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# Editorial

*Es para nosotros un gran honor y un placer el anunciar que el Prof. Charles D. Aring de Cincinnati Ohio, neurólogo de fama mundial, como científico y humanista, quien posee una extensa experiencia en publicaciones médicas, integrará el brillante grupo de especialistas que honran el Comité Consultivo de la Revista Internacional de Neurología.*

*El Prof. Aring significará una fuerza propulsora en nuestras aspiraciones de perfectibilidad, y su estimulante colaboración será de sumo beneficio para nuestros lectores.*

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*El problema de la amnesia merece actualmente interés muy especial por el significado que tiene para su estudio en la clínica neuropsiquiátrica y por los aportes importantes de la investigación científica básica vinculada a una de las funciones más trascendentes que tiene el sistema nervioso que es la memoria y cuya falta constituye la amnesia. Los avances actuales en su conocimiento en cuanto a sus fundamentos, han adquirido caracteres subyugantes.*

*Los psicólogos nos han brindado desde hace tiempo estudios valiosos acerca de la memoria, particularmente en lo relacionado con su mejor aprovechamiento, sobre todo en lo concerniente al aprendizaje.*

*Se sabe cuanto tienen de importancia para la misma otros aspectos de la actividad psíquica, como son el interés y la atención a lo que se procura que sea recordado, el estado emocional con que se recibe el dato a archivar en el recuerdo, el conocimiento previo que se tenga del tema, la repetición, elemento básico para determinar la fijación de los datos que se desea recordar, etc.*

*Todo ha servido para que se configuraran orientaciones acerca de la mejor técnica de estudiar y aprender.*

*La neurofisiología y la química en cuanto a la memoria vienen aportando adquisiciones de interés singular.*

*Comienzan asignando cierta topografía en el cerebro a dicha función; es así que al vincularse algunos tipos de crisis epilépticas, que significan la aparición en la conciencia de datos que estaban en reserva en el cerebro, con el lóbulo temporal, se piensa que en la función de la memoria intervienen circuitos que están instalados en este lóbulo.*

*Los progresos en el estudio del sistema límbico atribuyen a éste gran influencia sobre la función de recordar y se considera que uno de los aspectos fisiológicos más importantes le concierne con la memoria.*

*En lo anatómico, pero microscópico y con el progreso técnico de la microscopía electrónica se presta atención a la unidad neuronal en su sector sináptico. La memoria implicaría el asiento del fenómeno biológico que determina el influjo nervioso que habrá de retenerse en un circuito que se admite muy complejo.*

*Si pensamos en la fabulosa cantidad de recuerdos que se almacenan en el curso de una vida, si imaginamos que un cierto y complicado circuito nervioso*

debe soportar cada uno de ellos, hay que suponer que para que esto se dé anatómicamente deben actuar como elementos de conexión interneuronal no sólo las terminaciones nerviosas sino también las espinas de los cilindroesjes (gémulas de Cajal) dispuestas en número variado y abundante como pequeños brotes en cada cilindroesje; lo que aumentaría astronómicamente las posibilidades de nuevos contactos. Se piensa que con el aprendizaje se modifique el número de espinas en cada cilindroesje y que quizás también en toda ocasión en que debe almacenarse un recuerdo se modifiquen contactos sinápticos y que en esto participen también las espinas.

Otro concepto que ha sido expresado es que en un recuerdo la intervención neuronal es múltiple y numerosa y que cada unidad neuronal debe participar en múltiples circuitos correspondientes a múltiples recuerdos.

Se admite que se producen algunos fenómenos en las neuronas cuando se gesta un recuerdo, ellos son: cambios en la eficacia sináptica que duran alrededor de una hora; cambios en la actividad marcapaso, influencias tróficas más permanentes y cambios posibles en la capacidad de la neurona de generar espinas.

