1946

SIMULATION OF NERVOUS AND MENTAL DISEASE

Moses Keschner
Columbia University

Recommended Citation

SIMULATION OF NERVOUS AND MENTAL DISEASE*

Moses Keschner †

I

INTRODUCTION

S IMULATION may be defined as a wilful, deliberate and fraudulent imitation or exaggeration of illness intended to deceive the observer for the purpose of gaining a consciously desired end. Simulation of a physical or mental illness is usually resorted to: (1) by persons who have sustained an injury, the disability resulting therefrom being compensable by benefits payable under the workmen's compensation law or by damages in personal injury actions based on alleged negligence; (2) by persons who wish to obtain insurance benefits for disability in accordance with the provisions of health, accident and life insurance policies, and included in this group are persons who attribute to an accident which may never have occurred symptoms of disability which they know, or have reason to believe may arise from or be intimately connected with independent pre-existing natural disease; (3) by those who wish to evade conscription or, while in military service, to escape certain duties—simulation in the latter instance being popularly known as "gold-bricking"; (4) by persons who wish to avoid some legal or other responsibility.

Simulation refers not only to imitation or exaggeration of symptoms, but also to the time of onset of the manifestations of the alleged disability and to its causal relation to an alleged accident.

Some physicians, lawyers and insurance adjusters have a wholly unjustifiable tendency to confuse simulation with psychoneurosis.[1] They are two distinct and entirely different clinical entities. A psychoneurosis is an actual illness in which unconsciously developed symptoms of physical or mental disease offer to the patient a possibility of solving his inner conflicts and fear of loss of security. Both the reason for the symptoms and their development are not consciously understood by the patient. A psychoneurotic tells the truth or what he believes to be true;

See p. 689, infra, note. Footnotes followed by "*Ed.*" were prepared by the Editor-in-Chief of the symposium.

† MD., LL.B.; Clinical Professor of Neurology, Columbia University.

[1] A mental disorder caused primarily by some psychic conflict, because of maladjustment, and in which there can be found no demonstrable causative organic pathology, without disturbance of intellectual function, without distortion of reality and without primary mood pathology.

he honestly believes that he is afflicted with the condition to which he attributes his alleged disability. A malingerer practices deception consciously with the prospect of pecuniary gain or of deriving some other advantage. In workmen's compensation cases, it may be true that neither the malingerer nor the psychoneurotic wants to return to work, but the former is aware that he could go back to work if he chose, whereas the latter either is actually unable, because of his neurosis, to return to duty or honestly believes himself to be so.[2]

The consciousness of the deception and fraud is the determinant of the concept of malingering. There can be no fraud, in a legal sense, without "moral fraud" and, if a patient suffering from a neurosis (hysteria,[3] hypochondriasis,[4] etc.) practices deception without the prospect of gain, the deception may constitute a morbid symptom, but cannot be regarded as malingering in a legal sense. At one end of the scale, one encounters the blatantly fraudulent malingerer who is an outright liar: at the other end, is the weak-willed neurotic who, though he exaggerates his condition, does not practice conscious deception: between, one meets an individual who also practices conscious deception, but who cannot help it. All are simulators in that they exaggerate or distort their symptoms; but before a legal tribunal, the physician must be careful in differentiating these three types of simulation.

In this differentiation, it is important to recognize that there exists a condition designated by Brissaud as "sinistrosis," in which the patient, in the course of employment, or otherwise, has met with an accident following which he is imbued with the idea that every accident constitutes an injury for which the fullest indemnity must be claimed and received. These individuals are so preoccupied with their imaginary ills that they

---

[2] It was Cozens-Hardy who first laid down the principle in Eaves v. Blaenclydach Colliery Co., Ltd., [1909] 2 K.B. 73, that a workman is entitled to draw compensation benefits for a continuing disability due to a traumatic neurosis after all demonstrable physical lesions suffered from the accident have disappeared. For a collection of the British and American cases, and a discussion of the peculiar legal problems raised by traumatic neurosis, see Smith, H. W. and Solomon, "Traumatic Neuroses in Court," 30 VA. L. REV. 87 (1943).—Ed.

[3] A condition characterized by emotional instability and the presence of a great variety of symptoms, such as partial loss of memory, disturbances in sensation, motion, contractures of limbs, abnormal movements, disturbances in vision, olfaction (sense of smell) hearing, speech, and symptoms referable to the respiratory, circulatory, gastrointestinal, and genitourinary systems, all of which resemble organic disease without demonstrable objective evidences of the presence of such disease, but they are due to mental causes, such as autosuggestion, dissociation, or repressed emotions.

[4] A morbid state characterized by profound mental depression and anxiety with exaggerated or false notions with regard to the state of health and the presence of physical or mental ailments.

become subjects of an obsession which is in itself a disease. Sinistrosis must not be confused with so-called *traumatic neurasthenia;* [5] it is not directly produced by either physical injury or mental shock. Careful observation of these patients discloses that they unconsciously super-impose a psychogenic [6] elaboration of symptoms which, because of the existing low sensory threshold,[7] pivot chiefly on the sensory systems. Some of these patients improve surprisingly rapidly following an early and, to them, satisfactory disposition of the litigation. Prolongation of the litigation in these cases leads inevitably to the patient's constant brooding over his impending law suit or hearings, with their attendant examinations and legal consultations. Here and there, however, one meets a patient belonging to this group, who, even after the legal phases of his case have been disposed of, insists that he is still "suffering" and unable to resume his previous or any other occupation, despite the fact that there are no demonstrable reasons for his alleged inability to re-turn to work other than his morbid fear of engaging in the same activi-ties or others similar to those in whose performance he was injured. Such patients are said by Panton to be suffering from "ergophobia." [8]

Should patients suffering from any of the conditions described above be regarded as malingerers? Keeping in mind the concept of malingering as defined above, one is compelled to say that a patient who exaggerates or distorts his symptoms because he honestly believes that he is afflicted with them, cannot be regarded as a malingerer. Similarly,

[5] Neurasthenia following physical or mental trauma. Neurasthenia is an indefinite term used to denote a number of conditions in which there is a functional disturbance of the nervous system, combined with marked depression of the vital forces and a tendency to rapid exhaustion, due usually to prolonged or excessive expenditure of physical and/or mental energy.

[6] Originating in the mind; psychogenic disorders as opposed to organic disorders have no organic basis for them.

[7] An individual's limits of appreciating sensory stimuli; that degree of stimulus which just produces a sensation which is perceptible to the patient.

[8] An abnormal aversion to work. This often seems to be due to a deep rooted fear or dread not readily dispelled by reason. See the case of Summerskill v. Vermont Power & Mfg. Co., 91 Vt. 251, 99 A. 1017 (1917). The facts involved were as follows: P, a 23 year old graduate of McGill University in electrical engineering, decided to gain experience by working for a time as a lineman. D's employees failed to cut off the power from a line on which he was working. An electrical current leaped with a spark or flash from a wire 4 inches away, which carried 16,000 volts, and went through his body, entering at one of his hands, and going out at one of his feet. It was not possible to show how much electricity actually leaped from the high tension line, but apparently he was not so much injured as terrified. P developed mixed symptoms of neurasthenia and hysteria, in part manifested by temporary paralysis followed by in-voluntary twitching and shaking of his muscles, and by a deep rooted dread and fear of electricity which presented a serious obstacle to pursuit of his chosen profession. Verdict and judgment for P for $7,500.00 was affirmed on appeal.—*Ed.*

a patient who claims that he is suffering from organic disease when, as a matter of fact, he is only suffering from a functional or a psychogenic disorder or when he unwittingly attributes to an accident symptoms and disability which, in fact, are closely associated with the presence of an independent and even preexisting disease, should not be regarded as a malingerer. *In the absence of fraud, the imitation, distortion and exaggeration of symptoms, the undue prolongation of an illness and the unwillingness to return to work cannot be regarded as evidence of true malingering.*

II

SIMULATION IN GENERAL

It is impossible to formulate absolute criteria of simulation applicable to every case. In most cases, however, a reasonably certain conclusion can be reached after a thorough study of the clinical picture during prolonged observation, preferably in a hospital, in conjunction with a knowledge of all surrounding circumstances, such as a detailed description of the accident and the subsequent course of the clinical history, an investigation of the patient's premorbid and present personality, his environment, and his income from his occupation and from other sources. It is also advisable to obtain reliable information about previously expressed opinions by other examiners who may have suggested to the patient some of the symptoms from which he is allegedly suffering.

Simulation is much more common when litigation is a factor. In most cases under litigation, there is a tendency to exaggerate symptoms and disability. It is well known that the clinical picture of a disease following an accident in which legal liability is involved varies strikingly from that following a similar accident in which litigation plays no role. Litigation appears to be an important determinant in exaggerating alleged pains and in perpetuating disability. Due consideration should be given to the relationship between the duration of an illness after an accident and the diminished efficiency, capacity and "will" to work following it. Prolonged idleness leads to introspection[9] and rumination,[10] with consequent exaggeration of symptoms and an early blunting of the sense of responsibility which, in some instances, may produce marked changes in personality and moral deterioration, in which lying becomes a prominent feature.

Most simulators are readily detected because they grossly exag-

---

[9] The process of self-examination, especially of one's thoughts and feelings.
[10] Meditation; continuous thinking on a subject.

gerate their "manufactured" symptoms, which usually occur imme-
diately after the injury. A simulator, unless he was "well tutored,"
resents examinations and will do anything to prevent them; he resists
all efforts at cure. This is in marked contrast to the neurotic who re-
quests repeated examinations and is eager to submit to any form of
treatment. During the examination, a simulator avoids looking the
examiner straight in the face; he resents being questioned and his
answers are either non-committal, hesitating or absurd.

At the first contact with a suspected simulator, all anamnestic [11]
data should be recorded in his own words as nearly as possible; these
should be re-checked at subsequent visits and verified by other, pre-
ferably disinterested, sources. Only a "well trained" simulator will be
able to reproduce such information on repeated re-examinations with-
out contradicting himself as to essential incidents. Inquiry should be
made about relevant occurrences immediately before and after the al-
leged accident. If, in the case of a head injury, the patient can recall
everything up to the time of the accident and can give a coherent ac-
count of what happened immediately after it, there is very little likeli-
hood that the injury was associated with a cerebral contusion [12] or other
serious brain damage. Significant information about the severity of the
alleged injury may be obtained by ascertaining whether, following it,
the patient was able to get up unassisted; whether he could reach his
home or a hospital without assistance; if he went to a hospital, how he
got there, how long he remained there, what was done for him and by
whom, what type of treatment he received and from whom after he left
the hospital; how soon after the accident he was able to resume work
and whether he was as efficient as before.

In questioning a suspected simulator, the physician must be patient
and persevering; his attitude must not be harsh and inquisitorial; he
must appear sympathetic and, at times, even give the impression that
he is credulous, lest he arouse the patient's suspicion that his credibility
is being doubted. It is well to have the patient re-enact the accident
and to show the position of his body and limbs before and during the
accident. A simulator, during such re-enactment, may move his body
or allegedly injured parts thereof so smoothly and freely that there
will be no doubt that the abnormal attitude or gait assumed during the
examination is voluntary and deliberate. Similar observations while the
patient is undressing and dressing, in the course of which his attention is

[11] Pertaining to a patient's history.
[12] Bruise of brain tissue; used by many neurologists as synonymous with "concus-
sion of the brain."

distracted by irrelevant conversation, may also aid in detecting a simulator.

If a suspected simulator does not cooperate in a test one day, he may cooperate in the same or in a similar test on the next day. A simulator may know enough to sway violently during the Romberg test,[13] but will "forget" to sway when his attention is distracted, while in the Romberg position, by an examination of his pupils, ocular movements, hearing, cardiac activity, blood pressure or some such other function.

A "poorly tutored" simulator claiming blindness may betray himself during the examination of the pupils or ocular fundi,[14] by looking continuously towards either corner of the eye without fixing it on the requested point; he may claim inability to keep the eyes open and complain of photophobia,[15] although the strongest light thrown into the eye produces little or no lacrimation.[16]

Collie[17] cites Mr. Morley, who reported a compensation case in which an allegedly totally blind workingman was led into the dressing room of a county court for examination. The "blind" man was requested to remove his collar and tie; after this was done and his attention was distracted, the physician knocked the workingman's collar button down on the floor and proceeded to examine his vision. In the course of the examination, the physician dropped a match stick on the carpet and asked the worker to pick it up. This, the latter claimed to be unable to do because he could not see the match. But when the examination was concluded and the patient was adjusting his collar, he bent down and picked up the collar button without the slightest difficulty.

III

Simulation of Symptoms Referable to the Nervous System

General Considerations

Simulation of symptoms referable to the nervous system is perhaps the most common form of simulation. The tendency to exaggeration of symptoms and preoccupation of complaints in post-traumatic clinical pictures[18] has already been referred to. Even patients with definite ob-

---

[13] Swaying of the body when the patient stands with the feet together and the eyes closed; it indicates incoordination of station.

[14] The interior of the eye.

[15] Abnormal sensitiveness to light.

[16] Tearing.

[17] COLLIE, MALINGERING AND FEIGNING SICKNESS 81 (1917).

[18] A short-hand expression for the medical condition of the patient following injury.

jective evidences of injury to the nervous system usually show a psycho-genic[19] coloration of the clinical picture, especially in cases of injury to the head and back. The prevailing opinion, however, seems to be that true malingering in cases of injury to these parts is relatively rare; it is much more common in the neuroses and psychoses.[20]

Given a patient who has actually sustained an injury to the nervous system, the first requisite is to ascertain whether the symptoms com-plained of are due to structural alterations, to disturbances of function of the affected parts or to subconscious psychic processes, or whether they are feigned. The not infrequent coexistence of all these features in the same patient may present a formidable diagnostic problem. In the evaluation of such a confusing clinical picture, the physician's mental attitude must be entirely objective. He must constantly bear in mind that, under our system of jurisprudence, when an individual has sus-tained an injury as a result of another's negligence or in the course of employment, he is entitled to damages in the former and to compensa-tion in the latter, only for pain and disability due to organic, functional or psychic disturbances resulting from the injury. No damages or com-pensation are allowable for deliberate simulation or conscious exaggera-tion of symptoms. It is, therefore, incumbent upon the physician deal-ing with a case of this type to determine which components of the clini-cal picture resulting from the injury are genuine and which are pre-tended.

IV

SIMULATION OF SYMPTOMS REFERABLE TO DISTURBANCES OF MOTOR OR MUSCLE POWER

The feigning of motor weakness is very frequent in simulators. Complete hemiplegia[21] or paraplegia[22] is rarely simulated, but weak-ness of a limb or of a part of a limb is frequently complained of by sim-ulators.

In examining the motor power of an allegedly weak or paralyzed limb in a suspected simulator, it is generally found that he makes little or no effort to contract the muscles necessary to execute the desired movement. Reliable evidence that the patient is not exerting all his power during a forceful voluntary movement can be elicited by watch-

[19] See note 6, supra.

[20] Any disease or disorder of the mind; any disorder characterized by mental aberration.

[21] Paralysis of one half of the body.

[22] Paralysis of the lower half of the body and lower-limbs.

ing the contraction of the agonists[23] and antagonists[24] during the execution of the movement. It is a well established physiological principle that, during fine and feeble movements, the antagonists cooperate with the agonists, acting as brakes on the latter, so that the desired movement can be executed with delicacy and precision whereas, during coarse and forceful movements, the agonists alone contract and the antagonists relax, rendering the action of the former more effective. This principle is the basis of a test devised by Erben[25] to detect simulation of weakness of motor power. In this test, the motor power necessary for the execution of a voluntary movement can be ascertained by directing the patient to contract against resistance the muscles of the part tested. Thus, when the patient is requested to bend his elbow forcibly against resistance and, in so doing, contracts the triceps muscle, it may safely be assumed that he is simulating weakness of flexion of the elbow because he contracted the triceps, which extends the elbow, in order to diminish the effect of the biceps and the other muscles which flex the elbow. The motor power of other muscles can be determined in a similar manner.

Schuster[26] and Hösslin[27] have found that in simulated muscle weakness, if energetic resistance is applied to the movement of a group of allegedly weak muscles, e.g., the extensors of the knee and the resistance is suddenly withdrawn, the leg does not immediately become extended but remains flexed, because the patient has voluntarily contracted the antagonists (in this case, it would be the flexors of the knee). According to Oppenheim,[28] however, a similar phenomenon is frequently observed in hysteria[29] and, therefore, he does not regard it as unequivocal evidence of simulation. When, in addition to the alleged weakness, pain is present, the latter may give rise to an apparent reduction in motor power because, in order to overcome the pain, the patient executes the desired movements slowly and cautiously. In such cases, the simultaneous contraction of agonists and antagonists during a re-

[23] A muscle in a state of contraction with reference to its opposing muscle or *antagonist*.

