., figures in which the whole configuration can be changed by varying the attention or manner of looking at them. It is clear that already for the normal subject, certain difficulty arises, and it is no surprise that these subjects fail when they try to move from a type of visual configuration to a very different one, since it is already enough achievement for them to grasp the most evident or immediate configuration. The remarkable thing in them is rather the difficulty to admit the double meaning in spite of all explanations and aid given to them. There is a sort of adhesiveness to the first meaning which prevents the transit to a new understanding. Such mental viscosity or stickiness (called *Gebundenheit* by German authors) is very typical of mental operations in all kinds of brain diseases, and particularly it is very pronounced in brain-wounded patients suffering from agnosic disorders. It is more patent in subject M than in subject T who, by means of very detailed explanations is able to understand the double meaning of a simple ambiguous image.

A not easily accessible exercise for these brain-injured patients is putting together a puzzle, no matter how simple it is, for example an almost life-sized face, in color, divided obliquely into four parts. Subject M desists from the first moment to do the test in view of the difficulties. In another test on a different date, when he finally achieves a certain regularity in the construction of the figure, it results incorrectly oriented in its whole, although he believes it is correct. Subject T case also shows similar though more moderate difficulties, which vary according to the complication of the test. In the constructional difficulty concur both perceptive and schema defects. In the first group we consider, for example, in addition to the deficit of shape appreciation, the disorder in visual image orientation, which can hinder the orientation of the fragments, and also a somewhat different kind of defect that we call orthogonal disorder, discussed further below. As said, the intrinsic disorder of schema acts as well, leading to an ideational agnosia in the sense given by Liepmann (1908), which makes construction very difficult because the schematic conception is precisely here essential and indispensable to arrange the parts.

Similar considerations could be made regarding the difficulties to copy drawings, which reveal disorders of the type of *optic ataxia* and also of the *constructional apraxia* of Kleist (1911) in anyone of the two wounded subjects.

Finally, in the tests with the well-known sheets of Binet (1903) for clinical examinations of intelligence, the complex agnosic disorder of these subjects can be clearly seen.

Figure 78. The sheet of Binet (1903) "Snowball". Subject M interprets separately and somewhat incorrectly the different figures, but the meaning of the scene escapes him. He does not find any explanation for many of the details, others give rise to illusions, e.g., he believes that the boy's cap near the window frame is a broom. With facilitation by muscular effort, the partial understanding improves a little, without important advances.

Subject M, in the first sheet entitled "snowball", shown in Fig. 78, gives the following interpretation: "a man quarrelling with another one is grabbing him by the hairs and pulling it upwards… and another one poking his head out of a door or window (he refers to the hidden boy), I don't know what it is, window or door; when he lets the other one go, he'll grab this one". He is asked "what's going on up here?" (indicating the left angle to him). He answers "a woman or a man behind a window and here there is another small window, but there is nothing". Asking him again "and what is this?" (pointing to the boy's cap), he responds "I don't know what it'll be". After a long time, he says that it looks like a broom, because he extends it upwards along the window frame and wonders if it really is. Questioned about the boy's blackboard, he thinks it is a book.

These results are obtained after looking closely at the sheet for a long time and helping him to pay attention at what he should see. He does not understand the meaning of the scene at all, mistaking the boy with the adult, the woman with the man, etc. Under facilitation by muscular effort, everyone is better distinguished, but he continues ignoring what had happened, and even the illusion of the cap is maintained for a while or, in any case, he does not know what it could be. There is, then, a perception defect and also an inability to understand the whole situation, since only partial aspects are interpreted; thus showing the inability to construct a schema or idea about the scene presented.

Subject T, although a bit better, does not go farther either in the interpretation. He answers "the mother is beating the child and another one who's hidden". He does not recognize either the snow that is hanging, or the blackboard or the cap that is falling down. Feeling that his answer is not well received, he adds "she grabs him by the hairs for him

to go to school". As in other tests of this type, subject T resorts to all types of head movements and involuntary efforts when examining the scene. In the other two sheets, subject T succeeds relatively easily in the second one (it is the game blind man's bluff) and he fails in the third sheet (the gentleman's greeting), more difficult, since he says "he's talking with his girlfriend and he's had a fight with somebody and a woman arrives and pulls them apart", although he gets it pretty close. Subject M with facilitation by muscular effort and very indirectly understands the scene of the second sheet, although many details escape him; and regarding the third sheet, he does not properly understand who the people on the scene are, and he does not get the meaning of the situation at all, although he tries diverse explanations.

In these tests, apart from a diffuse perception, which harms the recognition of important details for a full understanding, the main defect surely lies in the inability to pay attention to the whole, i.e., to integrate it into a unit, as we have already noticed in previous tests (e.g., in the windmill and the street lamp), although the scenes of Binet are more complex. In any case, we find different degrees of a same type of functional disorder. This inability to integrate or apperceive the whole scene hinders the understanding of the situation. On the other hand, it is also possible to work on it in a successive manner, as we have mentioned when studying the perception of shapes and objects; but in this way, only juxtaposed aspects or partial actions are captured at most, without achieving the true total meaning. Therefore, the defect is not only a lack of unitary visual perception, but more exactly, difficulty of constructing an ideational schema corresponding to the whole of the scene, there is, then, a constructional inability, like in the case of putting together a puzzle or copying a somewhat complicated geometric drawing, the figure of a house, etc. This kind of disability occurs in the so-called *simultagnosia* of Wolpert (1924) in which the appreciation of a set of objects representing a scene fails, whereas each one of them, individually, can be recognized quite well.

Concerning the ability to visual mental representation, that is, the ability to evoke images in the absence of the object, is not abolished, even in subject M, although it can be slow and defective. Therefore, agnosia for mental visual representation (Charcot 1889) is not present in our subjects, contrary to what Goldstein and Gelb (1918) argue about their patient Schneider, in whom the difficulty for mental representation would play an essential role in the genesis of most of his disabilities, according to these authors.

In relation to the cognitive schema disorder, i.e., the inability for abstract ideas, we must mention the manifestations of *color agnosia* in our two subjects, namely, anomalies in understanding the name of colors. Besides the fact that colors are altered from a sensorial point of view (dischromatopsias, chromatopsia, etc.) and also spatially (flat colors), our subjects present a disorder of agnosic type that fits very well with a failure in the cognitive schema. When our subjects perceive colors well and can distinguish them from each other, they often cannot name them. The patient in question spends a lot of time until he gets the name right, which he does after some exercise because at the beginning of the test he usually does not find the right name or gives a wrong one.

Likewise, when a color is named for them to choose among several other colors, they do not immediately understand the denomination and remain puzzled for a while without knowing what is it about. Also, the evocation of colored bodies or objects is

deficient; the M case, for example, says from memory that violets (flowers) are green. It is typical in these two brain-injured patients and others we have studied, the indirect procedure to understand the name of a color. For example, when they are asked to separate the orange color from a pile of colors (M being under facilitation), they get perplexed, without being able to take a decision, showing that the indicated name does not mean anything to them. However, after a period of time, they decide to examine the colors and can finally extract the color in question. Interrogated about the cause of the first indecision and the correct behavior much later, it appears that initially the orange color does not make sense to them and is not conceived as corresponding to something with color, but later the name of the color has evoked the fruit of that name, which does offer a meaning of color to them, and this has been the starting point to reach the color in question. Therefore, they need to make a detour by means of the concrete representation of "orange fruit", since it is impossible for them to understand the color directly through "orange color". Due to these circumstances, they commonly use, in the denomination of colors, much more concrete names than the more conceptual or abstract ordinary ones; examples are blood color instead of red, sky color instead of blue, grass color instead of green, straw color instead of yellow, etc. They get oriented immediately with these concrete designations whereas the usual name generally appears to them vague or indeterminate, and some names, such as blue or yellow, particularly difficult. The disorder of color name understanding in these brain-injured patients is much more pronounced for those colors to which they present blindness or weakness, i.e., yellow, blue and violet in subject M, and blue in subject T who tends to confuse it with green. Thus, there is an agnosia for colors with marked predominance for some of them, which corresponds to partial color agnosia, described by Peritz (1918) in a case of lesion in the angular gyrus. This fact really shows that agnosia is more severe where sensory disturbance is also more pronounced, thereby revealing the unitary nature of the disorder.