La bioquímica le da materialización al fenómeno de la memoria, es decir en el recuerdo tienen vigencia determinadas sustancias. El ARN constituye actualmente un motivo de atención por los hallazgos experimentales, sea cuando se hacen estimulaciones con corrientes de alta frecuencia o en el foco epiléptico espejo, situaciones que se suponen parecidas a la de la memoria, o cuando se instruye a ratas a resolver problemas de laberinto; se observa que la concentración de ARN es significativa, aumenta con las estimulaciones de alta frecuencia en el foco epiléptico espejo y cuando a la rata se le merma, tiene más dificultades en aprender.

Además otros elementos químicos han recibido atención por su posible influencia sobre la memoria, unos para estimularla en dosis adecuada (estricnina, escopolamina, anfetamina, tricienoaminopropeno, potasio, etc.), otros para actuar como agentes que la bloquean (puromicina, cicloheximidas, etc.).

Agradecemos la colaboración de los autores Dres. Jacques Barbizet, Georges A. Talland, Serge Brion, Elizabeth Cany, W. Ritchie Russell y R. Diatkine que han contribuido al éxito de este nuevo número del "International Journal of Neurology".

VICTOR SORIANO.



# Editorial

*It is for us a great honour and a pleasure to announce that Prof. Charles D. Aring of Cincinnati Ohio, neurologist of world wide reputation, as a scientist, humanist, and endowed with a large experience in medical publication will integrate the outstanding group of physicians that enhance the Advisory Board of the International Journal of Neurology.*

*Prof. Aring will be a propeling force in our endeavor, and his stimulating collaboration will surely benefit our readers.*

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*The problem of amnesia actually deserves much interest particularly due to its significance in neuropsychiatric clinic and also because of the important contributions of basic scientific research related to one of the most important functions of the nervous system, memory, and whose absence constitutes amnesia. Recent advances in the knowledge concerning the fundamental principles of memory have acquired subjugating features.*

*Psychologists have offered since long ago valuable studies about memory, particularly concerning everything related to the better profit of this faculty for learning.*

*It is also known that other aspects of psychic activity are of chief importance for memory such as interest and attention to what is tried to remember, the emotional state in which the data to be kept are received, the previous knowledge of the subject, repetition, basic element for the fixation of the elements one wishes to remember, etc.*

*All this has lead to the formation of trends concerning the better technique of studying and learning.*

*Neurophysiology and chemistry have given contributions of outstanding interest.*

*They begin ascribing a certain topography in the brain to this function. By relating certain kinds of epileptic crises which mean the appearance in consciousness of data stored in the brain, with the temporal lobe, it is believed that memory function is under the action of circuits localized in this lobe.*

*Progress in the study of the Limbic System evidences its great influence on memory and it is considered that one of the most significant physiological aspects concern this function.*

*In the anatomic aspect but on the microscopic level and thanks to technical progress in electronic microscopy attention is paid to the neuronal unity in its synaptic sector. Memory would imply the seat of a biological phenomenon determining the nervous impulse, that will be retained, in a circuit believed to be most complex.*

*If we think of the enormous amount of remembrances stored in the course of life, if we imagine that a certain and complex nervous circuit has to support each one of them, we have to suppose that in order that this happens anatomo-*

*mically the participating elements of interneuronal connection are not only nervous endings but also the spines of the cilinderaxes (gemulae of Cajal) disposed in a varied and abundant quantity like a little protuberance of each cilinderaxe, which would increase astronomically the possibilities of new contacts. It is believed that perhaps with learning the number of spines of each cilinderaxe is modified, perhaps also each time a remembrance has to be stored synaptic contacts may be modified with participation of these spines.*

*Another concept stated is that in each remembrance the neuronal participation is manifold and abundant and that each neuronal unity has to take part in manifold circuits corresponding to manifold remembrances.*

*Some neuronal changes probably are involved in information storage, they are: changes in the synaptic efficiency during around an hour, changes in the pacemaker activity, more permanent trophic influences, possible changes in the ability of the neuron to generate spikes.*