[24] A muscle which acts in opposition to another (agonist), as a flexor, which bends a part, is an antagonist to an extensor, which extends it.

[25] ERBEN, DIAGNOSE DER SIMULATION NERVOSER SYMPTOME, Berlin, 3d ed., 146 (1930).

[26] SCHUSTER, DIE UNTERSUCHUNG UND BEGUTACHTUNG BEI TRAUMATISCHEN ERKRANKUNGEN DES NERVENSYSTEMS, Berlin, 62 (1899).

[27] Hösslin, "Zum Nachweise der Simulation bei hysterischen und Unfallskranken," 37 MUNCH. MED. WOCHENSCHR. 1521 (1902).

[28] OPPENHEIM, TEXT-BOOK OF NERVOUS DISEASES, trans. by Alexander Bruse, p. 1083 (1911).

[29] See note 3, supra.

quested forceful movement is no definite proof of simulation of motor weakness.

To test the power of the deltoid muscles,[30] the patient is requested to raise his arms to a right angle with the body and resist the examiner's attempt to press them down forcibly. If the patient resists this attempt, there can be little doubt that he is feigning weakness of the deltoids.

Another ruse is for the examiner to raise the patient's arms horizontally and to support them while in that position and then, without warning, suddenly remove all support. Should the arm be weak, as alleged, it will immediately fall to the side. In cases of malingering, however, the arm remains in the horizontal position for a second or two and, as the patient begins to realize the situation, he gradually and slowly drops the arm.

Another "trick" which may betray a simulator is having the allegedly paralyzed limb fully stretched out on the examining table and, while the simulator's attention is distracted by questions about other parts of his body, suddenly applying a painful stimulus[31] to the distal end[32] of the limb; the simulator, taken off guard, will quickly withdraw the limb.

When the patient contends inability to raise an allegedly weak arm above the horizontal position, he is instructed to bend completely forward and, while in this position, is requested to give the examiner the hand, ostensibly to feel the pulse; as a rule, the patient raises the arm, not realizing that the horizontal raising of the arm while he is in the bent position occurs in the same manner as the vertical raising of it while in the erect position.

Valuable information about the presence of an alleged weakness of an upper limb may be obtained by watching the patient's performance while dressing and undressing; a simulator may give himself away by raising his arms without difficulty while putting on his shirt, vest or coat whereas, during the examination, he was "unable" to raise his arm beyond the horizontal plane.

To test the grasping power of an allegedly weak hand, the suspected simulator is first requested to squeeze the examiner's hand with the "weak" hand and, then, the examiner's two hands with both hands simultaneously; involuntarily, a simulator will grasp the examiner's

---

[30] A muscle which has its origin at the outer third of the clavicle (collar bone) and scapula (shoulder blade) and is inserted on to the middle of the outer surface of the shaft of the humerus (bone of the upper arm); the muscle draws the arm forward and raises it and also adducts it and draws it backwards and upwards.

[31] Sharp pin-prick, a very hot test tube or a strong faradic current.

[32] The end farthest removed from the point of attachment or insertion.

hand more firmly with the supposedly weak hand than at first; in eval-
uating this test, it should be borne in mind that a much firmer fist can
be made with the wrist in extension than in flexion.

Another useful test is to request the patient to grasp his own hands
as firmly as possible; if he does this and holds both thumbs in abduction
(outstretched) [33] he is not utilizing all his power, because normally a
much firmer grip is obtained by opposing the thumbs to the other fing-
ers. A naïve simulator may also be detected by requesting him to grip
the examiner's hands with his own crossed; in this position, he may be-
come confused as to which hand is the "weak" one. A suspected simu-
lator may also become confused by getting him to perform these tests
with his hands behind his back.

Confirmatory evidence of simulation of a weak hand grip may be
obtained by requesting the patient to press with all his strength a dyna-
mometer [34] placed in his "good" hand; after recording the reading, an-
other dynamometer is placed in his "bad" hand and he is abruptly
commanded to press both instruments as strongly as he can; because of
the simultaneous attention he must give to both hands, a simulator will
be unable to control both of them, with the result that the reading from
the "good" hand will be much less than on the first reading. Continu-
ous constancy of the dynamometer readings is against simulation, as is
coarse trembling of the hand and forearm or of both during the tests or
an accentuation of a pre-existing trembling of these parts.

An untrained simulator usually resists every attempt by the exam-
iner to test passive movements of an allegedly affected limb; by re-
sisting the examiner's attempt to straighten out the flexed fingers, a
simulator demonstrates the presence of power in the flexors of the arm.

To determine the maximum voluntary power of flexion [35] and ex-
tension [36] of an allegedly stiff finger, the patient, preferably blind-
folded, is requested to hold out both hands and to close them both on a
given signal as quickly and as forcibly as he can; upon being given the
command unexpectedly, a simulator usually closes *all* fingers, including
the allegedly stiff one.

Valuable information about simulation may be gained by observing

[33] A drawing away of a limb or part of a limb from a position parallel to the
median axis of the body.

[34] An instrument for measuring the force of muscular contractions.

[35] The act of bending; the movement by which two parts which can form a single
straight line are made to bend upon each other so as to form a steadily diminishing
angle.

[36] Straightening out of a limb or of a part thereof as distinguished from bending
it (*flexion*).

the physiological associated movements during a volitional motor act. Thus, an energetic and rapid making of a fist is normally accompanied by the associated movement of extension of the wrist, and rapid opening of the closed fist, by the normally associated movements of slight flexion of the wrist and abduction and extension of the thumb. In the absence of these normal associated movements, basal ganglion disease [37] having been excluded, it may be concluded that the patient did not completely contract all muscles necessary to make and to open a fist. Similarly, forceful voluntary plantar flexion of the ankle joint is normally associated with "clawing" of the toes; failure of the latter, in the absence of evidences of disease of these joints, is indicative of feebleness of the voluntary innervation (contraction) and suggestive of simulation. Unusually energetic contractions of muscles during the execution of a voluntary movement is generally accompanied by a slow coarse trembling of the part tested, so that the appearance of a tremor during an apparently feeble voluntary movement would be against exaggeration or simulation.

During these tests, the patient's hands and fingers should be carefully inspected and compared on the two sides to determine whether the skin of one is cleaner and softer than that of the other; usually, when both hands are equally dirty, the patient is using both of them.

Simulation of weakness of the lower limbs is generally more difficult to detect. Reliable information is best obtained when the patient is flat on his back and the motor power of every segment of the limb is tested separately against resistance.

Observations of gait and station (the position assumed in standing) may furnish useful information about the motor power of the lower extremities. The suspected simulator should be persuaded to stand barefooted first on his heels and, later, on his toes for as long a period as possible; while on his toes, he may be induced, by requesting him to raise his "good" foot and place it on the upturned tip of the examiner's shoe, to maintain his balance on the terminal phalanges (toe bones) of the allegedly weak limb alone.

[37] An affection due to disease of the corpus striatum, namely, the caudate and lenticular nucleus and especially the globus pallidus and the connection of these structures with the optic thalamus, cerebellum and cerebral cortex. Such an affection may assume the pattern of chorea, dystonia, paralysis agitans, athetosis, etc. The cardinal clinical manifestations of a disease of the basal ganglia are generally an increase in muscle tone (rigidity) abnormal involuntary movements (tremor, shaking, athetoid movements, dystonic movements), poverty or loss of the *normal associated movements* (swinging of the arms while walking, extensor kick of the wrist on making a fist, etc.), absence of true paralysis and absence of sensory disturbances.

A simulated "lame" gait may be exposed by requesting the patient to walk on his toes when, not infrequently, he will "forget" to limp. Finding that the soles and heels of both shoes (provided they are not new) are equally worn down should arouse suspicion that a one-sided "paretic,"[38] gait or a clubfoot is simulated.

When a suspected simulator claims that one leg is weak, he should be requested to kneel on the floor with both knees and, then, to rise from this position; while he is kneeling down, the knee which is put down first and the leg used as a fulcrum[39] on rising should be noted; during these maneuvers, a simulator may inadvertently support his entire weight on the allegedly weak knee or leg.

Following an injury to the lower back, hip or lower extremity, a patient may pretend that he is suffering from so-called *sciatica* (a painful affection of the sciatic nerve). In genuine sciatica, the patient usually stands with his hip and knee flexed on the affected side and, in walking and bending, he spares the affected limb as much as possible and persists in holding the hip in flexion; when requested to sit on the floor or on a low chair, the patient will "let himself down" slowly and cautiously on the buttock of the unaffected side and will remain seated on that buttock, supporting himself with his upper limbs. A simulator, unaware of these postural peculiarities, will go through these tests in such bizarre fashion that there will be little doubt that he is feigning. Confirmatory evidence of simulation may also be obtained by the patient's ability to raise the allegedly affected limb in the so-called "straight leg raising test,"[40] while his attention is distracted during the test.

In a patient with true sciatica, there is usually observed, especially when he is in the erect posture, a scoliosis[41] of the vertebral column, with the convexity of the curve toward the painful side. The patient localizes his pain along the course of the sciatic nerve[42] and the examiner can usually elicit the tender *Valleix points* by exerting pressure at particular spots.[43] There is almost invariably present a positive Lasegue

[38] Weak.

[39] The support on which a lever rests.

[40] In the absence of paralysis the inability to completely raise the leg held in extension without pain usually indicates involvement of the sacroiliac joint if the pain is felt in the back, but if the pain is felt in the retropopliteal space (back of the knee) the involvement is usually in the hamstring muscles or in the sheath of the sciatic nerve.

[41] Abnormal lateral curvature of the spine with more or less rotary twisting of the bodies of the vertebrae.

[42] Back of the thigh, back of the knee and calf.

[43] In general, along the course of the sciatic nerve at the hip (over the posterior-superior spine of the ilium, the sciatic foramen, and midway between the tuberosity of

phenomenon.[44] In most cases of true sciatica, there are objective sensory disturbances and complaints of numbness, coldness and tingling in the distribution (course) of the sciatic nerve, and the ankle jerk (Achilles reflex) [45] is diminished or absent. In long-standing cases, muscle wasting, electrical changes [46] and vasomotor (circulatory) disturbances in the areas supplied by the sciatic nerve are usually observed. Although these manifestations are diagnostic of true sciatica, their absence or presence in a modified form cannot be regarded as unequivocal proof of simulation.

The recognition of motor weakness or paralysis due to pathologic alterations (abnormal structural changes) in the central [47] or peripheral nervous system [48] is relatively easy. The differentiation between simulated motor weakness or paralysis and that due to hysteria, however, may at times present a perplexing problem. In this differentiation, the following criteria may be of assistance: simulated paralysis cannot be maintained continuously for as long a period as hysterical paralysis; the latter is, as a rule, more profound than the former and is frequently accompanied by hysterical sensory disturbances in the affected parts. Hysterical paralysis is rarely limited to a single muscle or to muscles supplied by one nerve only; it is usually in the form of a monoplegia [49] or hemiplegia [50] or paraplegia [51] or it may affect only a hand or a foot. It usually involves parts of the body concerned in the associated movements of some special function, such as speech, chewing, breathing, etc.

the ischium and great trochanter) and at the knee [at the neck of the fibula (outer, "splint bone" of the leg) and below the external malleolus.]

[44] Pain and resistance on flexing the thigh at the hip and extending the leg at the knee; its presence usually indicates an affection of the sciatic nerve.

[45] Striking sharply the tendon of the muscles of the leg (below the ankle) causes a sudden extension of the foot.

[46] Electrical changes and electrical reactions: Normally nerves and muscles may be stimulated to contraction by both the faradic (interrupted)· and galvanic (constant) currents. The responses are quick. In pathologic conditions involving degeneration of the motor nerves to a muscle the contractions are altered both in quantity and quality. If merely a stronger current is necessary to cause a contraction the change is spoken of as *quantitative;* if the character of the contraction is changed and the reaction to the negative and positive poles of the galvanic current is altered the condition is spoken of as *qualitative,* and is designated as a *reaction of degeneration (RD),* which may be *partial,* or *complete,* depending upon the severity of the pathologic process.

[47] Brain and spinal cord.

[48] The nerves which extend from brain and spinal cord to muscles and organs throughout the body; these nerves have a power of regeneration whereas brain and spinal cord do not.

[49] A paralysis affecting a single limb.

[50] See note 21, supra.

[51] See note 22, supra.

Its onset is usually sudden, after an emotional trauma, pain, or hysterical seizure.[52] If a hysterical paralysis is incomplete, it can almost always be ascertained that only volitional movements are difficult or impossible, but automatic emotional reflex movements, such as gesticulation, are unimpaired.

Hysterical *astasia abasia*[53] is distinguished from the simulated form by the fact that although in the former, the patient is unable to walk or stand, he may be able to move his limbs while in the recumbent posture; in the latter, the patient claims inability to stand or to move his legs at all.

Hysterical paralysis[54] may be flaccid (lax or soft) or rigid. If rigid, it is frequently associated with contractures[55] and deformities. Resistance to passive movements is usually greater in simulated than in hysterical contractures. The pattern of a hysterical contracture of a limb is usually more persistently maintained (stereotyped) than that of a simulated contracture. The reason for this is that in most cases of hysterical contracture of a limb or of a part thereof, the contracture is the result of a continuing local defense reaction to pain, following injury to the affected limb. The contracture, therefore, is frequently associated with pain and hypersensitiveness of the contractured joints. The tenderness in these hypersensitive areas is characteristically limited to the skin and subjacent soft tissues; it almost never affects the underlying bony structures. Such phenomena are rarely observed in simulators, unless they have been "well tutored."

Hysterical paralyses and contractures may disappear suddenly after mental excitement or following psychotherapy, such as suggestion[56] or hypnosis.[57] This is not the case in simulation. The long volitional maintenance of a contracture or of an assumed deformity by a simulator necessitates such great attention, strength and effort that the "strain"

[52] A condition characterized by emotional instability and the presence of a great variety of symptoms, such as partial loss of memory, disturbances in sensation, motion, contractures of limbs, abnormal movements, disturbances in vision, olfaction (sense of smell), hearing, speech, and symptoms referable to the respiratory, circulatory, gastrointestinal, and genitourinary systems, all of which resemble organic disease without demonstrable objective evidences of the presence of such disease, but they are due to mental causes, such as autosuggestion, dissociation, or repressed emotions.

[53] Inability to stand combined with inability to walk, while sensation, motor power, and coordination, except for standing and walking, are retained.

[54] See note 52, supra.

[55] A condition of permanent contraction and rigidity especially of the muscles.

[56] A thought or idea conveyed from one individual to another.

[57] The production of sleep or hypnotism which is a mental state resembling sleep which is characterized by excessive suggestibility to the hypnotist and which is usually induced by psychological suggestion, by passes, or by having the subject focus the eyes on a bright object.

usually produces a rapid pulse and irregular respiration, with such "howling" and "groaning" while the affected limb is being manipulated, that there can be but little doubt that the entire difficulty is at the conscious level.

In recent years, cinematographic films taken while the patient was unaware that he was being observed have been of great aid in giving a convincing visual demonstration that an alleged paralysis, abnormal gait or posture, contracture or deformity in a simulator were assumed for the purpose of deception.[58]

In the evaluation of motor disturbances presented by suspected simulators, the relation of hysterical phenomena to voluntary processes must always be taken into consideration. There are some individuals who are able to dissociate[59] their muscular activity so that they are capable of isolated toe movements or of isolated contraction of one shoulder or one pectoral muscle or one vocal cord or one eyelid. Prolonged practising of such movements may lead to bizarre patterns of motility. There is also little doubt that the particular pattern of a contracture or of an abnormal involuntary movement in some hysterical patients is, to a great extent, determined by their capacity for dissociat-

---

[58] Motion pictures have been admitted in evidence in support of defendant's defense of malingering: Heiman v. Market St. Ry. Co., 21 Cal. App. (2d) 311, 69 P. (2d) 178 (1937); Metropolitan Life Ins. Co. v. Wright, (Miss. 1940) 199 S. 289; Snyder v. American Car & F. Co., 322 Mo. 147, 14 S.W. (2d) 603 (1929); Riboletti v. United Engineers & Contractors, 18 N.J. Misc. 219, 12 A. (2d) 251 (1940) (Workmen's Compensation case involving traumatic neurosis); Boyarsky v. G. A. Zimmerman Corp., 270 N.Y.S. 134, 240 App. Div. 361 (1934).—*Ed.*

In Denison v. Omaha & C. B. St. Ry. Co., 135 Neb. 307, 280 N.W. 905 (1938), motion pictures of a personal injury plaintiff taken without his knowledge were shown to the appellate court; the tribunal decided in view of the work plaintiff was obviously able to perform, a verdict of $5,000 was excessive, and ordered a remittitur of $1,000.