In the M case, the effect of facilitation in this color agnosia is much less than in other functions, and although some improvement can be achieved, it is not as regular as in other functions. We have already observed the same in other tests on cognitive schema.

As we said, color agnosia is influenced by the alteration of the schema, i.e., by the alteration of the abstract behavior. Gelb and Goldstein (1924), in a very important study, independent of that of the Schneider case, dedicated to the amnesia of color names, base this disorder on a category-specific deficit, that is, a loss of the *category* function (loss of abstract language molds). This conception is nothing more than the psychological aspect of the schema disorder (disorder in the ability to construct ideas). In fact, the disorder is parallel or identical to the one that is revealed in the tests of Binet (1903), i.e. the whole framework fails, and behavior becomes more concrete, particular or partial, as expression of a deficit in the structural differentiation process.

For now, we shall limit ourselves to these brief indications about color agnosia in our patients. This disorder will be further studied when dealing with language disorders in relation to auditory functions.

15.3 Structure of visual agnosia. Schema dissolution

Theories on the structure of visual agnosia, and in general of all types of agnosia, could be classified into two main groups. In a first group we consider theories that, starting from the ideas and methods close to those of Wernicke (1895), are based on the theory of primary and secondary *identifications*, resulting in the two forms of visual agnosia of Lissauer (1890), apperceptive agnosia (inability to perceive forms correctly) and associative agnosia (inability to recognize forms), depending on the degree of difficulty. Various authors have elaborated somewhat different theories, but always according to these general types, and involving in different ways complex psychological mechanisms of associative and constructive type. At the same time, they assume specific anatomical localizations, and thus the theory of specialized centers and association pathways is then decisive. A second very different and more modern group is initiated with Monakow (1914a,1914b), assuming, contrary to the earlier anatomic-psychological conception, a functional biological viewpoint in which the possibility of obtaining a specific anatomical localization for agnosia is rejected. The disorder is conceived, from a structural viewpoint, as a result of the disorganization of visual processes of very diverse complexity.

Both frames of reference (anatomic-psychological and functional) which assume at the same time very different brain conceptions, have contributed variously to the advancement in the knowledge of visual agnosia. The former, more used with clinical objectives and as a starting point for the description of new agnosic syndromes, has lagged behind in deepening the mechanism of visual agnosia. Instead, the functional point of view of the second theory has undoubtedly facilitated such deepening. In this theory, the more modern conceptions of Poppelreuter (1923), Goldstein and Gelb (1918), etc., could be included, and also Stein (1928, 1930) in relation to physiological aspects of great significance.

The conception about agnosia of Goldstein and Gelb (1918) derives mainly from the study of the Schneider case. Regarding the structure of visual agnosia, they stress the defect of organization between ground and figure, which corresponds to the viewpoint of the Gestaltists about the perception mechanism of figures. It is also considered very characteristic of agnosia, and in general of all kinds of brain disturbances in which higher functions are damaged, the loss of abstract behavior and the destruction of categorial function, so that patients only understand tangible or concrete situations. As for the physical basis of agnosia, it is thought to be a general brain disorder, but further precisions are lacking. However, the Schneider case has been considered by these authors as a very characteristic focal defect. There is not then a sufficiently delimited conception concerning the anatomical issue. As a whole, their idea on agnosia, and also on the brain in general, pays deep attention to the altered behavior of patients, although at times, like in Schneider, they introduce very questionable psychological hypotheses. On the other hand, they build a theory of the brain of biological characteristics that emphasizes both its functional character and its unity. However, despite all this, a physiological basis is still lacking, which gives a diffuse character to the theory of these authors. Apart from

that, it can be said that such authors are the ones who have made the most progress in brain research within the more or less usual currents of thought.

The criterion of Poppelreuter (1923) is also of interest. He considers visual agnosia as a set of perceptual and intellectual defects of a very diverse and complex nature, which can hardly be explained by anatomo-clinical conceptions and associative theories. Through a proper study of experimental psychology in numerous brain-injured patients, this author pointed out the defects in higher visual functions that, adopting very varied types, contribute to agnosia. This author is the one who revealed the slowness of perception in this type of patients, but he still explains it as a result of a higher psychic disorder. If we try to understand the meaning of the research of Poppelreuter (1923) as a whole, we find mainly the following features: psychological character although based on experimentation, extension of gnosic disorder to diverse activities (notation, attention, representation, etc.) and lack of a concrete physiological basis. According to this author, visual agnosia is not a perfectly delimited or theoretically founded set of clinical symptoms, but rather the extraordinary complexity of the higher process of visual recognition results in a multitude of isolated disorders in the differentiation process. Regarding the delimitation that he proposes, he believes that visual agnosia must be considered the summarized expression of all kinds of visual disorders that are not visual field alterations and sensory defects. This delimitation is criticized and rejected by Stein (1928,1930) who, enlarging even more the agnosia concept proposed by Ppppelreuter (1923), relates it to basic alterations in brain excitability, thus involving all kinds of alterations in the visual system. Therefore, in the work of Stein (1928), we find a physiological explanation for the first time, and the various psychological behavioral alterations would be derived from a brain excitability disorder. Thus, he states that a series of phenomena that until now only seemed susceptible of a psychological explanation, such as events referring to attention deficits and others, must find a physiological explanation in alterations of brain nervous excitability (Stein 1928). Consequently, he rejects, rightly in our opinion, that the slowness of perception in patients with agnosia is due to disorders of higher psychic functions as proposed by Poppelreuter (1923), and he lets it depend directly on simple alterations in brain excitability, i.e., on an increase in excitation time (visual chronaxie). Thus, sensory excitability disorder causes a destruction of sensory organization in different ways, according to the degree of the alteration. One might wonder what remains of the individuality of agnosia as understood in the theory of anatomical centers and pathological associations, and we must answer that there is nothing left, since it all comes down to a certain degree of sensory disorganization which depends on physiological conditions of brain excitability. The important physiological approach of Stein (1928,1930) finds a wide and effective confirmation in our studies, and the two cases exposed here fully agree with it. We must then consider Stein as a precursor of this kind of research. It should be emphasized that the ideas of Stein, with exception of his motor theory of perception based largely on the incomplete and partly erroneous studies on the Schneider case of Goldstein and Gelb (1918), are fully in line with reality and considerably simplify the problem of sensory organization.

Even leaving the physiological basis of agnosia aside, attentive examination of the patients reveals that it is by no means an alteration circumscribed to the intellectual

functions of recognition or higher understanding. On the contrary, careful, quantitative determinations always demonstrate there are more elementary sensory disturbances, e.g. in visual acuity, visual perception of motion, stability of the visual field, etc., in addition to slowness and sensitivity deficits in all types of functions, all of which is only revealed by means of appropriate examinations. Such general perturbation has been pointed out by the more modern previously mentioned authors, even though it was already exposed in older studies (Monakow 1905, Siemerling 1890, etc.). Hence, the idea expressed by Lissauer (1890) that a visual agnosia disorder only occurs when any other sensory and perceptual disturbance can be ruled out, should be considered inadmissible because it must necessarily be impossible. This last point of view is already exposed in posthumous publications of Gelb (1925/1926), when addressing the issue of the difficulty in separating seeing from recognizing, since each of these processes is already sufficiently complex, and in no way can be explained by simple formulas of classical psychology.