*Biochemistry materializes the phenomenon of memory, this means that in remembering there is an action of certain substances. RNA is the object of main attention for experimental findings when stimulations with currents of high frequency are performed or in the mirror epileptic focus; these situations are supposed to be similar to memory. The action of RNA is also studied in rats instructed to solve maze problems; it is observed that the concentration of RNA is significant, that it increases in the mirror epileptic focus and when it is reduced, rats evidence more difficulty in learning.*

*Besides, other chemical elements deserved careful studies because of their possible influence on memory, some of them as stimulants in adequate doses (strychnine, scopolamine, amphetamine, trycianoaminopropene, potassium etc.), others as blocking agents (puromycin, ciclobeximides).*

*We are indebted to the authors Drs. Jacques Barbizet, Georges A. Talland, Serge Brion, Elizabeth Cany, W. Ritchie Russell and R. Diatkine who have cooperated to the success of this new issue of the "International Journal of Neurology".*

VICTOR SORIANO.



# Physiopathology of Amnesias

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The successive experiences of an individual and also the way in which he associates them one to another are specific and characteristic of the individual. This data is fixed in the brain on neuronal structures according to various modes of engrammation that are no doubt extremely precise and which, taken all together, constitute a system which for a given subject at a given moment of his existence represents not only the basis of his aptitudes and knowledge, but also that of his motivations and consequently the whole of his personality. One can never sufficiently emphasize the relative fixity of character of an adult: his vocabulary, expressions, and prosody of language are his own; his gestures, awkwardness, and talent belong to him alone; his food habits, tastes and distastes do not vary to any extent and his deeper motivations may be found unchanged, masked behind reasonings or modes of behaviour that are to all appearances opposed. The mechanisms of entry as represented by the sensory systems, the diversity of the effector systems and especially the extensive association network, allow for a luxuriance of performance which should not disguise the fact that there exist limits to the possibilities of the system. The pathology of the brain by providing simplifications of the latter enables a better understanding of the existence of the neuronal circuits that are marked by learning, each stage of our experience being recorded and remaining engrammed in the region of the

brain where it serves as reference for later acquisitions.

The partial or total loss of experiences or knowledge is always, quite rightly, felt to be a failing by the subject or his entourage. The term amnesia covers numerous aspects of this unlearning, and for this reason rather than limiting it to "loss of memory" it is preferable to extend it in a more general sense and define it as an alteration of the previous engrammed experiences associated with a modification in the possibilities of using them and of making new acquisitions.

The various clinical types of amnesia are extremely numerous but it is nowadays possible to deal with them according to certain physiopathological criteria which means to say that with regard to an amnesia there are different points in question: Is the amnesia of an affective nature due to a pathological organization of a memory system functioning normally? Or is it a case of organic amnesia due to a disorder of the complex system that ensures the conservation and utilization of knowledge?

In order to approach the different varieties of amnesia while bearing this point of view in mind, we shall outline in a first section what is already known about the functioning of normal memory, without losing sight of the fact that it is the careful and systematic study of an extremely large number of patients suffering from organic disorders of the higher functions of the brain which has largely con-

tributed to the elaboration of the ideas we are about to formulate.

### 1) In what form is our experience and knowledge recorded in the brain?

The problem is to know how, among the thousands of stimulations which ceaselessly strike our sensory organs, our learning enables some of them to become individualized, and assume a signification and to subsequently release a form of behaviour.

Neither neurophysiology nor histological techniques, despite the extensive progress made over the past ten years, provide the least explanation as to the way in which knowledge is engrammed in the brain. One or other describe the circuits or networks involved by different series of nervous influxes, the topography and mode of propagation of which has become known in more and more detail, but it does not provide information as to the manner in which, depending on certain circumstances, a network of this type could or could not be the support of this experience.

Therefore how can one find out or identify the support of knowledge? Certainly it is already known that, taken by and large the latter depends on an immense neuronal combination but is one able to distinguish each fragment of experience, each "unit of knowledge"?