In McGoorty v. Benhart, 305 Ill. App. 458, 27 N.E. (2d) 289 (1940), plaintiff had contended that personal injuries inflicted by defendant had made him incapable of performing any kind of exercise without wearing a brace. Defendant introduced in evidence motion pictures showing plaintiff in action at a bathing beach subsequent to the accident and in one scene he was snapped lifting his girl comrade almost out of the water. Plaintiff offered to prove that the girl shown with him in the picture was either an employee of defendant or some one else interested in the suit and that it was at her insistence that he engaged in the aquatic sports portrayed. The appellate court held that it was immaterial what part the girl took in persuading plaintiff to do what the pictures revealed, since the pictures spoke for themselves and showed the plaintiff could do the very things he protested at the trial that he could not do.

As to conditions which should be established by testimony to make such pictures most dependable, see 27 ILL. L. REV. 424 (1932). See, also, 3 WIGMORE, EVIDENCE, 3d. ed., § 792 et seq. (1940).

And see, particularly, in the present Symposium series the following study: Scott, "Medicolegal Photography," ROCKY MT. L. REV., April, 1946.—*Ed.*

[59] To separate.

ing muscular activity. This capacity may be acquired by practising such isolated muscle movements of a part of the body in order to avoid pain from a pathological (abnormal) condition in that part or in structures adjacent to it. In such individuals, although the particular movement or posture was acquired by practising, which was begun volitionally as a defense reaction to pain, the abnormal movement or posture cannot be regarded as evidence of simulation unless there is also present the motivating element of deceit, which is the crux of simulation.

In the evaluation of these cases, therefore, the mere pattern of a movement, posture, or sensory disorder will not be of much aid without giving due consideration to such other significant factors as the mode of onset, duration and severity of the disorder and its response to rational therapy. Hysterical disorders following injury usually come on suddenly, are long in duration and are generally more profound than simulated disorders. The sudden and dramatic recession of the symptoms following suggestion, hypnosis and other forms of psychotherapy is almost never observed in simulation, at least not until the simulator has realized that his feigning has been or is about to be discovered or that it has served its purpose. The most difficult cases to evaluate are those in which an hysterical patient is also malingering.

V

SIMULATION OF EPILEPSY

Psychic epilepsy, variants of epilepsy (Wilson) and other epileptic equivalents,[60] attacks of petit mal[61] and Jacksonian seizures[62] are less frequently simulated than grand mal.[63]

[60] Disturbances of consciousness without convulsions, characterized by periods of stupor, confusion, excitement, ambulatory automatism and dreamy, clouded and twilight states. This designation is sometimes used synonymously with the designation "*psychic variants*," and "*epileptic psychic equivalents*."

[61] In this form of seizure there is a short loss of consciousness without convulsive movements except for some minor movements such as blinking of the eyelids, rotation of the eyeballs, movements of the facial muscles, or of the hands or feet. Petit mal seizures are more likely to occur in the minutes or hours after rising; they may be precipitated by emotion, excitement or in females by menstruation and are much less frequent during physical or mental activity. Girls are more often affected than boys. Mentality is not impaired even by scores of thousands of seizures. Petit mal seizures may last from five to thirty seconds. Lennox speaks of them as a form of *pyknoepilepsy* or dart and dome dysrhythmia, as demonstrated by the electroencephalogram.

[62] A form of epilepsy in which there occurs a convulsion, tonic or clonic in nature, beginning in a limited part of the body and usually due to some irritation of the surface of the brain opposite the side on which the spasms or twitchings occur. As a rule there is no loss of consciousness.

[63] An epileptic seizure characterized by loss of consciousness with tonic, followed by clonic convulsions. This is the most common form of so-called "*essential*" or

There is no manifestation of epilepsy which cannot be feigned, and a carefully rehearsed story may deceive the most alert physician. The demonstration of feigned epilepsy may depend upon finding: (1) contradictions in the patient's history; (2) disagreements between his story and a separately obtained story from a relative or friend; (3) refusal to submit to treatment and failure to present reliable evidence of previous treatment for epilepsy; (4) undue elaboration of the history, especially of a story of familial epilepsy or of deaths from epilepsy among siblings [64] and (5) a history of more or less continuous employment in a gainful occupation.

A simulated attack of grand mal is not preceded by an aura.[65] The simulator usually chooses the time and place for the attack, making certain that spectators are witnessing it and, when he falls to the floor, he is careful to avoid injury. In genuine epilepsy, the patient cries out loudly at the onset of the seizures; in simulated epilepsy, he cries or moans continuously throughout the fit. In true epilepsy, the patient holds his breath at first and becomes cyanotic [66] and rigid; after this, the convulsive movements set in, slight at first, rapidly increasing in severity and suddenly declining: in feigned epilepsy, there is generally no cyanosis nor asphyxia [67] unless the patient voluntarily holds his breath, and the severity of the convulsive movements does not follow the characteristic order mentioned above. In simulated epilepsy, there is no disturbance of consciousness, no pupillary changes,[68] such as dilatation and absent reaction to light, no spasm of the muscles of chewing, no changes in the reflexes and no pathological (abnormal) reflexes. In

"*idiopathic*" *epilepsy*. An attack of grand mal usually appears suddenly, but is often preceded by an *aura*. An aura may be *motor* (shivering, tremor, spasm, etc.); *sensory* (feeling of numbness, heat, cold, pain, pressure, flashes of light, blindness, abnormal tastes or odors, etc.); *visceral* (pain or other abnormal sensations referred to the pit of the stomach, a sensation of choking, nausea, cardiac palpitation, shortness of breath, etc.); *vasomotor and secretory* (redness, pallor, excessive salivation, etc.); *psychic* (dizziness, fear, weakness, mental confusion, dreamy states, etc.). Sometimes an aura may not be followed by a seizure; it is not always present before a seizure and tends to disappear with the progress of the disease.

[64] One of two or more offspring of the same parents.

[65] Epileptic aurae may be *motor*, such as shivering, trembling, twitching, etc., or *sensory*, such as a feeling of numbness, heat, cold, pain, pressure, flashes of light, blindness, abnormal tastes or odors, dizziness, etc.; or *visceral*, such as pain or other peculiar sensations in the pit of the stomach, sensation of suffocation, nausea, cardiac palpitation; or *vasomotor* and *secretory*, such as redness, pallor, excessive flow of saliva; or *psychic*—fear, dreamy sensations, etc.

[66] Blue or livid skin or mucous membrane due to insufficient oxygenation of the blood.

[67] State of suffocation.

[68] Changes such as alterations in size and contour and in their reaction to light.

a true epileptic attack, on the other hand, the pupils are wide and do not contract to a light stimulus, and all reflexes, including the corneal[69] are abolished, and there is present a positive Babinski sign.[70] Deep pressure over the supraorbital nerves[71] usually causes an inexperienced simulator to wince and to make facial grimaces, indicating that consciousness and perception of painful stimuli are retained; nor is there present rapidity of the pulse unless the patient volitionally increases the rapidity of his respirations. During a true epileptic fit, the patient generally holds his fingers tightly flexed over the thumbs which are embedded in the palms, whereas a simulator usually holds the thumbs flexed over the fingers. Tonic contractions[72] during a fit can be simulated much more easily than clonic,[73] so that, in most cases of pretended epilepsy, the former predominate and the contractions themselves are more irregular and less intense. Although in most cases of grand mal epilepsy,[74] the patients froth from the mouth and bite their tongues, these two manifestations cannot be regarded as absolutely indicative of genuine epilepsy; I have seen malingerers bite their tongues or the inside of the cheeks and simultaneously produce suction within the mouth resulting in blood-stained frothing from that cavity. In contrast to genuine epilepsy, neither urinary nor fecal incontinence,[75] nor seminal emissions are ever seen in simulated epilepsy. Hemorrhages on the face, neck, upper part of the chest, conjunctivae[76] and other mucous membranes, and bruises, fractures and dislocations are not observed following a feigned attack of grand mal. The transient paralyses and diminished or absent reflexes following a genuine epileptic seizure are conspicuous by their absence in simulated epilepsy. This is also true for the postepileptic abnormal mental states. Except in cases of status epilepticus,[77] a genuine epileptic seizure rarely exceeds a few minutes— at most ten or fifteen—while a simulated "fit" may last indefinitely.

[69] Blinking and closure of the eye on touching gently the cornea with a wisp of cotton.

[70] The presence of a Babinski sign indicates irritation of the pyramidal tracts in the brain and spinal cord: the pyramidal tracts originate in the motor centers of the brain and transmit impulses for voluntary movements; the sign is said to be present when there occurs extension of the big toe, flexion and fanning of the little toes on scratching lightly the sole of the foot.

[71] The nerve situated above the socket of the eye.

[72] A continuous involuntary muscular contraction of longer or shorter duration.

[73] Alternate contractions and relaxations of muscles.

[74] See note 63, supra.

[75] Inability to restrain a natural impulse; thus, here, unable to control the urge to urinate or defecate.

[76] The mucous membrane lining the inner surface of the eyelids and outer surface of the eyeball.

[77] A state in which grand mal seizures may continue for hours and days.

Between attacks, malingerers may simulate a state of imbecility,[78] but may betray themselves by introducing symptoms which are incompatible with the condition which they simulate. For example, they may display an amnesia[79] for past events which is most unusual in a mentally retarded epileptic.

Differentiation between simulated and hysterical seizures may sometimes prove difficult. A hysterical convulsive attack also occurs in the presence of an audience and usually, therefore, when the patient is out of bed. The patient does not injure himself in falling, nor does he bite his tongue or lose control of his sphincters; [80] he may be able to detail some of the events which occurred during apparent unconsciousness. The convulsion itself consists of hyperextension[81] or, more commonly, of more or less purposive throwing about with his limbs. The patient makes striking or grasping movements. During the convulsive jactitations,[82] he may laugh or cry alternately, or sing or whistle. He may respond to sharply uttered unexpected commands and carry them out correctly. The pupillary,[83] corneal[84] and other reflexes remain unchanged, and there are no pathological (abnormal) reflexes. The post-convulsive headaches, nausea and mental confusion so often seen in true epilepsy are rarely observed in hysterical patients and, in simulators, only when they have been "properly tutored." Confirmatory evidence as to the hysterical nature of an epileptiform attack[85] is previous knowledge that the patient has an infantile personality and the presence of motor and sensory hysterical conversion phenomena.[86] The absence

---

[78] A condition in which there exists from birth or from early age a mental defect which does not amount to *idiocy*. Imbeciles can understand spoken language and talk with a varying degree of fluency but are incapable of managing themselves, or their affairs, or, in the case of children, of being taught to do so, and who, according to the *Binet Simon Intelligence Tests*, fall in the age groups of 3, 4, 5, 6 and 7 years. They can be materially improved by training but not sufficiently so to enable them to take a place in the world.

[79] Loss of memory.

[80] Sphinteric disturbances: Involuntary urination and defecation.

[81] Extension involves a movement which brings the members of a limb into or toward a straight condition (straightening of back); hyperextension involves extreme or excessive extension (arching of back).

[82] Tossing to and fro.

[83] Each pupil, the other eye being covered, dilates and contracts as the eye is alternately shaded by the hand and exposed to light and the vision is constantly fixed upon some distant object. Normally when a pupil contracts to light (direct reflex), the pupil of the other eye also contracts (consensual reflex).

[84] Blinking and closure of the eye on touching gently the cornea with a wisp of cotton.

[85] An attack resembling epilepsy or its manifestations.

[86] A conversion of emotional disturbances into physical symptoms referable to motility, sensation, coordination, vasomotor and secretory functions.

of such phenomena and reliable information that there is a reason for pretending in a patient who has been observed to have an epileptiform attack whose symptomatology does not conform to that of true epilepsy, is presumptive evidence of simulation.

In this differentiation, the most difficult cases to diagnose are those in which, following a severe trauma (injury) to the head, the clinical (medical) picture of organic brain disease [87] is distorted by a psychogenic [88] elaboration and exaggeration of symptoms at the conscious, as well as the unconscious, level. Proper evaluation of such cases may be possible only after prolonged observation in a hospital, during which the patient is subjected to hyperventilation [89] with and without hydration,[90] blood sugar determinations, hypnosis,[91] suggestion [92] and after a study of the results of electroencephalography [93] and pneumoencepha-

[87] Brain disease involving structural damage to the brain tissue.

[88] Originating in the mind; psychogenic disorders as opposed to organic disorders have no organic basis for them.

[89] Hyperventilation (hyperpnia): excessive inhalation of air; this causes an alkalosis and thereby increases instability of the cortical potentials and in epileptics usually brings on seizure and changes in the electroencephalogram.

[90] A process by which the water content of tissues is increased. It has been found that in epilepsy there is a close relationship between the occurrence of seizures and change in the water balance of the body. A negative water balance, no matter how produced, has been shown to lessen markedly the tendency to convulsions, whereas a positive balance favors their recurrence. In view of this, *dehydration* is employed by some physicians as a therapeutic measure for the purpose of ameliorating epilepsy as far as the frequency of seizures is concerned.

[91] The production of sleep or hypnotism which is a mental state resembling sleep which is characterized by excessive suggestibility to the hypnotist and which is usually induced by psychological suggestion, by passes, or by having the subject focus the eyes on a bright object.

[92] A thought or idea conveyed from one individual to another.

[93] With this procedure the electrical potentials of the brain can be "led off" from the intact skull, amplified a millionfold or more, and made to trace a record on a moving film or paper. It is analogous to electrocardiography in the study of abnormal heart rhythms, except that the changes in the potential changes of the brain are only about one-hundredth the voltage of the potential changes of the heart. In recording the electrical activity of the brain, the electroencephalogram visualizes what is occurring in the brain during an epileptic seizure. An epileptic seizure is invariably accompanied by a disturbance in the electrical activity of the brain. The type of activity varies in different types of seizures. In *petit mal* the main characteristics of the rhythm are always the same (an alternation of large slow waves and sharp spikes), but the pattern of the waves is slightly different in each patient; in a given patient, successive petit mal seizure records are almost identical in form though not in length; the seizure record is the same whether the seizures are spontaneous or induced artificially as by hyperventilation. Patients suffering from epilepsy may show abnormal and characteristic disturbances of electrical activity without accompanying subjective or objective evidences of a seizure. In *grand mal* (which can be detected electroencephalographically before there is subjective or objective evidence of a seizure) there occur characteristic fast

lography[94] and of his reactions following intravenous sodium amytol injections.[95]

A useful test in the diagnosis of genuine (idiopathic) epilepsy is the so-called water-pitressin test. The test is based on the fact that in a genuine epileptic, seizures can be induced by intracranial, intracellular hydration following the administration of water and pitressin. During the test the patient should be kept in bed on an ordinary diet with a minimized salt intake. If luminal has been used it should be continued. An enema is given, the patient passes his urine, which is measured, and his weight is taken. An initial injection of 0.25 cc. of pitressin is given subcutaneously, and thereafter 0.5 cc. at 4 hour intervals until 10 injections are given, or until the patient has a seizure. If seizures occur before the tenth injection, water and the ordinary diet are stopped, and 5 ounces of cream are given every four hours until the seizures cease. The patient is weighed every four hours and for 16 hours after the discontinuance. The urine is measured at the same time and a chart of each test is made. If a positive water balance cannot be maintained, the amount of water given may be increased to 500 cc. at each time and the pitressin increased to 0.6-0.75 cc. per dose for one or two doses. A positive water balance must be induced in a valid test, i.e., there must be an increase in the patient's weight amounting to 3-6 per cent of the weight recorded at the beginning of the test. Untoward reactions, in addition to pallor, may be slowing of the pulse, headache and gastro-intestinal symptoms (abdominal fullness, nausea, vomiting and colic). If the latter and the headache are unusually severe, or a perceptible edema occurs, the test should be discontinued. The test is contraindicated in the presence of coronary artery disease, arterial hypertension, arteriosclerosis, arteriosclerotic heart disease and/or kidney disease. The procedure, as a rule, does not induce a fit in a person who is not an epileptic.

spikes; these may also be present in larval form. *Psychic variants of epilepsy* also seem to have a characteristic wave—a slow, flat-topped form.