Regarding our two cases, it is clear that the general disorder of their visual functions is very evident from what has been explained in previous chapters. In addition, it has to be taken into account that the central lesion disturbs the sensory brain in its two halves equally, causing in other sensory systems similar alterations to those studied in the visual system. Agnosia appears as a consequence of the deficit in the sensory organization of visual functions; this deficit is in turn based on the nervous excitability disorder of the cortex. Since functions get reduced according to their degree of physiological demand, shape or figure structures requiring a great complexity of organization are seriously altered, all the more so visual schema which is directly derived from them and constitutes a more complex and differentiated aspect.

In the same way that the ability to discriminate between two simultaneous tactile stimuli is more easily lost than if they were successive stimuli (due to a reduction in functional capacity), the perception of forms and schemas is altered. In other words, the more complex the functions, the more easily they are lost. Therefore, they all are disorders of the same nature and depend on the degree of the functional level of visual activity, and there is no need to resort to secondary or associative influences of other sensory systems, since the diverse visual functions are based solely on the degree of differentiation or organization of their *own* activity. Agnosia involves both an altered perception of forms that causes a diffuse and metamorphopsic vision, and the inability to construct a schema, which breaks down into more concrete or accessory activities in no other way than juxtaposition, due to the lack of a framework or general ideation. This does not mean that a visual chaos be established, but rather a change in vision that prevents or hinders the comprehension of reality, as we have had the opportunity to verify in the various tests. As in other functions, disaggregation of the function `schema´ is characteristic of agnosia, leading to a degradation in the organization.

Summarizing what was exposed in the different tests to our injured patients, the alterations in visual schema can be grouped as shown in Table 16.

Table 16. Alterations in visual schema.

Dissolution of visual schema (visual agnosia)	
Changes in vision	*Constructional deficit*
Illusions by: totalization, equalization, etc. Metamorphopsia (In general, new dynamic equilibrium in figures)	Incomplete apperception Disaggregated apperception Failure of figurative schema Simultaneous agnosia (Binet's tests) Constructional apraxia (build figures, copy, etc.) Color agnosia (In general, concrete behavior replaces abstract or schematic behavior)
Changes in vision and *constructional deficit* influence each other. No clear separation can be established between the effects of both factors.	

All these alterations represent a reduction in the *degree of organization.* This reduction is just a dynamic reduction caused by nervous asynchrony. Concerning this organization, it should be emphasized that there is only a differentiation or progressive discrimination that depends entirely on the dynamic state afforded by the sensory visual field, which in turn is an expression of the level of brain excitability. Such visual organization is developed by itself, that is, without needing support from other sensory systems, contrary to what is generally supposed in the so-called associative agnosias in which a disorder in the association with other senses is assumed (Wernicke 1895, Lissauer 1890, etc.). Depending on the visual structural level, motion is perceived or not, acuity increases or decreases, visual schema is more diffuse or more coherent, but in all these cases it is not necessary to resort to the secondary help of touch or hearing to explain the disorder.

This approach to visual agnosia as a failure in the structure of the schema applies equally to tactile and auditory agnosias, as we shall see later on. Much more complete considerations on the general problem of sensory organization will be developed in the third part of this work.

16 Schema in spatial orientation

16.1 Alteration in orthogonal[9] orientation

We shall now discuss the meaning of the various objects in the outside world in relation to their position and orientation in space. In this case, a spatial orientation schema is developed which imprints a specific character on perception, since perception then presents new and important properties. Restricting ourselves to the essentials, we shall expose the most striking characteristics of the alteration of the spatial orientation schema. These characteristics can be reduced to two types: loss of the orthogonal[9] orientation and alteration of the allocentric spatial orientation. In both cases, the general nature of the disorder is of the same type as that already studied in the cognitive visual schema; that is, changes in vision, constructional deficit, etc.

16.1.1 Orthogonal property of figures

Spatial orientation of figures and objects is a very important attribute in their aspect and recognition, to such an extent that it might turn out to be decisive. This property appears in many different degrees. On one end are figures and objects whose appearance depends almost essentially on their spatial orientation, for example, writing signs (letters and numbers), so that an alteration of their usual orientation makes them recognizable with difficulty, at least initially. These figures then have a strongly developed *orthogonal property*, i.e., the meaning of a figure is highly linked to its usual vertical up orientation (particular orientation in the orthogonal plane to the line of sight); these kinds of figures can be denoted as figures of "orthogonal" signification. Many objects also acquire that property in varying degrees (e.g., furniture), whereas for other figures and objects that continually adopt varied orientation in ordinary life (e.g., tools, cutlery, etc.), their "orthogonal" signification can be null. Accordingly, figures and objects can be classified, in a simplified manner, into "orthogonal" and indifferent.

In the M case, in the inactive state and with good illumination, an indifferent object such as the arrow used previously for the orientation test, is seen in the same position as a normal subject. However, another object with orthogonal property, such as a writing,

[9] Orthogonal orientation means orientation on the plane orthogonal to the line of sight.

which a normal subject is not able to read it or does it very slowly when it is rotated 180º in the frontal plane, is read by subject M with the same ease whether it is presented in normal position or rotated 180º. In addition, he believes and states repeatedly that it is in normal position indeed. Therefore, the orthogonal property has been lost in our case M, and for this reason, figures are recognized independently of their spatial orientation, without offering any change in their aspect. If he believes that the writing is in normal position in spite of being really upside down, it is precisely because of this lack of change of aspect so characteristic for a normal subject who is not used to read an inverted writing. However, this result does not merely imply that all figures behave as indifferent for the M case, since those that are really indifferent, like the test arrow, can be perceived in many different positions and admitted that it is in different positions, provided there is enough illumination to avoid the asynchrony studied. By contrary, writing is always admitted in normal position no matter its real orientation.

We face a complex disorder because, on the one hand, the anomalous orientation does not hinder the perception of the characteristic aspect of the object, contrary to what happens in the normal subject, but on the other hand, since the change in the position is not registered, all orientations are considered equally correct. There is a loss of "orthogonal" signification and, in addition, lability and diffuse orientation perception.

Initially, in 1938, this type of disorder hampered the study of visual image orientation disorder or inverted vision, in the same way as chromatopsia hampered the analysis of color disturbances until chromatopsia was sufficiently clarified. In the previous experiments on visual image orientation, we intentionally used a very simple arrow as a test object, which in addition to the ease of perceiving its shape, allow us to exclude effects such as those provided by objects with "orthogonal" signification.

Although, due to the nature of the issue, the graphic representation of the phenomena by means of curves is not possible, the phenomena perfectly adjust to the dynamism of stimulation conditions and to changes in excitability by means of facilitation, and can be considered among the most remarkable phenomena shown by our patients. For instance, in visual perception of numbers or letters by subject M in inactive state, in good illumination for the shape to be correctly perceived, he recognizes them easily in any orientation, and in addition he believes that they are in normal position. However, as soon as facilitation by muscular effort acts, the illusion disappears right away, and their orientations are perceived correctly. It is a sudden change that allows correct perception, but no data could be obtained from the patient about the transformation process. There are also different types of vision as in other visual functions, as well as intermediate states between correct perception and absolute loss of the "orthogonal" signification, so that we are dealing with phenomena susceptible of being studied in accordance with the same general characteristics (excitability, gradations, etc.) as the other visual functions already discussed.