It is perhaps in terms of an individual own personal history, and the way in which his experiences have been built up that one is able to discover, while still retaining the notion of successive stages in learning, a type of sub-division which would provide us with a useful approach in an attempt to distinguish significant neuronal sequences.

Are not the latter functionally evident when they become the support for certain behaviour? It is probably the constraint of the outside world and society which governs successive conditionings, isolating by experience and reinforcing by repetition certain circuits, which, depending on

circumstances, serve as support to a great variety of activities. Hence there seems to be a connection between an experience that has been lived and a neuronal system which functions during this experience which establishes a similar type of coding between one and the other. Acquired during successive experiences each one of these series—which we shall call a meta-circuit—possesses its own topography and is probably the support both of perception and of the corresponding reaction to a lived experience. The repetition which ensures its perennality, otherwise it fades away and is forgotten. But just as long as it retains a functional existence this meta-circuit will be the cerebral representation of a certain experience that has been lived. Each meta-circuit thus appears to be the unit of human knowledge and the necessary and specific support of each item of our experiences.

This notion of a meta-circuit—and here we are only dealing with a hypothesis—calls for a number of specifications which we can only outline here.

From the structural point of view one could consider that each meta-circuit probably consists of a very large number of neurons of the cortex and central nuclei of the grey matter that correspond exactly with the neurons that are brought into play when a situation is experienced, the meta-circuit being the cerebral reflection of the latter. Any change in this situation causing a change in perception, conscience or in reaction would then have as material support the functioning of another neuron group differing from the previous one and specific of the new situation.

Although specific in function, a meta-circuit never exists as an isolated condition, it establishes numerous functional connections with others. These contacts, or junction points are acquired during the establishment of new circuits. In fact, when an individual lives a new situation, he always compares it to one or several previous situations he has lived through and characterizes this new situation in terms of his previous knowledge.

Consequently, any increase in knowledge that we may have of an object or of a situation is expressed by an increase in the underlying meta-circuits. The more a situation, or human being or object is of importance to any one individual, the better he knows them. This psychological fact is reflected from the neuron point of view by the establishing of a very large number of circuits, each one specific of one aspect of this knowledge.

Hence, besides the anatomical organization of these cerebral neurons according to which the cells having the same function (motor, sensory, visual, auditory, vegetative etc.) are grouped together in certain specific areas of the brain, a functional organization seems to exist which is superimposed on the latter, and according to which these same cells form part of the extremely numerous networks of the meta-circuits. The anatomical organization is of a genetic nature and characteristic of the species; the functional organization is acquired and characteristic of each individual.

From the point of view of *dynamics* one might suppose that when a situation recurs in an identical manner, the meta-circuit is activated in the same way and thus reinforced. The fact that a situation which has already been experienced induces the functioning of an already existing meta-circuit explains the recognition of this situation. In fact in this sense, *recognition* is nothing but the renewed display of functioning of the reactivated meta-circuit. It is indeed of capital importance to realize that with regard to meta-circuits, information does not "circulate" but is "displayed", and every passage of nervous influx determines the functioning of each of the constituent neurons of the meta-circuit the (sensory, cognitive, affective or motor) expression of which is displayed, one after the other, in a rapid chain reaction the whole of which constitutes the exact state of consciousness related to the situations that are being experienced.

But in many cases this reactivation occurs in the absence of the situation that

created it: i. e. recollection. Here it is no longer a question of specific stimulations issuing directly from the situation that will act but a series of non-specific influxes which, on reactivation, enable the display of previously engrammed meta-circuits. This display does not occur by chance but is more often than not induced by an external stimulus causing, by contiguity, a certain number of associations which in turn depend on the individual organization of the circuits of the person involved.