[94] Encephalography is the visualization, by X-ray, of the cerebral cortex, intra-cerebral ventricles, subarachnoid and subdural spaces, following the removal of cerebrospinal fluid, by spinal puncture, and its replacement by air. This procedure is known as *pneumoencephalography*. In cases in which pneumoencephalography is contra-indicated because of .the presence of clinical evidences of markedly increased intracranial pressure (as in posterior fossa tumors), *ventriculography* is usually performed; this consists of the introduction of air into the ventricles of the brain after the withdrawal of fluid by "tapping," through a small trephine hole, the ventricles themselves.

[95] A hypnotic belonging to the barbiturates. In 3 grain doses injected intravenously it is helpful in neuropsychiatric conditions to establish contact with an uncooperative patient.

VI

SIMULATION OF ABNORMAL MOVEMENTS

The abnormal movements most commonly simulated are: tremors,[96] spasms,[97] especially blepharospasm,[98] and tics.[99] Here and there, a "trained" simulator may attempt to imitate choreiform[100] or dystonic movements;[101] but the patterns of these are so distinctive that the simulation can be readily detected. Localized tics can be simulated for a long while without fear of detection unless the patient can be "caught" free from them for a reasonable period.

In the evaluation of such abnormal movements as myokymia[102] or tremor, it is well to remember that excitement, fright, fatigue and exposure of the naked body in a cold examining room may produce shivering or shaking movements which, by themselves, are of no significance because they disappear as soon as the patient is relaxed and the examination conducted in a warm room.

A fine tremor of isolated groups of muscles or of an individual muscle or of the lids or tongue cannot be simulated. A tremor, however, limited to the head should arouse the suspicion of simulation. The oscillations (i.e. vibrations) of a genuine tremor generally do not vary during diversion of the patient's attention. Simulated tremors usually come on or become more intensified during the examination; they diminish in intensity or disappear entirely when the patient's attention is directed elsewhere. The oscillations of a genuine intention tremor[103] gradually become coarser and slower during action and become finer and more rapid as soon as the raised extremity is lowered. The oscillations of a simulated intention tremor also become coarser and slower during action, but their amplitude or range becomes more irregular,

---

[96] Trembling.

[97] A sudden, violent, involuntary contraction of a muscle or a group of muscles.

[98] A spasmodic contraction of the eyelids.

[99] Involuntary muscular movements which may involve a single muscle or a group of muscles; the movement is usually convulsive in character. It may be due to affections of the basal ganglia, or it may be psychogenic in origin (habit spasm).

[100] Involuntary purposeless jerky movements of various parts of the body usually observed in the choreas (Sydenham's chorea, or St. Vitus' dance, Huntington's chorea, encephalitis, etc.).

[101] Involuntary bizarre twisting and turning movements of the entire body or of segments thereof with alternating contraction and relaxation of the muscles; they are usually observed in some of the diseases affecting the basal ganglia (see diseases of the basal ganglia).

[102] A transient quivering of muscles.

[103] An intention tremor, sometimes called an *action tremor*, is one which appears or becomes more marked during activity of the trembling part.

and because of the patient's voluntary effort to persist with the trembling his pulse and respiratory rate usually increase markedly. A simulated tremor of the fingers may change in amplitude or disappear altogether during flexion or extension of the wrist or during pronation[104] and supination[105] of the forearm or during extreme flexion of the elbow; it may also disappear if the patient is requested to put his hands behind him or to assume a similarly uncomfortable position. A simulated tremor of the outstretched fingers will usually cease when the patient executes, on request, small regular movements of the thumb of the trembling hand. A tremor of the fingers is probably genuine when one or more fingers are artificially fixed and the others continue to tremble.

The fact that strong tetatinization[106] of a trembling muscle promptly suppresses the tremor may be utilized to determine the genuineness of a tremor. When a faradic current[107] applied to a trembling muscle is suddenly interrupted, although the battery is still allowed to buzz, and the tremor, without a free interval of time, regains its original intensity, it is probably genuine; but should a reasonably long period of time elapse before the reappearance of the tremor, it may be concluded that this interval was utilized by the patient to "decide to continue his trembling."

In a suspected simulated tremor of an arm from the shoulder down, Collie[108] suggests that the examiner put his entire weight on the trembling shoulder; if the tremor is genuine, the weight of the examiner on the patient's shoulder puts the latter at rest, and the tremor either ceases altogether or appears only in the distal segments[109] of the extremity; but if the tremor is simulated, the patient will attempt to bring it out more intensely with the shoulder, and the examiner resting on it, will perceive a resistance and voluntary contraction of the muscles of the shoulder.

Simulation of a tremor in the lower extremities may be readily detected by having the patient assume the squatting position in which volitional trembling is quite impossible. Another useful ruse is to request the patient to lie on a table on his abdomen, and to raise his legs

---

[104] Turning the arm, forearm or hand so that the palm is directed downward.

[105] Turning the arm, forearm or hand so that the palm is directed upward.

[106] The induction of tetanic (interrupted) contractions of a muscle with a faradic current.

[107] An intermittant alternating current obtained from the secondary winding of an induction coil.

[108] COLLIE, MALINGERING AND FEIGNING SICKNESS 118 (1917).

[109] The end farthest removed from the point of attachment or insertion.

by flexing the knees; if the tremor disappears while the patient is in this position but reappears as soon as he drops his legs and the toes touch the table, it is probably simulated.

A voluntarily induced tremor of the lower extremities will also usually disappear while the patient is running, holding a heavy weight in each hand.

In the absence of other evidences of hysteria,[110] it may be impossible to distinguish feigned tremors and tics from those so frequently observed in hysteria. Of possible diagnostic aid may be the fact that simulated abnormal movements cannot be maintained for as long a period as hysterical ones without producing fatigue, tachycardia (rapid heart beat), irregular and rapid breathing and excessive sweating.

VII

SIMULATION OF DISTURBANCES OF SENSATION

The determination with reasonable certainty whether an alleged pain is genuine, exaggerated or entirely absent may sometimes be almost impossible. Feigned pain is generally vague and not constantly located. In fact, almost any suggested spot on the body may be claimed to be painful. A simulator generally complains bitterly about pain and withdraws the "painful" part even before he is being hurt; he constantly looks at the part under investigation so as not to miss an opportunity to cry out with pain when the part is being manipulated. An appreciable interval of time between the application of the painful stimulus and the patient's groaning response should arouse suspicion of simulation. In genuine pain, the patient will cry out or wince almost instantaneously upon the application of the painful stimulus. In evaluating the presence and severity of alleged pain, there should be noted, in addition to the patient's statement, his facial expression, the presence or absence of muscle twitchings in and around the alleged painful area, as well as withdrawal or any other reflex and defensive movements[111] during the examination.

No final conclusion about the results of an objective sensory examination[112] should be made without taking into consideration the fact that, because of defects in concentration and attention, fatigue, undue

[110] See note 18, supra.

[111] Voluntary or automatic movements utilized for the purpose of self-protection against pain and other harmful stimuli; the most common defensive movement is the withdrawal of the part which is being hurt.

[112] Objective tests employed by the neurologist to determine whether the patient's responses to sensory stimuli are normal or abnormal.

suggestibility or prejudice, the patient may be unable to maintain a detached and impartial attitude, so that his sensory threshold [113] is subject to great variations and is therefore unpredictable.

Many tests and tricks are employed to detect simulated pain; they are all based on distracting the patient's attention in the hope that, being caught unawares, he will contradict himself in his responses to painful stimuli applied to the areas claimed to be painful. The success of all such tests depends upon the resourcefulness of the examiner and the naiveté of the simulator. With a "well trained" simulator, the most ingenious ruse may be futile.

A useful test is one based on the fact that even a mild faradic current [114] applied to a painful area accentuates the pain. For the purpose of the test, a faradic battery [115] is set in action, and the indifferent electrode [116] is applied to a part of the body remote from the alleged painful area; the electrode with the interrupting key is then gradually applied to an area at some distance from the painful spot, and, although the battery is still "buzzing," the interrupting key is so held that the current is entirely shut off; the patient is requested to state whether he feels the current as the electrode is being slowly moved toward the painful spot; an unsuspecting simulator, hearing the noise of the battery, assumes that the current is on and complains of "sharp" pain; should the simulator have been "coached" and the test fail, he is blindfolded and instructed to say when he perceives the current, which, he is told, will be turned on and off as the electrode is applied near and over the painful area; while the patient's attention is thus occupied in attempting to ascertain whether the current is off or on and going through the area which he claims to be painful, the examiner may succeed in exerting firm pressure with the electrode on the allegedly painful area without the patient's slightest complaint, proving that the area is not sensitive at all.

For the detection of simulated pain, Mannkopf's sign may sometimes be useful. This consists of a temporary acceleration of the pulse rate of from 10 to 30 beats per minute during pressure over a genuinely painful area or on the placing of the affected part in a painful position; in

---

[113] An individual's limits of appreciating sensory stimuli; that degree of stimulus which just produces a sensation which is perceptible to the patient.

[114] See note 107, supra.

[115] An electrical apparatus producing induction currents of electricity used for stimulation of muscles and nerves.

[116] The electrode which is placed on some far-distant "indifferent spot" where any muscular contractions that may occur will not interfere with the part which is stimulated and whose slightest response to the electric current is being looked for.

cases of pretended pain, the pulse rate generally remains unchanged; similarly, pressure over a painful area may also cause a temporary rise in arterial tension (arterial blood-pressure), a phenomenon which obviously does not occur in simulation. In evaluating these signs, it should be taken into consideration that muscular movements of non-painful parts may increase the pulse rate and arterial blood-pressure temporarily and that some individuals are able to accelerate their heart beat by directing their attention to the heart. It is imperative, therefore, that during these tests the patient avoid muscular activity and forced breathing and that his attention be distracted.

To some extent, the presence of genuine pain may be ascertained by watching the size of the pupils during pressure over the allegedly painful area; the latter procedure is usually accompanied by sudden dilatation of the pupil, although a similar phenomenon may be observed on pressure over the abdomen, even when the patient is not in pain, and on stroking or pinching the skin of the neck. In the absence of sudden paling or flushing of the face or of excessive sweating or of an intensification of a preexisting tremor upon the manipulation or compressing of an allegedly painful area, the genuineness of the pain may be questioned. So, indeed, it may in the absence of a history of insomnia (abnormal wakefulness), loss of weight, digestive disturbances and general ill-health.

The presence of evidence of hysteria in the absence of signs and symptoms or organic disease is no conclusive proof against simulation. The possible coexistence of hysteria [117] and wilful exaggeration of symptoms, especially in cases under litigation, must always be borne in mind. Although hysteria is a protean disease, nevertheless its clinical manifestations assume more or less characteristic patterns which are rarely observed with any constancy in deliberate simulation. This is particularly true of the symptom of pain.

Hysterical pain may be referred to any part of the body, but it is especially common in certain locations. Thus, hysterical headache, often of the "clavus" type, [118] is a boring pain in one small area of the skull. Occipital headache, [119] hemicrania (one-sided headache) and pain in the region of the mastoids [120] are very common in hysteria. While

[117] See note 52, supra.

[118] A headache in which the patients complain that they have a sensation of a nail being driven into the top of the head.

[119] Pain in the back of the head.

[120] A nipple-shaped process at the tip of the temporal bone (in back of the ear) containing irregular variously sized air chambers communicating with one another and with the antrum which is a large air chamber opening into the middle ear.

generalized hypersensitivity may occur, hyposensitivity (diminished sensitivity) over one half of the body is more common, and small islets of excessive hypersensitivity interspersed in the alleged hyposensitive areas are still more common. Particularly hypersensitive spots are usually found over the vertebral, inframammary,[121] · epigastric,[122] ovarian and inguinal regions; [123] they are less frequent on the head and very rare on the extremities. Pressure over these hypersensitive spots not infrequently may provoke a "hysterical fit."

The most characteristic hysterical sensory disturbance, especially in posttraumatic cases, is diminution of perception of cutaneous as well as of deep sensory stimuli over one half of the body, usually the side on which the injury has occurred. In some cases, the alleged diminution of sensibility does not necessarily affect simultaneously and to the same degree all types of sensory perception, e.g., pain alone or touch alone or temperature alone or various combinations of these may be affected; at times, the vibratory sense [124] alone or the sense of position alone is affected; in other cases, the sensory disturbances, though predominatingly unilateral (i.e., on one side), may be more marked on the face or on the limbs than on the trunk of the affected side and, as has been stated above, the hyposensitive areas may be interspersed with islets of hypersensitivity to one stimulus or another. In addition to the objectively demonstrable sensory disturbances just described, many hysterical patients also complain of numbness, tingling or formication [125] over the affected parts.

In attempting to distinguish simulated from hysterical and from genuine sensory disturbances, the examiner must avoid all suggestive and leading questions. Areas of diminished or increased sensitivity at variance with anatomical nerve distribution should arouse suspicion of malingering in cases of peripheral nerve injury,[126] although it is important to bear in mind that anatomic nerve distributions may be subject to variations in different persons. When the alleged sensory dis-

---

[121] Underneath the nipple.

[122] The upper middle portion of the abdomen lying over the stomach.

[123] The groin.

[124] Vibratory sensation: The perception of vibration from a tuning fork pressed against the bony prominences of the toes, fingers, ankle, wrist, shin bone, olecranon process (the curved process projecting upward from the back part of the ulna at the elbow), iliac spines, collar bones, ribs and sternum (breast bone). Diminution or loss of this sensation indicates an affection of the nervous mechanism subserving deep sensibility.

[125] A sensation of insects crawling over the body.

[126] Injury to the portion of the nervous system which lies without the central mass of the brain and spinal cord.

turbances are more or less constantly limited to the same limb in which, on repeated testing, there are also found changes in the motor power (power of muscular contraction) and reflexes, there is every probability that they are neither simulated nor hysterical. Insensitiveness of the cornea [127] can hardly be simulated. Useful information about the truth- fulness of a patient's responses during the sensory examination may also be obtained by varying the intensity of the stimulus and by comparing the sensitiveness in various areas which are normally less sensitive with others which are more sensitive. If, for example, there is elicited slight diminution of sensation over a certain area, this will be especially evi- dent as regards epicritic tactile sensibility,[128] whereas firmer tactile stimuli will be perceived or, should there be acknowledged a diminu- tion of tactile sensibility over the leg, tactile stimuli will not be felt at all on the soles or balls of the toes, where normally they are perceived but slightly and, in other places, they will be less intensely felt. Gross contradictions in the patient's responses during these tests may be con- firmatory evidence of untruthfulness.

Although diminished skin electrical resistance [129] and normal dermal reactions following histamin [130] injections in areas allegedly insensitive to pain are against the presence of organic disease of a peripheral nerve in the tested area, the mere absence of organic disease does not neces- sarily imply that the patient is malingering, since similar reactions to these tests may also occur in hysteria.

Valuable as are the above-mentioned criteria about the genuineness of a sensory disorder in some cases of suspected malingering, they can- not, by themselves, be regarded as absolute, but must be evaluated along with the rest of the clinical picture and the patient's social, eco-

---

[127] The horny circular transparent membrane which forms the front portion of the eyeball.

[128] The appreciation of fine touch.

[129] By studying the resistance of the skin to the passage of a minute direct current through it, it is possible to map out areas affected by injuries of peripheral nerves as well as to map out areas of referred pain and areas affected by tumors or other destructive processes of the spinal cord. The successful use of this method does not depend in any way on the cooperation of the patient. In cases of suspected simulation of pain the elec- trical resistance of the skin as determined by a dermometer supplies a more reliable test than the patient's subjective report.

[130] A substance which occurs in combined forms in all tissues and is liberated in putrefaction (decomposition of albuminous or other complex nitrogenous principles by the agency of various bacteria and fungi). It is destroyed in the digestive tract, but when injected intracutaneously or intravenously it produces an extensive fall of blood pressure due to dilatation and increased permeability of the capillaries; the cutaneous reaction of histamin is used as a test for peripheral nerve function and also as a test for vasomotor (circulatory) affections.

nomic and psychic status, and especially with the merits of his legal, claim.