We have observed the orthogonal disorder quite clearly in our two patientss; in 1938 in M, and in the beginning of 1939 in T, but at present, only subject M has it, generally in the inactive state, although also under facilitation by muscular effort when illumination is weak. There is then a difference in behavior in both patients similar to that found in chromatopsia; very pronounced and completely steady in M and only traces and very

fleeting in T, according to the different functional level of both subjects, which implies a different degree of asynchrony or disaggregation of the sensory structure. Nevertheless, although the T case, due to the greater recovery from his brain disorders, does not present this peculiar alteration in recent years, there is a marked tendency to it in transient states of greater brain deficit (e.g., after epileptic seizures). In these states, it seems necessary to admit a somewhat pronounced degree of asynchrony, at least similar to that of M under facilitation and in monocular vision. It is very likely that the Schneider case of Goldstein and Gelb (1918) presents this alteration as well.

We are not aware that this singular disorder, or at least an alteration as pronounced as that of our two brain-injured patients, has ever been described. However, it is conceivable that many described cases of constructional apraxia, optic ataxia, etc. could participate of this remarkable disorder and even find their main origin in it. We have only found a mention that refers to the Ziegel case of Kleist (1934, p 579), a war-wounded patient with a biparietal bullet shot (entry and exit). In that reference, two lines of the medical history of the patient indicate that he recognizes figures or pictures presented upside down with somewhat more difficulty than in the correct position, but he does not know if they are in correct position, upside down or toward what side they are oriented. This case and others that also present diverse disturbances of the optical apraxia type, are considered by Kleist (1934) as cases of location blindness (Ortblindheit).

16.1.2 Loss of orthogonal orientation (to the line of sight)

We shall expose here the most outstanding manifestations of this complex disorder, which lends itself to a wide variety of studies. It was largely studied in subject M in the summer of 1938; his disorder being more or less in the same state at present.

When diverse figures, especially portraits of people, pictures, etc., are presented to subject M in the inactive state, he recognizes them in the correct or upside down position indistinctly, and he does not perceive the slightest difference, believing that in both cases they are in a normal or correct position. Asking him to place those objects in the most favorable position for him, after having turned them over and over, he ends up often placing them upside down. Instead, he easily recognizes the horizontal position as anomalous, although this position neither is very problematic for him. When he leafs through an illustrated newspaper in the correct position and he finds a difficult figure for him, he spontaneously inverts the newspaper. In his pocket watch, although he handles it in correct position, he reads the time in any position without noticing any change. In general, the correct and upside down positions are indifferent for him, and he only recognizes the horizontal position as anomalous. These tests are carried out in binocular vision and under as good illumination as desired, he being in the inactive state. If facilitation by muscular effort is activated, he immediately realizes the real position of the drawings or pictures, and his behavior then does not differ from that of the normal subject.

Therefore, without facilitation, abnormal orientation of figures does not hamper their recognition, and the patient tends to admit they are in normal position in almost all situations. Two big mutually inverted portraits, one in normal position, the other one

upside down, are both recognized with the same ease, and he considers them equally in correct position. It is evident that the orientation of figures does not prevent their recognition; however, it is not so evident that any orientation be perceived as normal. Is there really an inner reinversion in the imagination, so that figures tend to be seen in their usual position? Or is it just that the patient expresses his perceptions in a diffuse way? Or do both things happen in part? Experimental proofs in favor of either opinion are not easy to obtain, given the lability of the process. Thus, when in the case of inverted portraits that the subject affirms to be in correct position, he is asked to point to the forehead or hair, he indicates with his finger rather automatically toward the upper part (the forehead or hair being in reality on the lower part), however, pressing him to indicate more definitely the details of the asked object, he appears insecure and finally changes direction towards the lower part. Therefore, it is doubtful that a re-inversion be established. It seems more likely that the recognition of the figure per se is so independent of its position that its orientation is left practically excluded, and the figure, by being perceived unaltered, is considered to be identical either in the correct position or upside down, a wrong consideration that unconsciously is added to the perception. The recognition of the figure is so independent of its orientation, that the recognition is the dominant sensation for the patient, and if there is no alteration of the appearance by modification of the orientation, there is no reason to believe that the figure is in an abnormal position. This is the simplest explanation which is also in agreement with the nature of the general disorder in our patient. In general, one should not rely on the explanations or viewpoints of brain-injured patients, but only taken them as data to be verified. Admitting that the patient truly sees the inverted figure in the correct position, we would have to assume a re-inversion of the image in the imagination, which would correspond to a structural process similar to the one the Gestaltists call law of good form. That is, ultimately, the figures would really be perceived in the right position because it is the most appropriate, and this easy re-inversion would be favored by instability in the perception and lack of firmness in their position and orientation. However, this would entail a strong activity of the orthogonal property described above, which fails precisely in this patient. On the other hand, the instability in the perception of orientation could also be considered to be the cause for excluding the influence of orientation on the figure. In short, it must be admitted that the deficit in the organization of orientation is the cause of its lack of influence on the aspect of figures, contrary to what happens in the normal subject. For the patient, the aspect of the figure is always the same, and is right in any position.

However, one might then ask why the behavior with respect to objects or figures that are indifferent from the point of view of orientation, is so different. This is probably because they are not subjected to a change of aspect according their orientation, and above all because they are very simple objects or figures whose positions or orientations are much easier to determine. Thus, in the case of an arrow, it is not difficult to know whether it is in a vertical position or tilted since it is only just about the position of a straight line, and the same would apply in the case of a spoon, for example. Whereas for the normal subject a difference can be established between forms with “orthogonal” and indifferent signification, for case M, all forms behave as indifferent. Nevertheless, he can recognize their orientation in some of them, and in others the orientation seems to him the same.

This is due to the fact that in the most complicated figures (portraits, pictures, letters, numbers, etc.) the interest of the figure per se predominates over anything else and the perception of form is realized independently of the spatial background, contrary to very simple figures in which the figure-background relationship is easier to establish. Therefore, it really has to do with the diverse complexity to organize forms in the space or background that contains them. In all cases there is lability or difficulty of orientation, but in the case of extremely simple figures, their orientation can still be recognized, whereas in the case of more complex figures, there is a greater requirement for perception since, in addition to understanding them, they must be oriented with respect to the background, and the latter does not take place (lost orthogonal property), so all orientations seem approximately identical. For this reason, it is very difficult to admit that a portrait is upside down since it offers the same aspect as when it is in normal position.

This type of orientation fault may contribute to hide in part the visual image orientation disorder studied in precedent chapters, e.g. objects that should be perceived as tilted due to poor illumination and small size may not be perceived as tilted. Thus, subject M being in the theater realized that the actors were upside down or at least that they were highly tilted. This happened to him immediately after the beginning of the performance. However, the inverted scene did not feel totally strange to him, and there were moments where he failed to notice the inversion.

He has very remarkable confusions about the position of objects of daily use, for example, chairs. In inactive state he makes mistakes constantly, and no matter whether chairs are in correct position or upside down, they always seem to him that they are in a good position. Invited to sit in a chair upside down rested on the wall (whit his hands in his pockets to avoid being oriented by touch), he tries to sit resolutely without any qualms, and is very surprised when he slips. He believes that the chair might be broken, and then he does not agree to sit on another one in normal position under the excuse that it is a hoax. These mistakes disappear as soon as facilitation by muscular effort acts. But also in the inactive state, when the observation lasts a long time and he is asked to look hard, he ends up by showing doubts about the true position of a chair put upside down. He finds certain abnormalities, for example, that sticks come out too much or that there is a crossbar that does not correspond; he just gets to say that the chair is a little crooked. Similar mistakes arise when he tries to know if a table is in normal position or upside down. A table upside down with a chair next to it in normal position seem to him in a good position to sit down to eat. It seems that the set surprises him, but he is not able to understand the situation; then, after a long time, he thinks the chair is fine and the table legs a little crooked. If he comes very close to objects, he ends up recognizing their position correctly. In general, he confuses very easily the positions up and down in all these tests; instead, he perceives rather well a chair lying on its side.