VIII

SIMULATION OF DISORDERS OF COORDINATION

The most frequently simulated disorders of coordination are ataxia,[131] the Romberg phenomenon [132] and vertigo.[133]

Simulated ataxia of the upper limbs can readily be detected by watching the patient's performance during the finger to nose, finger to finger and finger to ear tests. A simulator who exhibits a "wild" ataxia during these tests may show no difficulties in buttoning und unbuttoning his clothes or in lacing and unlacing his shoes when he is engaged in conversation. He may claim inability to write well with a pencil or pen on a ruled straight line and betray himself by writing well on unruled paper or vice versa.

When a patient with genuine ataxia is requested to pick up a small object, say a pin, from a smooth surface, he fumbles during the attempt or pounces on the object in a sudden, jerky way; a simulator either makes no attempt to pick up the object or keeps on moving it around, shaking his fingers, and finally picks it up slowly and with deliberation. A simulated ataxia is generally as "wild" with the eyes open as when they are closed. A simulator rarely attempts to reach an object by the shortest way and, unless he has been carefully "coached," makes it evident that he is volitionally augmenting the requisite degree of force by contracting forcibly the muscles necessary for the performance of the required movement. A naïve simulator may have ataxia only during attempts to reach a goal on his own body, but not during attempts to pick up an object or to touch the tip of the examiner's finger. He may also betray himself by having ataxia only in one or two fingers or only in one segment of the limb; or he will be atactic[134] only during the beginning of the examination or while he is being tested for ataxia and, as the examination progresses, the assumed ataxia gradually disappears.

In genuine ataxia, the raised atactic extremity cannot be kept still,

---

[131] Incoordination: the state in which the various movements required for the performance of any act are improperly adjusted to each other so that the act is carried out imperfectly. It may be peripheral, from a lesion of the peripheral sensory neurones, or cerebellar, due to a lesion of the cerebellum or its tracts, or vestibular, due to a lesion of the vestibular nerve, or cerebral, due to a lesion of the cerebral hemispheres.

[132] The Brauch-Romberg symptom: Swaying of the body when the patient stands with the feet together and the eyes closed; it indicates incoordination of station.

[133] (Giddiness; dizziness) A disordered condition of the sense of equilibrium, giving rise to a feeling of unsteadiness and a sensation of an apparent movement either of the body itself (*subjective vertigo*) or of surrounding objects (*objective vertigo*).

[134] An individual suffering from ataxia (incoordination).

but oscillates in the vertical or in the horizontal plane; in simulated
ataxia, the allegedly affected extremity will either remain still or move
alternatingly in all planes.

In the examination for ataxia in the lower limbs, whatever informa-
tion as to its nature may be obtainable by observing the patient's gait
and station (vide supra), additional information will become available
by studying his performance of the heel to knee test [135] or while he traces
some design on the floor with his allegedly atactic leg. During these
tests, an ataxia which is as severe with eyes open as when they are shut,
should arouse suspicion of simulation. In true ataxia, when the patient
is on his back and is asked to raise the atactic limb, it is not lifted straight
and steadily in the vertical plane, but is adducted [136] or abducted,[137]
rotated inwards or outwards, and thrown from one position to another
and, when finally raised, it oscillates more or less continuously either in
the vertical or in the lateral plane; the patient, being asked to bring
down the oscillating raised limb, does not bring it down slowly, but
drops it down, not alongside the other leg, but either across it or away
from it or in some other position. A simulator, ignorant of these
phenomena, will perform all sorts of fantastic "stunts" which will read-
ily betray him. In evaluating these tests, it is well to bear in mind that
in so-called pseudoataxia [138] due to motor weakness, the raised limb may
also oscillate, but it will do so in the vertical plane only, and the oscil-
lations will be noticeably increased when the eyes are shut.

During the Romberg test,[139] a patient who is truly atactic, no matter
how severely he may sway, will invariably make every effort to main-
tain the erect posture and to save himself from falling. A simulator
generally begins to sway long before his eyes are shut and the feet ap-
proximated; he falls as soon as his eyes are shut, usually en masse and
backward, without making the slightest effort to maintain his equili-
brium. When a simulator is requested while in the Romberg position [140]

---

[135] A test employed to determine the presence of incoordination in the lower
limbs. Inability to touch the knee with the heel of the other foot with eyes open, and/
or shut, provided there is no defect in the joints of the knee and ankle and no paralysis
or marked weakness of the limbs is indicative of the presence of disturbance of coordi-
nation (ataxia) of the limb whose ankle touches the opposite knee.

[136] Adduction: A drawing of a limb or part of a limb inward or toward the
median axis of the body.

[137] Abduction: A drawing away of a limb or part of a limb from a position paral-
lel to the median axis of the body.

[138] Not a genuine ataxia (incoordination); usually observed in hysteria and in
malingering.

[139] See note 132, supra.

[140] Ibid.

to place both his hands on the examiner's palms, he usually makes little or no effort to touch the examiner's hands, but immediately begins to sway and fall. In contrast, a patient with true ataxia, especially a tabetic,[141] grasps the examiner's hands firmly to give himself support and avoid falling.

Erben performs the Romberg test [142] in a suspected simulator in the following way:[143] he makes the patient sit up straight in bed; in this position, he usually sways markedly with the upper part of his body, whereas a true atactic will sway much less because the broad sitting surface allows him to maintain his balance better than the narrower supporting surface of the soles of his feet, when in the erect posture, and because, in the sitting posture in bed, the contact of the entire length of his lower limbs furnishes him with more reliable information as to his position in space.

Freund and Sachs employ the following ruse to detect a simulated Romberg phenomenon: while the patient stands with his feet closely approximated, he is commanded, in rapid succession, to touch his nose, chin, ear and other parts of the body, now with the right index finger, and now with the left; during the execution of these commands, he is suddenly ordered to shut his eyes and other similar commands are given so rapidly that he has no time to deliberate over the situation and usually "forgets to sway and fall."

Schuster unmasks a simulated Romberg by the following trick: the patient stands up with both feet closely together; he is then ordered to shut one eye while the examiner repeatedly tests the pupillary light reaction in the open eye; gradually, the light is shut out and, if no sway-

[141] A person suffering from tabes dorsalis (locomotor ataxia). This is a chronic disease of the nervous system characterized by degeneration of the posterior columns of the spinal cord and the centers for pupillary light reaction and of the sensory nerve trunks, and manifested clinically by the presence of lightning-like pains, abolition of the tendon and periosteal reflexes, contracted pupils that do not react to light, peculiar paroxysms of pain (so-called *tabetic crises*) in the stomach, larynx or other viscera, trophic disturbances (nutritional disturbances) of the bones and joints, impairment of sensation (vibration and sense of position of the joints), impairment of sexual power, retention or incontinence of urine and feces and progressively increasing incoordination of movement (ataxia). It is a disease of middle life, especially frequent in males and is due to syphilitic affection of the nervous system. The pains and abolition of reflexes and pupillary disturbances are usually the first symptoms, forming the preataxic stage of the disease. Its course is slow, usually progressive, and may be associated with general paresis, commonly known as "softening of the brain"; in such cases the condition is generally spoken of as *taboparesis*.

[142] See note 132, supra.

[143] ERBEN, DIAGNOSE DER SIMULATION NERVOSER SYMPTOME, Berlin, 3d ed., 62 (1930).

ing occurs during this procedure, the patient is probably pretending a positive Romberg.[144]

In a simulated Romberg, there are never observed the irregular restless contractions of the tendons of the extensors of the foot, nor the tendency of the planter surfaces (soles) of the feet to "grip" the floor—phenomena usually observed in the tabetic atactic, who automatically performs these movements to secure a firmer base for his proper orientation in space.

In the evaluation of the significance of a positive Romberg it is well to remember that slight swaying movements occasionally occur in anxious individuals even though they are free from organic nervous disease; this type of swaying, however, gradually recedes as the examination is continued and as the patient recovers his poise. In doubtful cases, Oppenheim's procedure [145] of eliciting a Romberg may furnish valuable information. This consists of asking the patient to bend forward as far as he can and then to raise himself, first slowly and then more rapidly, with eyes closed; this will usually increase the swaying if it is genuine. A simulator who has been "taught" to sway only when he is in the erect posture with eyes closed may betray himself by not swaying at all when bending down and getting up.

In some cases, it may be impossible to distinguish simulated from hysterical incoordination. In both forms, the ataxia may be made to disappear by distracting the patient's attention. As a rule, a simulator who has "trained" himself to imitate incoordination is less apt to show the periodic variability of the symptom than the hysteric. On the other hand, hysterical ataxia may disappear following suggestion as suddenly and as dramatically as it originally appeared, whereas simulated ataxia will disappear only after it has served its purpose, unless it has been exposed before that.

The detection of simulated vertigo [146] is probably one of the most perplexing problems that a physician may be called upon to solve. Anything which alters an individual's spatial relation may produce vertigo. Some of these conditions are: diplopia,[147] sudden change of position of

---

[144] See note 132, supra.

[145] OPPENHEIM, TEXT-BOOK OF NERVOUS DISEASES, trans. by Alexander Bruse, p. 147 (1911).

[146] See note 133, supra.

[147] Double vision: a condition in which an object seen appears double; *monocular diplopia*, in which one eye sees objects double, is due to irregular astigmatism, early cataract, or any condition producing a double pupil; it is also observed in hysteria and in malingering; *binocular diplopia*, in which each eye sees singly, but both together

the head, especially rotatory in direction, looking down from great altitudes, intracranial hypertension,[148] affections of the cochlear-vestibular apparatus,[149] disease of the cerebellum[150] or its pathways, cerebral (brain) and general circulatory disturbances from whatever cause, migraine,[151] the epilepsies, gastrointestinal disorders, hepatic disease,[152] acute and chronic intoxications, the anemias, neuroses and psychoses, not forgetting impacted cerumen.[153] Vertigo may be experienced, as Oppenheim has pointed out, in normal individuals by closing the eyes and standing on one foot. Vertigo (subjective or objective or both) is probably the most frequent sequel of head injury; in these cases, it is noteworthy that the severity, extent and nature of the injury may bear no relation to the severity and duration of the vertigo.

True vertigo is often accompanied by nystagmus,[154] nausea and vomiting, pallor of the face, feeble pulse, excessive sweating and some incoordination, especially of the lower extremities. The absence of these symptoms, however, is not inconsistent with the presence of true vertigo. Similarly, if, during the examination, the patient with eyes shut is made to bend forward and touch his toes with the tips of his fingers and then suddenly, to straighten up, approximate the feet and open his eyes, and he does not complain of vertigo (giddiness, dizziness) one cannot say that the patient may not have had vertigo on other occasions. Certainly, no diagnosis of simulated or hysterical vertigo should be

see double; it is due to squint paralysis, or insufficiency of the ocular muscles, or displacement of the eyeball causing the axis of one to deviate from the object of fixation; binocular diplopia is *homonymous* when the image seen by the left eye is on the left side and vice versa, in which case the visual axes converge too greatly; *heteronymous* or *crossed diplopia* occurs when the image seen by the left eye is on the right side in which case the visual axes diverge; *vertical diplopia* is the condition in which one image stands above the other, in which case the eye corresponding to the lower image is relatively too high.

[148] The pressure under which the cerebrospinal fluid circulates within the cranium. It is usually measured by a water or mercury manometer through the lumbar puncture needle.

[149] This term is employed to designate the anatomico-physiologic mechanism subserving hearing (cochlear) and the maintenance of equilibrium (vestibular).

[150] A portion of the brain which regulates coordination of complicated movements.

[151] A paroxysmal nervous disorder associated with headache, often unilateral and usually severe, and often ushered in by visual disturbances and frequently accompanied by nausea and vomiting (*sick headache*). It usually runs in families.

[152] Disease of the liver.

[153] Ear-wax.

[154] An involuntary rapid movement of the eyeball which may be either lateral, vertical, rotatory, or mixed, i.e., composed of two varieties.

made until the Bárány[155] and hearing tests[156] have demonstrated the absence of organic disease of the cochlear-vestibular apparatus.[157] After all known causes of vertigo have been excluded, one must be guided not only by the results of the various examinations, but also by all surrounding circumstances before giving an opinion that a symptom like vertigo is simulated.

IX

SIMULATION OF DISORDERS OF SPEECH

The disorders of speech most commonly simulated are stuttering or stammering, aphonia,[158] and mutism.[159]

In genuine *stuttering,* there occurs repetitive vocalization because of difficult enunciation of certain consonants and vowels. The disturbance is manifested only at the beginning and end of a syllable, mostly at the beginning of an important word, phrase or sentence. The word or syllable which is the "stumbling-block" is emitted explosively and is repeated several times with accompanying spasmodic contractions of some of the speech muscles and excessive movements of expression, mimicry and gesticulation, rapid breathing and, in severe cases, with shaking of the entire body. After this momentary disturbance is overcome, speech becomes smooth and fluent till the appearance of the next "stumbling-block." The entire performance represents an "internal struggle" to speak which is rarely observed in pretended stuttering and, if it does occur there, it cannot be continued for any length of time without its becoming obvious that the individual is making an unusual voluntary effort. In simulated stuttering, speech appears inhibited regardless of any particlar consonant or vowel.

Usually, a simulator stutters at the onset of the examination, but the speech becomes smooth as the examination continues. Simulation of stuttering should also be suspected when its severity is significantly varied at different periods of the examination. Genuine stuttering usually disappears when the words are sung or whispered; a simulator, not aware of this fact, will continue to stutter while singing and whispering.

[155] These are tests used for the determination of the vestibular nerve and labyrinth. (See vestibular nerve, note 207, infra.)

[156] Hearing is tested by conversation, ticking of a pocket-watch, tuning forks of high and low pitch, Galton's whistle, audiometer, and a Bárány noise apparatus.

[157] This term is employed to designate the anatomico-physiologic mechanism subserving hearing (cochlear) and the maintenance of equilibrium (vestibular).

[158] Speechlessness due to affections of the organs of voice and not to brain disease.

[159] Dumbness.

In pretended stuttering, in contrast to hysterical stuttering, the impediment of speech rarely begins suddenly, nor can it be made to disappear following suggestion, hypnosis and speech re-education.

Aphonia[160] is less frequently simulated than *mutism*.[161] Both these disorders of speech are much more common in hysteria.

Hysterical aphonia, like other hysterical manifestations, comes on suddenly after an emotional crisis; the voice is generally completely lost, the patient speaking only in whispers.[162] Laryngoscopic examination[163] discloses the vocal cords in abduction or partial abduction during phonation.[164] A similar laryngoscopic picture may also be seen in simulated aphonia, but hysterical aphonia is generally associated with diminished or lost sensitivity of the pharynx[165] and larynx and a depressed or lost gag reflex; such sensory and reflex changes are usually not encountered in simulated aphonia.

Simulated mutism must be distinguished from aphasia[166] and hysterical mutism. An aphasic, no matter how speechless, tries to speak; a hysterical mute may make a great effort, but cannot produce a tone, not even a whisper; when he attempts to speak the lips and tongue appear completely motionless, although they may be moved otherwise, ,or they may be distinctly contracted without producing any sound. In *aphonia*, the patient's behavior leaves no doubt that he understands everything and yet is more "dumb" than an aphasic, who can generally

[160] See note 158, supra.

[161] See note 159, supra.

[162] Judge Ulman vividly describes the case of an actress known as "The Sweet Singer of the South," who was grazed when defendant's stage hands negligently manipulated a curtain so that part of it fell in plaintiff's locale. Plaintiff swooned, and while no medical evidence could be found of injury to her head or neck, she developed hysterical aphonia. In a suit against defendant, plaintiff recovered a jury verdict for $50,000, and this was later settled for $40,000. Within a year or two following the settlement, plaintiff had fully recovered her voice and was again giving concerts. ULMAN, A JUDGE TAKES THE STAND (1933).—*Ed*.

For other court cases involving an impairment of speech on a hysterical basis, see the following: Ross v. Clark, 35 Ariz. 60, 274 P. 639 (1929); Kupke v. St. Louis Transit Co., 122 Mo. App. 355, 99 S.W. 472 (1907); Davidson v. St. Louis Transit Co., 211 Mo. 320, 109 S.W. 583 (1908); Weissman v. Wells, 306 Mo. 82, 267 S.W. 400 (1924); Dowd v. McGinnity, 30 N.D. 308, 152 N.W. 524 (1915); Morris v. International Ry. Co., 174 App. Div. 61, 159 N.Y.S. 993 (1916).—*Ed*.

[163] The examination of the interior of the larynx (voice box) by means of the laryngoscope, an apparatus which permits ocular examination of the larynx.

[164] The production of articulate sounds.

[165] The musculomembranous sac between the mouth and nares and the esophagus. It is continuous below with the esophagus, and above it communicates with the larynx, mouth, nasal passages and eustachian tubes.

[166] An impairment in the expression of ideas by speech (reading, writing, etc.) due to affections of the speech centers in the brain.

utter a few sounds or words or parts of words. In some hysterical pa-
tients, phonation may be unimpaired and the mutism incomplete, so
that with a visible effort he enunciates the first sound or syllable of a
word, then, after a pause, the second, but no more, thus differing from
the aphasic, who either can say nothing or continuously perseverates
(repeats) the same words or sounds. In simulated mutism, the patient
hears nothing, understands nothing and does not make the slightest
effort to move his lips and tongue, nor to raise his soft palate. A hyster-
ical mute can read and write, whereas one pretending mutism generally
neither writes nor reads. Hysterical mutism disappears as suddenly as
it comes following suggestion, hypnosis, an administration of a general
anesthetic or intralaryngeal faradization. A simulator will not get
better from suggestion, nor from hypnotism and will probably not sub-
mit to general anesthesia nor to intralaryngeal faradization.[167]

X

SIMULATION OF DISTURBANCES OF THE SPECIAL SENSES

Simulation of disturbances of the special senses is fairly common
among litigants claiming disability following head injury. A simulated
anosmia[168] or *ageusia*[169] in a "trained" malingerer, as a rule, cannot be
determined with a reasonable degree of certainty. A naïve simulator
may give himself away when he is requested to smell a very disagree-
able substance and, being unaware of the ruse, responds with the reflex
movement of narrowing the nostrils with a facial expression of disgust.
Of course, in performing this test for the sense of smell, one must be
certain that the substance smelled is not strong enough to irritate the
sensory nerve endings in the mucous lining of the nose derived from
the trigeminus nerve.[170] Complete bilateral loss of the sense of smell,
in the absence of structural changes within the nose or of organic disease
of the brain, is strongly suggestive of simulation. Hysterical anosmia
is usually unilateral and on the side of the hysterical motor or sen-
sory paralysis;[171] in such cases, the other special senses are also usually

---

[167] The application of a faradic current to the vocal cords.

[168] Loss of the sense of smell.

[169] Absence of the sense of taste.

[170] *Trigeminus nerve (Trifacial nerve)*: The fifth cranial nerve whose motor
division innervates the muscles of chewing and whose sensory divisions supply the
face and scalp.

[171] Motor paralysis is the inability to carry out voluntary movements; sensory
paralysis is the inability to perceive sensory stimuli for pain, temperature, touch, vibra-
tion, two point discrimination, joint sensibility and the recognition of the shape,
weight, texture and nature of objects by tactile (touch sensibility) stimuli (impaired
stereognostic sense).

involved on the same side. Unlike hysterics, simulators rarely complain of hyperosmia[172] or parosmia.[173] In this connection it is well to remember that one affected with organic anosmia, despite the preservation of the organs of taste, is unable to distinguish flavors, for the recognition of which the sense of smell is essential; if this disability is preserved despite an alleged anosmia, the latter is either simulated or hysterical in nature.

In testing for taste, due consideration must be given to the fact that the sense of taste is most keen at the edges and top of the tongue in its anterior two-thirds, whereas the posterior third and the region of the palate and pharynx are much less sensitive. A suspected simulator claiming partial loss of taste may be detected by repeating the tests from day to day and noting his responses, bearing in mind that acids are tasted on the anterior[174] parts of the tongue better than on the posterior parts, while the latter and the soft palate[175] are more sensitive to bitter tastes.

XI

SIMULATION OF BLINDNESS

Simulators rarely pretend total bilateral blindness. They usually claim more or less complete unilateral blindness. Exaggeration of a preexisting visual defect due to corneal opacity,[176] to myopia[177] or to other refractive errors[178] or to congenital blindness[179] is quite common. The unexpected discovery of a need for strong convex lenses[180] may be coincidental with a recent head injury and be the motivating factor in a claim that the visual defect is attributable entirely to the injury. A blind or amblyopic eye[181] frequently squints when the sound eye is

[172] Excessive sensitiveness in appreciating odors.

[173] Perversion of the sense of smell.

[174] Situated before or in front of.

[175] The palate is the roof of the mouth; the soft palate is that part near the uvula toward the back of the mouth.

[176] A condition of the cornea which renders it impervious to light.

[177] Near-sightedness.

[178] Any defect in the optical apparatus which prevents a separate and distinct visual impression being focused on the retina gives rise to an error of refraction. There are three different forms of abnormal refraction: 1. *hypermetropia* in which the principal focus of parallel rays of light lies behind the retina; 2. *myopia*, or short sight, in which the principal focus of such rays lies in front of the retina; 3. *astigmatism* in which the refraction of the eye is different in its different meridians.

[179] Blindness present at birth.

[180] A lens which brings light to a focus.

[181] Amblyopia is a decided impairment, but not complete loss of vision, especially for colors in the early stages. It is sometimes congenital (from birth), occurring then in connection with other congenital defects of the eye. When acquired it may be due

fixed. A statement that an eye which presents a squint has suddenly and recently become blind is suspicious of simulation, because the production of the squint is generally a gradual process following prolonged disorganization of vision.

Detection of simulated blindness is not always easy. One must have opportunity to observe the patient without his knowledge. The malingerer usually wears dark eyeglasses. An individual totally blind for a long time has acquired confidence in the guidance of another person, so that he usually walks briskly when led by the arm, but, when unaccompanied, he walks with a short-stepped gait. An untutored malingerer walks normally, taking care to avoid chairs and other obstructions placed in his way.

The pupil of a genuine totally blind eye is dilated and does not react to light, but, if only feeble vision still remains, the pupil will react to light, so that a persistently good reaction to light in an alleged totally blind eye is strongly indicative of malingering although the presence of the pupillary reflexes [182] to light is no proof that the patient sees, for this would be quite compatible with a cortical lesion [183] causing total loss of sight. The failure of dilated pupils to expand more when the eyes are in a shadow, if no lesion of the ocular fundus [184] is demonstrable, should arouse suspicion of the fraudulent use of a mydriatic, [185] especially if the conjunctivae are also found to be injected (congested and inflamed); "tutored" malingerers, however, will avoid the injection of the conjunctivae by the instillation of a mydriatic a day or two before the examination. In simulated blindness, ophthalmoscopy [186] reveals no abnormalities.

The presence of nystagmus [187] in a totally blind eye in which fixation

to prolonged disuse of the eye, as from a squint or cataract; to peripheral irritation (reflex amblyopia): or to central causes, such as in hysterical amblyopia, and toxic amblyopia (the latter including particularly alcoholic amblyopia from chronic poisoning with alcohol, and amblyopia nicotinica, from the excessive use of tobacco).

[182] Each pupil, the other eye being covered, dilates and contracts as the eye is alternately shaded by the hand and exposed to light and the vision is constantly fixed upon some distant object. Normally when a pupil contracts to light (direct reflex), the pupil of the other eye also contracts (consensual reflex).

[183] A lesion involving the surface of the brain.

[184] The interior of the eye.

[185] A drug which causes dilatation of the pupil.

[186] Examination of the eye with an ophthalmoscope, an instrument which casts a beam of light and enables the examiner to view the interior of the eye, especially the optic nerve, the retina, choroid and blood vessels.

[187] An involuntary rapid movement of the eyeball which may be either lateral, vertical, rotatory, or mixed, i.e., composed of two varieties.

is impossible is in favor of genuine blindness. When a blind man is asked to look at his own hand placed in front of his face, he will attempt to look at it, not far from its true position because, by the help of general sensation, he is aware of the relative position of the hand and face; when a simulator is requested to place his hand in front of his face, not infrequently he will put it to the side; when the patient's hand is placed by the examiner in different positions and he is asked to look at it, he will make no attempt to follow the direction of the hand but will look elsewhere purposely (Schmidt-Ripler test). In the Burkhardt modification of this test, the patient is requested to touch the forefingers of his two hands. The blind person will do so, whereas the simulator usually misses. A simulator may betray himself by wrinkling his forehead in order to inhibit the light reflex[188] to sudden stimulation.

An alleged inability to read anything but the large type of Snellen's chart[189] may be unmasked by the following device: the patient is asked to stand 20 feet from a looking glass in front of which is a Snellen's chart; after he has reached the last line which he states he is able to read, the chart is removed from in front of the mirror, and the patient is asked to stand 10 feet nearer the mirror; another Snellen's chart with letters of the same size as on the first chart, but printed backwards, is then placed in front of his chest as he faces the mirror, and he is asked to read the letters which he sees in the mirror. Being half the distance from the mirror, he may be induced to read twice the number of lines he read when he stood 20 feet from the mirror, if he is ignorant of the laws of reflection.

A fraudulent claim of defective vision in one eye, short of blindness, may be detected by a ruse which is based on two facts: (1) if both eyes are open, it is impossible to know with which one sees an object and (2) anything colored red cannot be recognized as red if looked at through a red glass by reflected light. A chart on which there is a line of letters, alternatingly colored white and red, is placed in front of the patient and a red lens in front of the "good" eye, and he is asked to read the letters on the chart; if he reads all the letters—both red and white—he can see with the allegedly blind eye. Unilateral feigned blindness may be unmasked by the following test: the patient is asked to read small print from a book; suddenly, while he is

---

[188] A circular spot of light seen reflected from the retina with a retinoscopic mirror.

[189] These are test type charts consisting of block-letters drawn to scale, so as to be just distinguishable at a given distance; they are used for determining visual acuity.

reading, a pencil is placed in the middle of the page or 3 to 4 inches in front of it; if he sees with both eyes, he will continue to read because he can "see around" the pencil; but if one eye is blind, he will stop reading, because one or two words on each line are hidden by the pencil. Duane employs the following ruse to detect simulated blindness in one eye: he renders the sound eye myopic[190] by placing a convex lens of 5 diopters (diopter is the refractive power of a lens with a focal distance of one meter: it is assumed as a unit of measurement for refractive power) in front; assuming the eye is emmetropic,[191] its far point[192] now is about 8 inches distant and with this eye, the patient cannot read fine print further away. The patient is first asked to read aloud with both eyes at quite a short distance and, now, if the print is gradually withdrawn farther than 8 inches and he nevertheless continues reading, he is obviously seeing with the allegedly defective eye which he brings into use when the artificially induced myopic eye is put out of range by the withdrawal of the print.

Another ruse employed by Duane to discover feigned unilateral blindness is to place a prism[193] of 4 degrees with the base downward before the pretended blind eye while the patient is reading aloud; if the vision in that eye is defective, as contended, the prism in front of it will not affect the ability to read; but, if he sees with that eye, the prism will produce double images with consequent confusion and inability to continue reading and at the same time the eye will rotate inward for the sake of single vision, an effort which a blind eye would not make.

Harlan places a plane or weak concave glass minus 25 diopters before the pretended blind eye and a strong plus glass, say 16 diopters, before the sound eye; if the patient reads the distant type, he sees with both eyes.

A simple device to test for monocular blindness[194] is Bishop Harman's diaphragm test which is based upon two facts: (1) that a person

---

[190] Near-sighted.

[191] An emmetropic eye is one in which when its accommodation is relaxed, and the eye is correctly adjusted for parallel rays, so that in case no opacities exist in the media, distant objects form a sharp image upon the retina.

[192] The farthest point at which, with the greatest relaxation of accommodation, objects can be distinctly seen.

[193] A solid with a triangular or polygonal cross section. A triangular prism splits up a ray of light into its constituent colors and turns or deflects light rays toward its base. Prisms are used to correct deviations of the eyes, since they alter the apparent situation of objects.

[194] Blindness in one eye.

is unable to tell with which of his eyes he is seeing when both are open and (2) that objects on the right side are seen by the left eye, and those on the left side, by the right eye. The test is performed as follows: A flat ruler 18 inches long is constructed so that at one end there is a wooden carrier, set at right angles, on which is placed a small card with letters or numbers on it; five inches from the carrier, there is a small vertical screen, pierced by a hole three-quarters of an inch in diameter; the end of the ruler opposite the carrier is placed on the patient's upper lip, and he is asked to read the letters or numbers on the card through the hole in the screen; if the card has the letters A B C D E F G on it and the patient reads only D E F G, it is obvious that he does not see with the right eye, whereas, if he reads only A B C D, it is equally clear that he is using only his right eye and that the left is defective because of the crossing of the visual axes.[195]

To detect feigned monocular blindness, the stereoscope[196] may be employed in various ways. The patient's eyes must be exposed constantly to view, and the examiner must be certain that the patient does not see the lettered cards before they are inserted into the slide. If a card with the letter L is placed on one side and another card with the letter F, on the other, the combination of both making the letter E, and the patient states that he sees an E, he has no monocular blindness. Other figures, letters and designs which differ on the two sides may be used in a similar manner.

There are many other devices employed to detect simulation of blindness, but they are too complicated, and some require special apparatuses and technics. If a suspected malingerer cannot be unmasked by the relatively simple procedures described above, he should be referred to an ophthalmologist[197] for further investigation.

A. *Simulation of Visual Field Disturbances*

In the absence of evidences of organic disease of the visual apparatus, a smaller visual field[198] for blue than for red should arouse suspicion

---

[195] The visual axis of the eye is an imaginary line passing through the center of the cornea and the posterior pole of the globe of the eye.

[196] A binocular optical instrument by means of which two pictures appear as one. and stand out in relief.

[197] A physician who specializes in the diagnosis and treatment of affections of the eye.

[198] The space within which, when one eye is closed, objects can be seen by the other eye, the gaze of the latter being fixed on some one object or point (see hemianopsia).

that the patient consciously or unconsciously is not telling the truth. Simulation may be detected in the following manner: the patient is subjected to a perimetric examination,[199] and the results are charted; at a subsequent examination, the distance of the test object is either increased or diminished, and correspondingly there should be an increase or diminution in the size of the field; however, should the field remain within the same limits despite the variation of the distance of the test object, the patient is probably simulating. According to Wilbrand, a wider visual field when the second examination is conducted after the patient's prolonged stay in a dark room is also suggestive of simulation. When perimetry is not feasible, the following simple procedure may betray a simulator: a board or large sheet of paper is taken and, on it, there is mapped out the patient's field of vision, at first, at a distance of one foot, then, of two, three, four or five feet, each eye being examined separately; the visual angle must naturally increase in extent, the farther away the measurement is made, but a simulator, ignorant of this fact, does not allow his field of vision to increase in proportion to the distance.

The differentiation between hysterical and simulated visual field disturbances can rarely be made with absolute certainty. Hysterical *hemianopsia*[200] is very rare; concentric contraction of the field,[201] more marked in the eye on the side of the other hysterical manifestations, is the more common field defect. Very marked contraction of the visual fields giving rise to so-called "tubular" vision[202] is generally regarded as pathognomonic[203] of hysteria. In hysteria, the field may become progressively smaller and smaller as the examination continues, producing a helicoid[204] perimetric tracing.

This type of field is never observed in organic disease; it is usually due to fatigue. Although it is generally believed that neither the con-

[199] *Perimeter:* An instrument used to map out the dimensions of the field of vision.

[200] Hemianopsia (*Hemiopia*): A condition in which one half of the field of vision is obliterated in each eye. It may be *homonymous* (absence of temporal half of one field and of the nasal half of the other), due to a lesion of one optic tract or its central connections: *temporal* (absence of the temporal halves of both visual fields), due to a lesion affecting both outer sides of the optic chiasma.

[201] A narrowing of the field of vision with an almost equal restriction in all directions.

[202] See concentric contraction of the visual fields, note 205, infra.

[203] Absolutely indicative.

[204] Spiral-shaped.

centrically contracted field[205] nor the so-called "fatigue field" can be simulated, I saw a self-confessed simulator with a concentric field defect which could not be distinguished from that of hysteria.

B. *Simulation of Other Symptoms Referable to the Visual Apparatus*

A simulated *ptosis*[206] may be detected by asking the patient to look at the ceiling, and in so doing, he may raise the "ptosed" lid at the same time that he raises his head, thus betraying himself. The pseudoptosis of hysteria is generally bilateral and due to a spasm of the orbicularis palpebrarum[207] which does not diminish when the patient throws his head back. Sometimes it may be impossible to diminish a hysterical from a simulated pseudoptosis because, in both conditions, on attempting to raise the drooping lid, the examiner can feel and see a contraction or a fibrillary tremor[208] of the lid. In contrast to true ptosis, in hysterical as well as in simulated ptosis, the affected eyebrow is generally lower than the unaffected one and the secondary compensatory contraction of the frontalis muscle[209] so characteristic of true ptosis occurs neither in hysterical nor in simulated ptosis. In congenital ptosis,[210] there are occasionally observed associated movements of the levator palpebrae superioris[211] which are not under voluntary control and which can be made to appear by contraction of the jaw muscles on opening the mouth or during chewing and grinding movements as well as during lateral (side to side) movements of the jaw; the presence of these manifestations is indicative of a ptosis from birth, rather than of a recently acquired one as might be contended by a simulator.