The confusion about spatial orientation occurs more commonly in drawings and pictures and less in tangible objects. Thus, whereas a portrait is perceived equally in any orientation, especially vertical up and vertical down, there is less confusion with tangible objects of moderate size, and generally the subject knows their true position. A particularly difficult case is to recognize an orientation among others in a set of variously oriented objects or drawings; it usually occurs that all of them rank equally as regards

orientation. Moreover, whereas he is able, after paying close attention, to recognize the orientation of a single object isolated from the set, if the object is in the set, he may equate its orientation with that of any other object in a very different position. Differentiation for any class of function is a complex activity, hence its failure is easy. Therefore, it is understandable the great difficulty when trying to assemble a puzzle or to build a drawing that requires many lines.

All these kinds of disorders are presented to the maximum in the signs of writing (letters and numbers), where they can be easily studied.

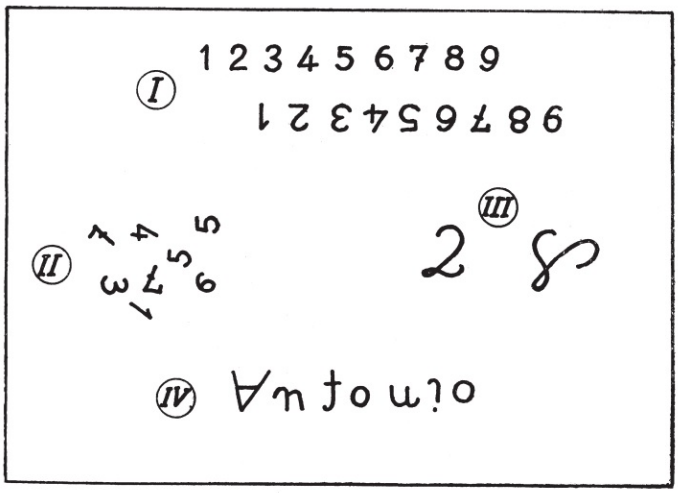


Figure 79. Numbers and letters to test the loss of orthogonal orientation in subject M, in the inactive state. I) The two rows of numbers are recognized with the same ease, and he claims they are in the same position. II) He names correctly all the numbers claiming they are all equally in normal position and in two rows. III) He has no difficulty in recognizing two `twos´ with very different aspects, whereas a normal subject can easily take the altered `2´ for an `8´. IV) He reads his name perfectly without noticing any anomaly. By means of facilitation by muscular effort, he becomes aware of the inversions and other orientations.

Figure 79 shows some of these tests. The M case recognizes and names the series of natural numbers in any orientation, both in normal and upside down position, without noticing any difference and, of course, he thinks they are fine in both cases (Fig. 79 I). Numbers variously oriented and placed disorderly, are all of them read by him with the same ease and he believes they are all equally oriented and in two rows (Fig. 79 II). A very demonstrative case of the loss of influence of spatial orientation on the aspect of the figure is the test we carried out with the number `2´. This number drawn in a very anomalous position, as indicated in Fig. 79 III, is not recognized by a normal subject or he thinks it might be an `8´ in any case. However, our patient takes it for a `2´ with the same ease as when it is placed correctly. Besides, he believes it is in normal position, as the `2´ written correctly. In this example, whereas the normal subject tends to see an `8´ due to its orthogonal property which appears then in favor of that number, the M case keeps on seeing a `2´ because the “orthogonal” signification does not intervene, and there is no confusion with an `8´. Under facilitation by muscular effort, the different position is well distinguished; the normal `2´ is more easily recognized whereas the altered `2´

appears strange at the beginning and at times it is taken for an `8´, as it happens to a healthy subject.

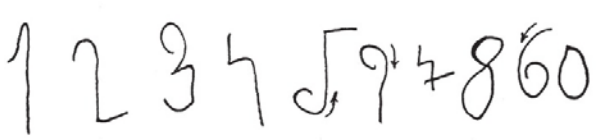


Figure 80. Spontaneous writing by patient M of a series of numbers. He reverses the `6´ and the `9´, which he confounds with each other. In addition, perseveration of a number in another one is observed (he begins the `3´ as a `2´, the `5´ as a `6´, the `7´ as a `4´). The difficulty for numbers 6 and 9 is quite persistent.

Also noteworthy is the way in which subject M writes the series of natural numbers (Fig. 80), the maximum difficulty being in numbers `6´ and `9´, which he draws almost always mutually changed. In addition, some perseveration of the shape is observed when passing from one number to another number; the number `2´ reminds the number `1´, the `3´ is written as a `2´ initially, the `5´ is written imperfectly and beginning from the bottom; the `6´ is made like a `9´; the `7´ reminds the `4´, and finally the number `9´ has been mistaken for `6´. This last mistake occurs constantly. When he is asked to write `96´, he writes `69´ most frequently, and when is asked separately to write `6´ and `9´, very rarely he can do it correctly and writes them inverted and also reflected. Furthermore, anyone of these two numbers is taken for a `6´ or for a `9´ indistinctly, the same number thus offering two very different meanings. This situation is similar to that of the normal subject with regard to ambiguous images (Vexierbild in German) and drawings with interpretation either in hollow or in relief. By means of facilitation by muscular effort, he easily realizes the confusion between `6´ and `9´ and then writes them correctly.

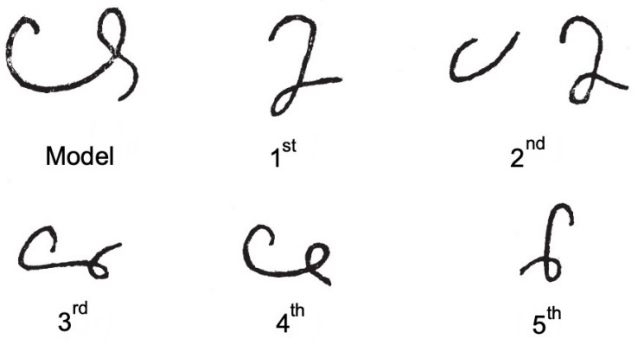


Figure 81. M case. Several stages in the copy of the abnormally oriented `2´ which subject M considers in correct position.1st attempt: he writes automatically a normal `2´. 2nd attempt: asked to concentrate on the model, he tries to copy it but he starts again and writes again a normal `2´. 3rd attempt: the copy resembles more the model. 4th attempt: finally, the copy is almost equal. 5th attempt: if told to write the number without paying attention to the model, he draws a mirrored `2´.

When he is asked to copy a number that is inverted and that he admits as being in correct position, the copy may be a true copy, i.e., inverted, although he still maintains it is in the normal position. If he is then asked to write the same number by heart, without looking at the inverted model, he does not draw it completely normal, the result being intermediate between the previous inverted copy and its normal position. These remarkable peculiarities can be observed in the various stages displayed in Fig. 81. He immediately recognizes the `2´ of the very abnormally oriented model, and when he is asked to copy it, he writes a `2´ in normal position, which he does automatically, without worrying about the original model; he finds them both identical. Urging him to copy the model in a second attempt, he begins the copy faithfully, but ends up writing the `2´ automatically in normal position. A third time he tries the copy and does it rather well, and a fourth time almost completely well. If he is now asked to write the number without looking at the model, he does it but in a reflected way. In every case, the numbers seem to him to be in the same position even though the drawing has been different.

These tests show both the antagonism between automatic writing and model imitation, and the lack of influence of writing movements on the perception of the orientation of the writing. The exact copy can be reached after many attempts and very slowly, but the change of movements is not sufficient to establish a different understanding in spite of the modifications in the motor attitude during the attempts, as is shown by writing finally the number without looking at the model. The lability of spatial orientation perception is therefore extensive to writing movements, this being also the reason for the mutual confusion between `6´ and `9´ when drawing them.