Some individuals may acquire by practice the ability to converge both eyes so that they may easily imitate an internal *squint*. A unilateral squint, however, is much more difficult to feign. Erben[212] made the

---

[205] *Concentric contraction of the visual fields:* A narrowing of the field of vision with an almost equal restriction in all directions.

[206] A drooping of the upper eyelid.

[207] A circular muscle of the eyelid which closes the lid moderately or tightly depending on the force of the contraction of the muscle.

[208] An abnormal involuntary movement in which there occur contractions and wave-like undulations which rapidly pass from one muscle bundle to another in the same muscle, so that the entire muscle appears as if a wave passed over it.

[209] The frontal portion of the occipitofrontalis muscle whose function is to move the scalp forward, to raise the eyebrows and skin of the forehead into transverse wrinkles.

[210] Ptosis, or drooping of the upper eyelid present since birth.

[211] A muscle which raises the upper eyelid.

[212] ERBEN, DIAGNOSE DER SIMULATION NERVOSER SYMPTOME, Berlin, 3d ed., 155 (1930).

following observations in cases of simulated ocular muscle paresis:[213] (1) the allegedly affected lateral rotator is "paretic" only when the patient looks slowly and with deliberation to the side, but when he is suddenly commanded to "look to the side quickly" and he does so, the "paresis" disappears; (2) as soon as the patient is asked to "look up" from the extreme lateral position in which he holds his allegedly affected eye, both visual axes having become parallel simultaneously, the "squint" vanishes; (3) when the patient is asked to look at an object in the center of his visual field and his head is turned so that the corresponding lateral position of the eyes is established, the alleged immobility of the "affected" eye is found to have disappeared. When there occurs, during this test, an associated contraction of the muscles of the forehead, it is corroborative evidence that the internal "squint" is caused by an exaggerated voluntary contraction and not by a paresis (paralysis) of the adductor (muscles) of the eye. Although Erben claims that these diagnostic criteria are very helpful in exposing a simulated squint, their validity in all cases has not as yet been generally accepted.

Simulators frequently claim to have diplopia.[214] In genuine diplopia coming on suddenly, the double images are at first very annoying, often leading to almost automatic closure of one eye and turning of the head to one side to obviate the seeing of double images. In the diagnosis of diplopia, it must be remembered that in paralysis or paresis of the extraocular muscles,[215] the affected eye is displaced by the unopposed antagonists in a direction opposite to the direction of traction[216] of the paralyzed or paretic muscle, and that the false image, seen by the affected eye, is displaced in the direction of traction of the paralyzed or

---

[213] Weakness or paralysis of the muscle moving the eyeball producing squint.

[214] Double vision; a condition in which an object seen appears double; *monocular diplopia*, in which one eye sees objects double, is due to irregular astigmatism, early cataract, or any condition producing a double pupil; it is also observed in hysteria and in malingering; *binocular diplopia*, in which each eye sees singly, but both together see double; it is due to squint paralysis, or insufficiency of the ocular muscles, or displacement of the eyeball causing the axis of one to deviate from the object of fixation; binocular diplopia is *homonymous* when the image seen by the left eye is on the left side and vice versa, in which case the visual axes converge too greatly; *heteronymous* or *crossed diplopia* occurs when the image seen by the left eye is on the right side in which case the visual axes diverge; *vertical diplopia* is the condition in which one image stands above the other, in which case the eye corresponding to the lower image is relatively too high.

[215] The muscles moving the eyeball.

[216] Pulling.

paretic muscle. A suspected malingerer should, therefore, be subjected to the so-called red glass test[217] and his responses about the position of the alleged false images should be noted. Unless the simulator is unusually intelligent and has been "tutored" intensively, there should be no difficulty in exposing him, especially after repeated testings. The genuineness of a paralysis or paresis of an ocular muscle may occasionally be confirmed when the patient is examined after recovery has begun; in such cases, if the patient is made to look steadily in a direction necessitating active movement[218] of the weak muscle there will be observed slight involuntary oscillations of the eye ball (i.e., paretic nystagmus).

Monocular diplopia[219] or *polyopia*[220] may be encountered in patients suffering from hysteria; on closing one eye, an hysterical patient may claim to see double or multiple images with the other; this can be elicited by making the patient fix his vision on an object which is gradually being moved away from his eye; usually at a distance of about 10 to 15 cm., he will claim to see a double image and, at a greater distance, there is superadded another image, close to or above the second image.

Oppenheim[221] denies the occurrence of hysterical paralysis of the ocular muscles, especially of one individual muscle and of the iris.[222] The literature contains reports of cases which seem to contradict Oppenheim's views, but a critical review of the cases casts some doubt upon whether they were cases of uncomplicated hysteria. In the present

[217] Ordinarily the existence of diplopia can be ascertained by asking the patient to fix his vision at the examiner's finger with both eyes and to state the precise moment when he sees two fingers. If this is not satisfactory a red glass is held in front of the patient's sound eye; if the patient has diplopia he will see two fingers, one of which will appear red, and the other will be of the usual color. It must then be further determined at what part of the field of vision the diplopia appears, the position of the images and their separation from each other when the direction of the patient's gaze is altered. The image which appears in the sound eye is the "true" image, and the other, the "false." If the false image corresponds with the side of the eye by which it is seen, the diplopia is said to be a *homonymous diplopia*; in other cases, it is said to be a *crossed diplopia*. As a rule convergent strabismus (squint) is accompanied by homonymous, and divergent strabismus by crossed diplopia.

[218] Movements performed voluntarily by the patient.

[219] See note 214, supra.

[220] A condition in which several images of one object are seen simultaneously by one eye.

[221] OPPENHEIM, TEXT-BOOK OF NERVOUS DISEASES, trans. by Alexander Bruse, p. 1087 (1911).

[222] A circular muscle which contracts and dilates the pupil.

state of knowledge about the hysterical manifestations referable to the ocular muscles, it is impossible to formulate definite criteria for the positive differentiation between simulated and hysterical ocular muscle defects.

XII

SIMULATION OF DEAFNESS

Simulated deafness may be total or partial, bilateral or unilateral. Marked deafness appearing after a head trauma (injury) in the absence of definite evidence of concomitant labyrinthine [223] (vestibular) [224] involvement, should arouse suspicion about the genuineness of the deafness. Simulated deafness is most commonly unilateral. Individuals who have been deaf for years generally raise their usually monotonous voice during conversation; they also keep their eyes "glued" on the speaker's lips and face, watching for every gesture by the speaker, to make up for their inability to hear well. A deaf man who is eager to hear, automatically turns his good ear towards the speaker. Generally, vowels are heard better than consonants. A clearly enunciated whisper is more audible than loud but indistinct shouting. A "well tutored" malingerer ignores sounds and words directed towards the ear in which he claims to be deaf; he also learns when to raise his voice during the examination.

Before examining a patient for deafness, the external ear must be thoroughly inspected for the presence of impacted cerumen (ear-wax), which, if found, especially in accident cases under litigation, should be removed, preferably by the patient's physician. All hearing tests should be performed with the patient blindfolded.

Where unilateral deafness is alleged, if responses to the *Weber* [225]

---

[223] Pertaining to the labyrinth; the *internal ear* is the essential part of the organ of hearing; it consists of a cavity, the osseous labyrinth, contained within the petrous portion of the temporal bone, and encloses the *membranous labyrinth*, in which the *cochlear and vestibular* nerves end.

[224] A branch of the auditory nerve which has nothing to do with hearing; its function is to conduct impressions influencing equilibrium to the central organs, especially to the cerebellum and to the nuclei of the ocular nerves.

[225] For this test, a vibrating tuning fork is placed on the vortex of the skull (top of the head), or just above the root of the nose (glabella) or on the teeth; if the patient's deafness is due to obstruction of the sound conducting apparatus (middle ear), the fork is heard louder and longer in the affected ear; when the deafness is due to an affection of the cochlear nerve (which conveys auditory stimuli to the brain) or its terminations, the fork is heard better in the non-affected ear.

and *Rinné tests*[226] are so inconsistent that it is questionable whether the patient's pretended deafness is due to middle ear disease or to involvement of the cochlear nerve,[227] and especially if the onset of the deafness is claimed to have been sudden, the possibility of simulation must be seriously considered.

If simulation is suspected, it is advisable to plug up the patient's "good" ear and to ask him whether he can hear with that ear; an affirmative answer will prove that he hears with the allegedly deaf ear.

In another test, the "good" ear is apparently closed by the examiner's holding his extended fingers tightly against it but leaving a chink between his fingers at the auditory meatus;[228] if the patient states that he hears nothing, there can be little doubt that he is not telling the truth.

A useful test in cases in which one ear is claimed to be partially deaf is the following: the patient is asked to occlude completely the sound ear with his index finger after which, the examiner ascertains the hearing distance of the "deaf" ear; following this, a piece of india rubber tubing, with its lumen left open, is gently inserted into the allegedly partially deaf ear. An untrained simulator, believing that this ear has been entirely occluded, will betray himself by claiming that "now he hears nothing."

A naïve simulator may be caught by some such simple ruse as the following: the examiner expresses his sympathy to the patient for the plight he is in and distracts his attention by counting his pulse or listening to his heart or examining his eyes, when suddenly, but in a low voice, he asks him to protrude his tongue or raise a limb. Compliance with the request will generally leave little doubt that hearing is unimpaired.

A simple test to detect unilateral deafness is *Lombard's test*,[229] in

[226] Normally a vibrating tuning fork is heard longer and better when held in front of the ear than on the mastoid process; if it is heard better in front of the ear, the test is said to be positive; in the opposite event, the test is negative.

[227] Cochlear-vestibular apparatus: This term is employed to designate the anatomo-physiologic mechanism subserving hearing (cochlear) and the maintenance of equilibrium (vestibular).

[228] The passage to the ear comprising the external meatus which leads to the ear canal.

[229] This test is used to detect unilateral deafness. The patient is asked to read aloud a selected passage; as long as he hears his own voice, there is no change in pitch; the Bárnáy noise apparatus is then inserted into the sound ear, while the patient continues reading; if he is actually deaf in the other ear, he will raise his voice; if he does not raise his voice, he probably hears with the allegedly deaf ear.

which the patient is asked to read aloud a selected passage; as long as he hears his own voice, there is no change in pitch; the Bárany noise apparatus is then inserted into the sound ear, while the patient continues reading; if he is actually deaf in the other ear, he will raise his voice; if he does not raise his voice, he probably hears with the allegedly deaf ear.

Another simple test for the detection of feigned deafness is the so-called *"double conversation test."* [230] This test is performed by two examiners; simultaneously, each of them speaks to the patient through a tube leading to each ear; in unilateral deafness, the patient will ignore the conversation directed to the deaf ear. A "trained" malingerer, however, may have learned to ignore the conversation directed toward the allegedly deaf ear.

A useful test for simulated deafness is the *Bloch-Stenger test,* [231] which is based on the physiological principle that normally, when two vibrating tuning forks of different pitch are held simultaneously and equidistantly in front of both ears, only the one of higher pitch is audible. If, therefore, an individual claims not to hear, say on the left side, a vibrating low-pitched fork in front of his right ear will be heard on the right side; without removing this tuning fork, another fork of higher pitch is made to vibrate simultaneously and equidistantly in front of the left ear; if the patient is either totally or markedly deaf in that ear, he perceives no change; but if he hears with the left ear, the moment the higher-pitched fork begins to vibrate in front of that ear, the sound of the low-pitched fork on the right will not be heard, and the patient simulating deafness on the left side will probably say that now he does not hear at all. Simulation of deafness is detectable by this method only when the hearing in the allegedly deaf ear is normal; should the hearing in that ear be more or less defective, the test will be of no value.

The so-called *stethoscope test* may sometimes be employed successfully in detecting feigned unilateral deafness. An ordinary stethoscope [232] having one tube closed with a wooden or rubber plug is ad-

---

[230] This is a test utilized in detecting simulated deafness. It is performed as follows: simultaneously, each of two examiners speaks to the patient through a tube leading to each ear; in unilateral deafness, the patient ignores the conversation directed to the deaf ear. A "trained" simulator, however, may have learned to ignore the conversation directed to the allegedly deaf ear.

[231] Useful only to determine whether unilateral deafness is simulated.

[232] An instrument of various form, size, and material, through which sounds are conveyed to the ear of the examining physician. By this means, breath sounds, heart sounds, etc. are made more audible.

justed to the patient's ears with the open tube to the allegedly deaf ear
and the closed one to the "good" ear. The examiner speaks into the
bell of the stethoscope and asks the patient to repeat what he hears;
the stethoscope is then removed, the "good" ear is tightly closed, and
the same procedure repeated; the patient will say that he cannot hear,
whereas he has already repeated after the examiner, when the "good"
ear was tightly closed with the plugged tube of the stethoscope. Of
course, if the simulator has "learned" to ignore all sounds going to the
allegedly deaf ear, the test will not be successful.

Valuable information about the presence of simulated deafness may
be obtained from the *Veraguth psychogalvanic reflex test*[233] and from
other similar tests, as well as from repeated testings with the audio-
meter.[234] For detailed information as to these tests, the reader is re-
ferred to special text-books on otology.[235]

In differentiating simulated from hysterical deafness, the following
criteria may be helpful: Hysterical deafness is usually a transitory
symptom appearing suddenly after some unpleasant emotional experi-
ence; it is frequently associated with hysterical anosmia,[236] blindness,
and mutism. There are reported cases of hysteria in which, in the pres-
ence of total bilateral deafness, musical perception remained unaffected,
and F. Schultze, cited by Oppenheim,[237] saw a boy with hysterical deaf-
ness who continued a melody whistled near him. Unilateral hysterical
deafness is usually partial and on the same side as the side on which
the hysterical motor and sensory disturbances are found.

Feigned as well as hysterical deafness following trauma (injury) is
notoriously observed in individuals whose physical injury has been
minimal.

None of these criteria including the results of the various tests that
have been described are infallible in distinguishing hysterical from
simulated deafness. The tests, by themselves, are of value only in un-
tutored persons who had not previously been subjected to extensive
otologic investigations. There are altogether too many cases encount-
ered in practise in which, after the most thorough investigation and even
the taking of all surrounding circumstances into consideration, one can

[233] Increased electrical resistance of the body to the galvanic current as a result
of mental or emotional agitation.

[234] An instrument for testing the acuity of hearing.

[235] The study of the ear and the diagnosis and treatment of its diseases.

[236] Loss of the sense of smell.

[237] OPPENHEIM, TEXT-BOOK OF NERVOUS DISEASES, trans. by Alexander Bruse,
p. 1069 (1911).

never be absolutely certain whether he is dealing with an hysterical or with a simulated deafness.

XIII

SIMULATION OF ABNORMAL MENTAL STATES

Simulation of a mental disease is usually practised by: (1) criminals to escape punishment, especially murderers when they realize that they may be subjected to capital punishment; (2) persons desiring to be declared incompetent in order to repudiate contracts which they regret having made; (3) persons desiring to evade military duty and (4) sensation-seeking journalists, in order to gain admission to an institution for mental disease to investigate alleged abuses and mismanagement by the authorities.

Here and there one encounters an individual who feigns sound mental health when, as a matter of fact, he knows that he is suffering from mental disease; this is known as *dissimulation*. Chronic alcoholics and paranoiacs [238] occasionally practise dissimulation to obtain release from a mental hospital or to remove their previously adjudicated incompetence. Mental disease is also occasionally denied by patients suffering from a depressed state with suicidal tendencies; some of these patients may be very skilful in concealing their condition, proclaiming that they are better and actually appearing more cheerful in order to achieve a relaxation of the vigilance of those supposed to guard against their suicide. The writer has seen many habitual criminals who knowingly concealed their mental symptoms because they preferred conviction and a definite prison sentence for the crimes with which they were charged to being adjudged mentally incompetent and committed for an indeterminate period to a hospital for "insane criminals."

Occasionally, a patient suffering from one of the epilepsies, especially grand mal,[239] may deliberately deny his affliction either because of the prevailing popular objection to the marriage of an epileptic on eugenic grounds or because of the unwarranted hardships a known

[238] Individuals suffering from paranoia which is an abnormal mental state characterized by the presence of systematized and often connectedly elaborated delusions, particularly ideas of reference, persecution or grandeur and hallucinations in various fields. Although many paranoiacs appear normal and intelligent and may not be recognized as being psychotic, yet they may be the most dangerous of all psychotics. Among them are found individuals who are great schemers, inventors, artists and literateurs.

[239] See note 63, supra.

epileptic experiences obtaining gainful employment in some occupations.

Contrary to the general belief, simulation of mental disease is not common. The popular notion about mental disease is so different from what it actually is that an experienced psychiatrist, with the opportunity to observe the patient for a reasonable period, should have little difficulty in detecting simulation. An intelligent and cunning patient who has had occasion to observe the mentally ill, however, may imitate the manifestations of mental disease so adroitly as to defy detection for a long time.

Detection of simulation may present a difficult problem in a mentally ill individual who feigns a mental disturbance different in nature from the one from which he is actually suffering; this usually occurs in connection with criminals who have been, for some time, "border-line" cases able to control their conduct until the actual commission of the crime.

Simulation should be suspected in an individual who has an advantage in being regarded as mentally abnormal and who claims an utter lack of school knowledge and uniformly gives ridiculous answers to questions when in other respects, his behavior is not unusual; in such cases, however, it is important to bear in mind the possibility of the presence of a so-called Ganser syndrome,[240] in which the patient's every word and action is the opposite of what it should be.

It is not unusual for a simulator who feigns mental disease following a head injury to be "dumb," "not to know anything," "not to remember anything"; he makes every effort to appear dull and listless, answering all questions with a stereotyped "I don't remember: I don't know." It is remarkable how much ideational[241] productivity can be elicited from such an allegedly "stupid" individual following an intravenous injection of 3 grains of sodium amytal.[242]

Except in the very intelligent, the successful imitation of a definite clinical picture of a psychosis[243] is usually rare. To construct a non-con-

---

[240] This is regarded as an hysterical psychosis which consists of hysterical twilight states in which the patient appears dull, stupid, confused and indifferent to his surroundings; his answers to simplest questions are absurd and usually opposite to what they should be; it is usually met with in prisoners and generally clears up after they have been liberated from prison.

[241] Ideation: the formation of thoughts.

[242] See note 93, supra.

[243] A psychosis is an abnormal mental state characterized by a profound disturbance of the personality functions of the individual manifested by alterations in the mode of

tradictory clinical picture of mental disease requires skill and special knowledge which most simulators do not possess. But the simulation of one or more symptoms of mental disease, usually of a profound dementia[244] or of a manic state[245] or of a delirium-like reaction,[246] is much more common.

An "untutored" simulator can be readily unmasked because in genuine mental disease, certain symptoms exclude others, e.g., a quiet patient who is not suffering from a disturbance of consciousness and attention cannot be disoriented[247] for a long period; the total absence of most elementary knowledge can be associated only with imbecility[248] or with profound disturbances of memory. With a marked inconsistency and peculiarity of the clinical picture, therefore, unless there is some other valid reason for its peculiarity, the probability of its genuineness may be doubted.

The average simulator believes that all mentally ill patients are either stark raving mad and incoherent at all times or in a condition of imbecility. Unless "well tutored," a simulator is not apt to know that the excitement of a manic[249] or paretic[250] must have been preceded by other abnormal mental reactions. Nor is he apt to know that the excitement of a febrile delirium is associated with somatic disturbances[251]

---

thinking, feeling and acting and a blunting or loss of the sense of reality which makes satisfactory social adjustment very difficult, and in severe cases impossible.

[244] A defect or loss of the intellectual processes, memory and will power, acquired later in life in a person previously intelligent.

[245] In the manic depressive states there occur alternating periods of excitement (*mania*) and depression with usually, but not always, a comparatively normal period (*lucid interval*) between the two. The duration and severity of the attacks, and the duration of the relatively normal period are varying. During the manic stage there is a feeling of happiness and unusual well being with overactivity in speech, thought (*flight of ideas*) and action; during the depressed stage the patient is sad, has difficulty in thinking and acting, and is generally retarded.

[246] Mental excitement with confusion, rambling and incoherent speech. (Febrile delirium is a delirium caused by fever.)

[247] Disorientation: A disturbance in the correct appreciation of the environment and realization of the patient's relation with reference to time, place and person, singly or together.

[248] See note 78, supra.

[249] See note 245, supra.

[250] A patient suffering from *general paresis* (*general paralysis of the insane, dementia paralytica*) because of advanced syphilitic infection of the brain (see locomotor ataxia).

The adjective paretic is also used to designate weakness short of complete paralysis —a paretic muscle, or a paretic limb, is a weak muscle or a weak limb.

[251] Disturbances in the physical components of the body.

and, in severe cases, with clouding of consciousness, disorientation[252] and often with terrifying hallucinations[253] and distressing delusions,[254] none of which can be successfully imitated.[255] The feigning of an acute confusional state alone may offer some chance of success because of the lack of associated physical phenomena, because of the greater ease of imitating the incoherence of this disorder and because of the absence of that deep emotional state so commonly observed in manic-depressive states. If a confused or mildly excited patient stops to answer a question, which he usually does, he answers it with some degree of responsiveness; a simulator either does not answer questions at all or gives most absurd answers. As a rule, a simulator during the lucid periods of a pretended manic state does not recognize his friends or his surroundings or recollect anything that happened in the period for which he has a motive to make people believe that he was irresponsible. The true manic, during such lucid periods, recognizes his family and friends and, if he has committed a crime, while he may be sharp enough to desire to conceal his memory of it, he will not, if the examination is led up to the period of the commission of the crime, claim to have forgotten all circumstances before and after it, as the simulator, being always on his guard, does. Such profound amnesia[256] occurs only in the epilepsies, hysteria, severe intoxications and the dementias of organic brain disease.

The epileptic patient expresses concern, chagrin and bewilderment over a past period of amnesia; the hysteric seems little perturbed by the fact that all his memory is gone; the simulator is apparently totally disinterested in the discussion of the subject. In cases of feigned amnesia, the identifying features of amnesia of organic brain disease and of the psychoneuroses are absent. Recent electroencephalographic investigations[257] of patients suffering from various forms of amnesia would seem to indicate that a normal electroencephalogram (brain wave tracing) is presumptive evidence against a plea of epileptic amnesia.[258]

[252] See note 247, supra.

[253] A perception without there being anything to perceive; it is usually *auditory*, or *visual*, but may include any of the senses.

[254] A delusion is a false belief which cannot be disproved to the patient; as a rule delusions are not true as to facts; they are highly improbable, even manifestly impossible and often bizarre; they cannot be corrected by an appeal to reason and are out of harmony with the individual's education and surroundings.

[255] The attention of the reader is invited to the following paper in this Symposium series: Coon, "The Common Psychoses."—*Ed.*

[256] See note 79, supra.

[257] The reader's attention is invited to the following paper in this Symposium series: Gibbs, "Medicolegal Aspects of Electroencephalogram."—*Ed.*

[258] See note 79, supra.

A simulator gives himself away by allowing his feigned mental disorder to appear suddenly and to recede as suddenly. Another characteristic feature of simulation is the exaggeration of symptoms while under examination. A simulator generally becames more incoherent,[259] demented,[260] excited, depressed or catatonic[261] as soon as the physician or nurse approaches in order to bring his symptoms to their notice. A patient suffering from a genuine mental disorder, unless he is profoundly demented, will try to show a mental capacity which he does not possess and to appear better than he is, whereas a simulator will do all in his power to demonstrate to those around him that he is mentally ill. Not infrequently, it may also be possible by a hoax to suggest to a simulator certain symptoms, such as complete failure to perceive sensory stimuli (pinprick, hot and cold, touch, vibration), paralysis, fainting spells, etc.

The simulator also labors under the mistaken idea that all mentally ill patients do not reason at all when, as a matter of fact, too great a degree of incoherence in a delusion may be a justification for questioning the genuineness of the delusion. Even imbeciles[262] have some ideas within their limited range of knowledge and expression and adhere to

[259] Disconnected; having no proper connection or mutual relation.

[260] See note 244, supra.

[261] Catatonia is a form of dementia precox (schizophrenia) in which there occurs an alternation of stupor (catatonic stupor) and excitement (catatonic excitement). In catatonic stupor the principal symptoms are: stupor, negativism and muscular tension; in extreme cases the patient is perfectly quiet without making any movement whatever and without reacting to any external stimuli; he pays no attention to questions and remains absolutely mute (negativism); he not only refuses to eat but pays no attention to calls of nature, allows the saliva to accumulate in the mouth, and any effort to get the patient to do anything may be met by a response diametrically opposite to the desired act; an attempt to move the body is met by marked resistance and elicits a muscular tension in which the limbs are held rigidly; there also occurs bizarre facial grimacing which may be associated with a constant expression of scowling, or keeping the eyes tightly shut, the cheeks puffed out and the lips closed and protruded; in some cases instead of muscular tension there occurs a condition of remarkable flexibility of the body musculature and, instead of negativism, there is present suggestibility or command *automatism* in which the patients do mechanically just what they are told to do; this heightened suggestibility may be in the form of *echolalia* (a repetition of words and phrases spoken to him) and *echopraxia* (a repetition of movements).

Catatonic stupor may alternate with catatonic excitement in which the patient is constantly talking, shouting, gesticulating and throwing himself around as is usually observed in manic excitement, except that in the former the reactions are much more absurd and incoherent and are frequently interrupted by attitudinizing and stereotyped movements, constant repetition of movements, verbigeration with nonsensical rhyming of words, perseveration of speech and impulsiveness of acts.

[262] See note 248, supra.

them with a certain degree of consistency and can reason to some extent concerning them; whatever the incoherence in their ideation [263] may be, it is the result of confusion and digression of ideas because of defective attention and memory. The simulator of imbecility either talks more confusedly than harmonizes with the ideational flow he unwarily exhibits or he talks less confusedly than he should because of the apparent disconnectedness of his thoughts.

The cases in which, after prolonged observation, the simulation cannot be detected with reasonable certainty are very rare. A simulator's task is arduous when he is under continuous observation. The most ingenious imitator may find it extremely difficult to adhere to his assumed character for days and nights in succession without showing some evidence that some of his performances are voluntary.

Suspicion of simulation of mental disease should be aroused when (1) on the physician's entrance to the ward or prison cell or wherever the patient may be, the patient avoids looking at him and gazes at the ceiling or walls; (2) the patient's answers to the simplest questions are absurd; (3) there is unusual slowness and uncertainty in the patient's answers; although delay in answering questions is common in depressed patients, their appearance and facial expression generally harmonizes with inhibition of thought and action; (4) the patient, supposing himself watched, makes furtive glances to see whether any one is approaching; (5) the patient performs senseless rhythmical shaking movements of parts of his body or of the entire body in the presence of those who are watching him; (6) he claims to have delusions and hallucinations and uses those terms; one who actually has delusions or hallucinations will describe them without naming them; he will usually say that he is "lost," that he is "being followed by the devil, the police or his enemies" and that he "hears voices and sees faces" or feels "queer in the head"; (7) the mental symptoms appear immediately after the commission of a crime or after arrest, indictment, conviction or sentence, and there is no reliable evidence of a previous mental disturbance; (8) there is no evidence, in one simulating an acute psychosis, of insomnia, digestive disturbances, loss of weight and of general ill health.

Some of the following ruses may help to detect a simulator; (1) during the examination of the patient the examiner remarks in an undertone to a bystander or his assistant that if such and such a sign or symptom were present, he would know how to classify the patient's "insanity" or would know into which ward to put him and what type of

[263] See note 241, supra.

treatment to administer; a naïve simulator may betray himself by exhibiting the sign or symptom suggested at this or at some future examination; (2) when a simulator is accused of shamming, he may turn away from the examiner or suddenly lapse into a pretended semistupor or he may change his entire symptomatology; a genuinely psychotic patient, when accused of shamming, will usually show no changes in his behavior.

A suspected simulator should not be charged with simulation point-blank until all other means of detection have been exhausted. A "poorly trained" simulator transferred from one ward to another will imitate the patients he sees there; he may appear depressed or demented one week, excited and destructive the next, or claim to have delusions or hallucinations which he heard the various patients in the wards express; when placed in a ward in which some patients are suffering from the convulsive state, he may even go through the performance of feigning a "fit."

Various medicinal and physical agents have been suggested and employed for the purpose of detecting simulation of mental disease, but they are of little value. Perhaps the most reliable methods are the induction of general anesthesia or of mild convulsions, preferably by electric shocks. Occasionally, an individual simulating stupor may be exposed following the application of a strong faradic current. The frequent occurrence of temporary improvement and other changes in the symptomatology of the genuine psychoses and psycho neuroses[264] following some of these procedures should be borne in mind.

Useful diagnostic information may also be obtained by studying in these patients the results of psychometric tests,[265] especially the Rorschach[266] and the Kohs block-test.[267]

In the diagnosis of simulation of mental disease, it must constantly be kept in mind that occasionally a depressed patient may claim that, at

[264] See note 1, supra.

[265] Intelligence tests used to determine the intelligence quotient and/or the mental age.

See, also, in this symposium series, the following study: Hunt, "Use and Abuse of Psychometric Examinations."

[266] By means of this test are measured the higher mental processes and personality trends; a Rorschach record contains the patient's verbal and other observed reactions made during his interpretation of ten ink blots, five of which are colored.

[267] A performance test in which the patient is given a design and requested to reproduce it with a specified number of blocks (four or five). The test was designed by Kohs to determine mental age on the basis of the number of errors and the total time consumed in the performance of the test.

the onset of the disease, he knowingly exaggerated his condition. Also, that catatonic[268] negativism may, by its instinctive absurdity, give the impression that it is simulated, especially when there appears to be some reason for simulation.

There is general agreement among psychiatrists that simulation is, in itself, a type of reaction indicative of a defective personality. The number of "mentally healthy" individuals among simulators is very small, even though the actual mental disorder from which they are suffering may be entirely different from the one simulated. It is not unusual to find, among simulators of mental disease, individuals who, after prolonged observation, are found to be psychopaths[269] or suffering from schizophrenia,[270] feeble-mindedness, psychoneurosis[271] or other abnormal mental states. Hysterics, especially, may present clinical pictures in which it is impossible to separate the symptoms of hysteria from those of voluntary and deliberate simulation.

In diagnosing simulation, the individual as a whole, his family and previous history, habits, psychosexual life,[272] occupational, educational and social background and his surroundings and motives for simulation must be considered. Reliable information must be obtained about the suspect's previous character and whether there has occurred a change in personality and, if so, when it was first noted. If simulation is suspected in an individual charged with the commission of a crime, it is necessary to ascertain a motive for it as well as its precise nature, the manner in which the crime was committed as well as the individual's behavior prior to, during and after its commission. There are some

[268] See note 261, supra.

[269] A psychopathic personality is an abnormal personality, with or without psychosis, associated with emotional instability, defective will power, and a poorly developed ethical background and low character traits. Psychopathic individuals, as a rule, have no intellectual impairment but their judgment is poor; they are frequently antisocial; a number of them are chronic alcoholics, drug addicts and sexual perverts.

[270] A psychosis essentially of the period of puberty and adolescence, characterized by a dementia which tends to progress, though frequently interrupted by remissions. It includes the *paranoid, hebephrenic, catatonic, simple,* and *mixed* forms. Patients suffering from schizophrenia comprise by far the largest number of admissions to hospitals for mental disease. Some of the outstanding symptoms of schizophrenia are: marked *introversion* (withdrawal of the patient's interests from the outside world as objectively conceived), *narcissism* (love of one's self), emotional flattening, negativism, stubbornness, eroticism (sexual excitement on a physical, mental or emotional basis), delusions and hallucinations, all of which gradually leads to mental deterioration.

[271] See note 1, supra.

[272] Psychosexuality: The interrelationships and the various aspects of love and pleasure seeking.

crimes which, by themselves, suggest that their perpetrators were suffering from a mental disorder. It is not fair to assume that because the crime committed involved deliberation, premeditation and design it could not be the act of a mentally ill person. Such an individual may reason correctly from false premises. The simulator who commits a crime with skill and careful preparation betrays his feigning mental disease by claiming complete amnesia or saying that he must have lost his head or by showing a desire to appear feeble-minded, presenting, at the same time, a symptomatology of an abnormal mental state which is not consistent with any of the well established clinical entities of mental disease.