Concerning the particular conditions of stimulation and functional level which determine the failure in the spatial orientation of figures, we must note that there are several types of vision similar to those already discussed in other visual functions. Generally, in good illumination and adequate size of figures, subject M in binocular vision and inactive state recognizes what it is all about, but he fails in its spatial orientation which he always accepts as normal. However, under facilitation by muscular effort, the interpretation is perfect. However, even binocularly and with muscular effort, when the illumination or the size of the figures is considerably reduced, the same failure as in the inactive state appears. Here too, especial visual facilitations are possible. Thus, subject M binocularly, with muscular effort, good illumination and close enough, recognizes the different meanings of the numbers and letters shown in Fig. 79, but does not perceive the orientation defects or only succeeds with some number. However, as soon as intense light is directed toward his eyes, without changing the illumination of the figure, he perceives correctly the diverse orientation of the numbers and letters. This is then the result of binocularity, muscular effort, light over the eyes and good illumination of the figures. Such combination of facilitations may be needed in the case of small figures, or in tests of differentiation which are always most difficult. It can also happen that with muscular effort but looking at the figure with only one eye, the patient does not know its orientation, and by intense illumination over the other eye, which although open does not look at the figure, a normal visual function is obtained. Thus, a series of combinations can be carried out. In short, the functional level depends on both facilitation and intensity of the stimulus (size and illumination of the figure).

This series of tests allows us to establish different stages in the development of the normal function. There are mainly the following stages, from the simplest to the most complex: 1st, all orientations are indifferent; 2nd, only the opposite orientations in the vertical, up and upside down, are confused with each other; 3rd, orientation is correctly perceived. All these stages depend on the stimulation conditions and the excitability level by the action of different types of facilitation. At all stages, the interpretation of the figure can be as good as desired, perceiving details in a portrait about sex, age, etc., but this does not prevent the failure in recognizing its orientation.

As for the subject T, in his usual state at present, he does not show any disorder of this type, not even drastically reducing the illumination of the figures; or in any case, the disorder is unimportant and is quickly corrected. Nevertheless, we had the opportunity to observe a very evident alteration in his perception of orientation in early 1939, when trying to verify whether he presented inverted vision, previously discovered in patient M. Although he did not present inverted vision but only a significant tilt whose magnitude was not accurately determined at that time, it was quite clear the loss of orthogonal orientation, since he could read the newspaper fluently and with the same ease in the normal position and upside down, although in both cases somewhat slowly because of the cerebral deficit. In both positions he found the newspaper to be in the normal position, without noticing any change. Later, we learned that this brain-injured man, shortly after having suffered the brain lesion and long before being referred to us for observation, was often seen reading the newspaper upside down, without realizing it, and it seems that he became annoyed when, for this reason, he suffered the mocking of his fellows, mocking he could not understand at all since the change of position of the newspaper was indifferent to him. This singular disorder must have disappeared in patient T around 1940, and when he was re-examined in 1942, there were no signs of it. In the tests on ethyl action described previously, this alteration was also not found, or in any case, to a negligible degree. However, when he was observed in 1944 by chance, two days after a severe epileptic seizure which had reduced considerably his cerebral activity, he did show certain alterations in visual orientation close to being pathological. For example, he was able to read inverted writing without realizing it, and he used to believe that correctly oriented writing was badly oriented, but finally he was able to realize the real spatial orientations despite he easily confused some of them. Regarding his functional ability, he appeared to be in the intermediate state indicated above in which opposed orientations (normal and upside down) are easily confused with each other.

16.1.3 Structure of orthogonal orientation. Spatial level

Regarding the structure of the function `orientation of figures´, it is important to call attention to certain ontogenic and phylogenic peculiarities, i.e. its development in children and primitive humans, who show a behavior very similar to that of our two patients. Preyer (1882, 1890) discovered his 5-year-old son drawing most of the numbers either reflected or inverted. This behavior lasted a long time with the numbers 1 and 4. Bühler (1918) relates in his work that it is a very curious fact, which has caught the attention of many people, that many children look at their books of drawings with the same interest

whether the books are in a normal position or upside down; and when this fact is investigated it is found that they distinguish and name representations of men, women, animals, trees, houses, ships, etc, with the same ease in the normal position and also rotated at an angle of 90° to 180° from the natural position. This is consistent with the fact that when they start drawing, they often confuse the orientation in space, representing things upside down; for example, they draw men and animals upside down or a car with the wheels upward, and this is also shown in their first attempts at writing, where another type of disorientation is even more frequent, which is the reproduction of writing as seen in a mirror changing the right side for left side. All this constitutes a complex set of facts from which it follows that the concept of spatial form can be represented, up to a certain point, independently of orientation, and children often ignore orientation even when they already know how to draw forms. A more detailed theoretical study of this issue could significantly contribute to the general problem of perception of figures. Also for Stern (1909), form is for a child much more independent of its position than for us adults. Children look at drawings and pictures in any position, they copy the letters from a model in all possible orientations without any difficulty and it is not an effort for them to read a text reflected in a mirror, which they do as well as those who are trained to do it. A finding of Oetjen (1915) is very interesting in this regard; he observed that a rotation of 90º of reading material represented a much less difficulty for children aged nine to thirteen and a half than for adults. The influence of writing orientation on the time spent on writing recognition by adults is shown in the research of Prandtl (1927), according to which the positions that offer most difficulty are the two inverted oblique positions (↙ ↘), and the next in difficulty is the upside down (↓), whereas the slightly tilted positions (↖ ↗) allow reading the text in an almost normal time. Such differences are not present in children, neither in primitive people, since in both, recognition is independent of orientation, as is also the case in our two brain-injured patients. Concerning primitive people, there is an observation of Peschüel (1877) on the indigenous population of Loango (Bantu Negroes) in whom he verified that they saw and understood images representing familiar things equally well in both normal and upside down positions. Likewise, the few who knew how to read a printed text, they did it fluently either in normal position or upside down.

Therefore, the disorder of our two brain-injured patients presents a remarkable parallelism with the behavior of children and primitive people, since in all these cases the meaning of a figure is independent of its spatial orientation. In these cases, there is a *spatial level* (according to the Gestaltists) scarcely differentiated because the corresponding relationship between the figure and the spatial background that contains it is not established; thus, there is no tendency to establish the *constancy of the figure* corresponding to its normal orientation.

In the brain-injured subjects, the destruction of the visual orientation mechanism in the brain must also be taken into account, which already implies an extremely severe alteration of spatial organization. We know that even when a normal visual image orientation is obtained by appropriate stimulation, there is certain instability revealed by the influence of secondary factors (as in the slot experiment and others). This means that even in very simple shapes, we cannot count on a totally firm orientation, although in

general, for elementary shapes (e.g. stripes) the orientation is generally well perceived. However, in the case of more complex figures, there would be a *functional overload* that would prevent the development of the entire structural plan, the figure being perceived as segregated from its spatial orientation attributes. In this case, any type of orientation does not change at all the aspect of the figure, which conventionally can take on different meanings (case of `6´ and `9´). Therefore, it can also be said that there is a dissolution of the spatial orientation schema because, as was seen when studying the schema of forms, there is both an altered or unstable vision and a constructional deficit. The former because the abnormal orientation does not modify the aspect of the figure, hence visual perception is altered compared to a normal subject; the latter because all orientations are perceived as similar due to a failure in the spatial schema, that is, in the construction of references. There is thus a mutual influence between the two factors, and it is impossible to establish a clear separation between them. In short, the *spatial level* (figure-background) is not established, and everything is the same as regards orientation. The sensory field is too deficient to present then the differentiated function `schema´, which depends entirely, like other functions, on the type of stimulation and above all on the established physiological level. By means of facilitation, such level improves and at the same time increases the coherency of perception, i.e. a more differentiated sensory organization is produced.

16.2 Alteration of the allocentric spatial orientation

Finally, we shall briefly discuss a spatial orientation disorder with general features very similar to those of the disorder described above. It lies basically in the fact that the spatial references are not established in an absolute and constant way according to the space coordinates, i.e. according to an *allocentric orientation*, but rather an *egocentric orientation* is generated in which the position of the subject´s body decides on the orientation at every moment. This disorder is very pronounced in the M case in inactive state, and is notably corrected by facilitation in the same way as in the loss of orthogonal orientation of figures. This case M is one of the few cases in brain pathology in which *visual agnosia of spatial orientation* has been observed. A certain similarity is found with the patient studied by Siekmann (1932).

The difficulty for allocentric orientation derives both from instability of visual image orientation and from the intrinsic alteration of spatial schema by constructional deficit. Concerning the former, we have already indicated in previous pages that a tilt of the head causes the upright test arrow to be observed tilted, even when the luminous stimulation is intense enough to exclude asynchrony in visual image orientation. In the normal subject, such a tilt only occurs in the case of a luminous line observed in the dark, which constitutes the so-called *Aubert phenomenon* (Aubert 1865). According to Müller (1917), the following three systems cooperate in the mechanism of spatial location:1, the gaze system (with the three coordinate axes of the cyclops-like eye); 2, the head system and 3, the system originated by the trunk position. Although the three systems generally coincide, they may be different in special situations, for example in the *Aubert phenomenon*. In the wounded patients studied here, this mechanism is so labile and

unstable that the dissociation occurs at all times. The above-mentioned systems neither inform nor correct each other, and the compensations that take place in the normal subject fail. In this way, spatial orientation depends on the orientation of the body of the subject, remains adhered to it, and the egocentric reference controls the situation.

This new type of orientation is evidenced in several tests. For example, when subject M moves while sitting on a swivel chair, what seems to him to be turning or moving is not his own body but the visual scene, thus appearing a "movement induced" by a change in the spatial references. This change is due to the fact that the turn of the own body is not felt because of a deficit in the nervous centers, and what is felt then in movement is the outside, i.e. the visual scene. The coordination or mutual adaptation of the diverse sensations involved in orientation can be so weak that even when walking, the subject has the sensation that the ground is moving. All these anomalies are excluded by means of facilitation by muscular effort, or at least they are so minimal that they can be disregarded, appearing only in special tests. The information provided by the haptic domain is very weak and diffuse because the tactile mechanisms (pressures, joint movements, etc.) are as altered as the visual function, and only by means of facilitation is it possible to have a more efficient haptic activity. Therefore, the orientation is not fixed as it is in the normal subject, but is determined in a circumstantial way. This can also be shown in the following experiment. Subject M with his eyes closed is fixed to an operating table that can be rotated, and he is placed vertically with his head down. In this position he is asked to look at a vertical arrow pointing upwards and to indicate its direction (the vision being very clear and distinct); he then responds that the arrow is vertical pointing downwards. That is, he does not localize the object in relation to the absolute space, which is normally independent of the body position, but he localizes it egocentrically, i.e., in relation to his own body. When he is asked to make a strong muscular contraction, his perception changes and he answers: "No, the arrow is upward, and I am downward". Facilitation, by improving brain activity, modifies the spatial reference system and replaces the pathological egocentric orientation with the normal allocentric one. In this experiment, it is understood that the abnormal body position is not detected by the subject in the inactive state but is detected under facilitation by muscular effort, which causes the location of the arrow to change. It is clear that here the change is not due to an asynchrony in the perception of visual image orientation (since the subject always sees the arrow very clearly and distinctly), but to a change in the reference.

These alterations in spatial orientation involve, as has been seen clearly, a severe tactile deficit which we will deal with further on in the appropriate place. This deficit, together with the phenomenon of asynchrony in visual image orientation, can give rise to very singular disorientations of the own body, as occurs in subject M in the inactive state. For example, taking a finger of the patient and moving it away from him so that with weak illumination the movement of the finger is perceived with blurred vision, it turns out that when his own finger passes half a meter from his eyes from left to right, he sees it in the opposite direction, and when the finger has reached the right side and he is asked to take it with the other hand, he tries to catch it on the opposite side, that is, on the left. Since the passive joint movement of his own arm is not perceived by him in the inactive state, he only detects the finger visually, and in order to grab it he must necessarily be

guided by the perceived movement of the finger which is in the reverse direction, giving rise to a severe disorientation of his own body. These experiments can be made more complicated in several ways, showing effects that constitute a true *experimental autotopagnosia*. We shall deal with this later, in the part related to haptic functions.

Apart from the alteration caused by the lack of correlation between the various factors involved in spatial orientation, the subject has difficulty in appreciating the relative orientation of several objects. If there is only a single object, the subject knows whether it is to the right or left of his body, but if he sees two or three objects at the same time and their relative positions change, he does not notice any difference, at least at first, and when he is in an inactive state. This difficulty increases when dealing with drawings. Thus, he believes with respect to Fig. 79 (II) that all the numbers are simply in two rows, being unable to appreciate the diversity of their positions and orientations. He can also get very disoriented in special situations of ordinary life, for example on the top of a staircase. Thus, when he was at the top of the steps of an operating room, near the celling, he perceived the celling as the wall, confusing most directions of the room. Nevertheless, after many verifications he can get oriented, even without facilitation.

These alterations show analogous characteristics to other disorders previously indicated in spatial schema. These are, alteration in vision and constructional deficit; the former is originated as a consequence of the alteration of references, for example in the mentioned case of “induced movement”; the latter prevents absolute references and breaks up spatial schema, evidenced for example in the difficulties to appreciate the relative positions between several objects. All positions tend to be perceived as being the same, as it occurs in the loss of orthogonal orientation of figures. In general, it can be said that allocentric orientation is replaced by egocentric one, and the subject does not move within a space of fixed orientation, but the orientational space changes according to circumstances and follows the own body of the subject.

The alteration of the spatial orientation schema in the two aspects studied, loss of orthogonal orientation and loss of allocentric orientation, reveals the complexity of the visual orientation disorder. Whereas orientation in the normal subject is firmly unified, here, in our brain-injured patients, especially in case of M, it is split, due to nervous asynchrony, into a series of stages or independent partial functions with a very diverse level of excitability, all of which can be recruited either by increasing stimulation or by synchronization by means of facilitation. Considering the most pronounced changes in structuring spatial orientation, we can distinguish the following stages: 1, inverted vision (with indistinct vision); 2, normal oriented visual image although labile (with distinct vision) and orthogonal and allocentric failure, and 3, normal stage only reached by means of facilitation from the second stage.

Dynamic reduction, by excluding the most complex functions, follows an inverse order to the one we have exposed in the three stages. It must be noted that functional disaggregation is conditioned by the degree of asynchrony; thus, subject M with distinct vision can show allocentric and orthogonal failure, whereas the T case with also distinct r vision does not present such alterations due to a significantly lower asynchrony.

Complilation of visual functions

A brief compilation will serve to establish an overview of the multiple phenomena studied in the visual function, many of which are new, and the way they are addressed is completely new.

The alteration of the visual system is only a part of the sensory disorder in the central syndrome; disturbances of the same nature and intensity will be studied in other sensory systems. Sensory structures depend on the degree of nervous synchronization. A brain injury, causing an excitability deficit that affects functional elements differently, leads to an asynchrony between them. New functions then appear without any precedent in the normal subject (chromatopsia and dyschromatopsia, flat colors, inverted vision, metamorphopsia or change in shape perception, egocentric spatial orientation, reading upside down as easy as normal, etc.). However, an inextricable chaos does not occur, but a more reduced and simple organization where the general physiological laws remain valid, although at a different scale.

1) Regarding *general excitability*, there is a considerable increase in rheobase and chronaxie, in addition to the important anomaly of permeability to nervous summations such as facilitation and iteration. Luminous sensation presents long latency and also high persistence. Light adaptation is very slow and to a lesser degree than in the normal subject; there is therefore a certain hemeralopia of central origin. It is very important to take into account that visual sensation depends on the three following factors: stimulus, receptor and central state (which depends on the deficit created by the lesion and on the compensation provided by facilitation).

2) In *color vision,* there is a general alteration, but with very great predominance in the yellow-blue pair which is perceived very altered. There is a great increase in the photochromic interval, and in yellow, blue and white, a special decomposition that gives rise to a photo-heterochromatic interval. The significant asynchrony and different excitability of the different colors prevent the formation of white, and chromatopsia appears in its place. It should also be noted the inversion of color isopters, and the alteration of chromatic induction phenomena (increased edge contrast and abolition of negative afterimages). The color disorder corresponds to tritanopia (according to the nomenclature of the trichromatic theory). According to the physiological properties of colors, they can be classified into simple or primary (red and green) and composite (yellow, blue-violet, white), the last ones being differentiated from the first ones.

3) Regarding *visual forms*, we can mention phenomena such as concentric reduction of the visual field (prototype of dynamic reduction), irradiated localization which causes the pathological vision of flat colors and severely alters visual acuity, severe deficit in visual perception of motion, etc. Motion perception is broken down into different phases (static, inverted motion, seeming acceleration, etc.). Perception of figures and objects is very diffuse and unstable, there is a tendency to metamorphopsia, which creates a more stable and simpler figure. Understanding or recognition of objects is indirect and insecure, as well as slow.

4) With regard to *visual image orientation*, there is the very singular phenomenon of inverted or diversely tilted vision, as a result of asynchrony in the visual-haptic complex. Distinct vision (with intense stimulus) results in correct orientation, whereas blurry vision (with weak stimulus) causes inverted vision. Perceived orientation as a function of stimulus intensity follows the law of Fechner (1860), which can also be applied to other visual asynchronies. In relation to the factors involved in sensation, important phenomena appear such as re-inversion by means of facilitation, inverted vision in peripheral vision and also due to dazzling, types of vision with different maximum image tilt, etc.

5) Concerning *visual schema* (understanding of simplified figures and also of complex visual structures), there is a schema dissolution, both by an alteration of vision resulting from an unstable perception, and by a constructional deficit that disintegrates structures; drawings are seen differently and there is no capability to understand ensembles of them. Because of the alteration of spatial schema, there is a loss of orthogonal orientation (recognition of figures independently of their orientation, as reading an upside down text without being aware of it) as well as a loss of the allocentric orientation, which is replaced with the egocentric orientation, due to a change of spatial references.

Table 17. Visual functions of subject M in the inactive state.

1. General excitability	**2. Colors**	**3. Forms**	**4. Image orientation**	**5. Schema**
Rheobase and chronaxie 10 × greater than normal ------ 71% summation degree in iteration ------ Great permeability to facilitation, saving 1/3 of rheobase voltage ------ Long latency and persistence of sensation	In medium light: yellow-blue blindness (dyschromat.) ------ Chromatopsia ------ Inversion of color isopters. ------ Very increased edge contrast, etc.	Visual field up to 6º (OD), 4º (OS) ------ Acuity monoc. 1/25 binoc. 1/10 ------ Strong chromatic irradiation ------ Loss of motion perception except with very intense stimulus ------ Unstable shape perception, successive understanding	OD max. tilt 145º. In very strong light: about 5º for normal vision. ------ OS max. tilt 170º. Binocularly max. tilt 115º ----- Binocular image splitting ------ Change by: facilitation, dazzling, peripheral vision	Binocular and strong light: illusions, disaggregation and concrete behavior in the face of shapes and complex figures ------ Spatial orientation: loss of orthogonal and allocentric functions (only egocentric orientation).

Table 18. Visual functions according to three types of physiological level.

	M inactive	**M under facilitation by muscular effort**	**T inactive**
Excitability [10]	OD: rheobase 14.2 V, chronaxic cap. 3.5 µF. OS: more disturbed.	OD: rheobase 9.5 V, chronaxic cap. 2.7 µF.	OD: rheobase 7.8 V, chronaxic cap. 1.4 µF.
Colors	*In medium light*: yellow-blue blindness, etc., intense chromatopsia, pronounced inversion of color isopters, intense alteration of chromatic induction phenomena.	*In medium light*: practically normal color vision. *In very low light*: phenomena of the inactive state.	*In very low light*: tritanomaly (weakness to blue), traces of fleeting chromatopsia, partial inversion of color isopters.
Forms	Visual field up to 6º Acuity: monoc. 1/25, binoc. 1/10. Strong chromatic irradiation *in medium light.* Severe loss of motion perception. Unstable and diffuse shape perception. Very slow and successive perception.	Visual field up to 40º Acuity: monoc. 1/8, binoc. 1/6 -1/4. Irradiation only from red *in medium light.* Motion perception much better than in the inactive state. Better and faster shape perception than in the inactive state.	Visual field up to ~ 50º Acuity: monoc. 1/3 - 1/2, binoc. 2/3. Weak irradiation only from red. Slightly altered motion perception. Somewhat unstable shapes, and somewhat slow perception *in very low light.*
Orientation	OD max. deviation 145º OS max. deviation 170º Binocularly max. deviation 115º *In very strong light*: deviation about 5º.	OD: from max. deviation in inactive state and same stimulus, a strong re-inversion is obtained, from 145º to 30º and even 20º. OD max. deviation 97º, Binocularly max. deviation 27º.	OD max. deviation 25º OS max. deviation 16º Binocularly max. deviation 10º or less.
Schema	*Binocularly and in strong light*: illusions, disaggregation, concrete behavior, orthogonal failure, egocentric orientation.	*Binocularly and in strong light*: fairly well in general, but serious defects in complex tests, orthogonal and allocentric orientation. *In low light*: as in the inactive state.	Mistakes only in complex tests, although there are traces of weakness in all of them. Normal orthogonal orientation even in very low light.

[10] More data on excitability of the three types of physiological levels can be found in Table 6.

The dynamic unity governing brain activity is what causes the lesion to alter sensory organization as a whole, although in different degrees the different visual functions due to their different asynchrony; the greater their physiological demand, the greater the alteration suffered. The pathological asynchrony can be avoided by intensifying the stimulus. This can be achieved more easily the lower the degree of asynchrony. In highly altered cases, such as M, no matter how much the intensity of the stimulus is increased, in the inactive state there are always abnormal remnants (certain green hues of chromatopsia, deviation of visual image orientation by a few degrees, irradiation from red, somewhat unstable shapes and orthogonal and allocentric failure).

Such functional unity allows, from a single pathological datum, to presume the alteration of the rest of functions. In that regard, the overall state of visual activity in subject M in the inactive state can be summarized in Table 17.

Moreover, by counting on two patients with brain lesions of unequal intensity, it is possible to study sensory organization according to different physiological levels, which means asynchrony and dynamic reduction also different. Since in addition, in each patient both eyes are somewhat dissimilar, new different functional levels can be obtained which, together with facilitation by muscular effort and binocular effect, make possible very varied combinations that lead to about fifteen or more types of vision with different functional activity. For example, in the M case, a very substantial change is obtained by switching from monocular vision in an inactive state to binocular vision and maximum muscular effort; and there is still a bigger change between binocular vision in the T case and monocular vision in M inactive. The remarkable differences between the three most frequently mentioned physiological levels in this research can be seen in Table 18.

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