The reactivation of the meta-circuits is thus ensured either by recognition or by recollection. The integrity of certain axial formations: e. g. the reticular system and that of the hippocampo-mamillary bodies seem indispensable for this recurrence to take place i. e. recollection, and it would seem that it were necessary in order to ensure prolonged engrammation. Ritchie Russell<sup>17</sup> considers this, and no doubt rightly so, to be the explanation of the retrograde amnesia observed following head injury. In these cases, the alterations of the hippocampo-mamillary system prevent recollection of situations experienced before the accident — recollections which would normally have enabled these souvenirs to be well fixed in the mind. This inability to remember also seems — and we shall reconsider this point later on — responsible for the "gradual forgetting" that was observed during thiamin deficiency encephalopathies with lesions of the mamillary bodies.

The simultaneous or successive effect of experience and recall appear to be not only necessary for the maintenance of the reactivated circuits but also for the acquisition of new circuits. These circuits will be enriched not only by the creation of new circuits due to stimulations furnished by new experiences but also by the creation of new combinations between older circuits recollection playing a major part in this ability for combination that is characteristic of human thought.

What is the actual *nature* of the alterations which ensure the fixation of experience in the brain? It is as yet not well known and the characteristics of the ele-



mentary mnesic trace in the region of the neuron constitutes one of the key problems of the physiology of memory. Despite numerous histological studies which suggest the existence of morphological modifications at the neuronal level during their functioning there is no clear proof that enables one to consider them as definitely representing the specific traces left by an item of information in the cell.

The neuro-electric activity of the brain provides proof of much closer relationships between this activity and the fixation of experience. Hence through neuro-physiology we are informed that bio-electrical modifications can survive after the stimulations giving rise to them, for a period of several seconds and, under certain experimental conditions during several minutes or even several hours. They could ensure "short term memory". But in order to form more lasting connections between the different elements of a neuronal circuit that is the support of "long term memory" it is necessary to consider the possible existence of more durable modifications. The hypothesis of self-maintenance by reverberatory circuits first advanced by Lorente de No<sup>12</sup> in 1933 has been refuted by the author himself in 1962 because of the inability to explain in this case, the persistence of souvenirs following severe shock or deep anaesthesia which temporarily interrupt any electrical activity of the brain.

Permanent chemical alterations would therefore seem necessary in order to explain the persistence of acquisitions. Up to the present time the contribution of biochemistry to the understanding of the elementary mnesic trace is still very rudimentary. In spite of the experimental observations of Hyden and Lange<sup>8</sup> (1960) and those of Morrell<sup>13</sup> (1961) concerning the variation in the protein content under the influence of neuronal activity, and those of Bennet et al.<sup>2</sup> (1964), showing an increase in the cholinesterase of the auditory and visual cortex of rats submitted to intense activity, it is too early to be able to conclude as to whether these variations do in fact represent the biochemical basis of the ele-

mentary mnesic trace. Hence, for the time being one is obliged to limit oneself to certain hypotheses and according to Hyden<sup>8,9</sup> considerable importance should be given to the RNA-DNA system. According to this author a modulated stimulation on reaching a neuron leaves its trace on this system, and when a new identical stimulation occurs it produces the discharge of the post-synaptic influx towards the next neuron and so on.

This hypothesis is ingenious but in view of our present knowledge, to rank the mechanism of human memory with the mechanism of genetic memory gives rise to certain difficulties. Indeed, how can one regard the functioning of a system consisting of neurons that are marked in an identical manner by a "small scale experience" affecting a macro-molecular structure? The specificity of a given RNA for a given neuronal circuit must surely appear to be in opposition to the multiple combinations necessary for the associations which ensure the successive elaborations of the different elements of human knowledge?

The data described above have led to the concept that for an experience to be able to exist, be maintained and displayed, a fairly widespread neuronal constellation would be required: i. e. the meta-circuit. If it is indispensable those biochemical alterations take place in each individual constituent neuron of the meta-circuit in order to maintain a functional homogeneity, would it not suffice that each of these elementary mnesic traces consist of a substance which, by its presence, absence or amount would either enable or prevent the passage of influx from one cell to another? If this were so then the elementary mnesic trace would not represent the whole of the knowledge supported by a meta-circuit any more than a footprint in the sand represents the whole of a walk.

"The unit of knowledge" would not occur within a single neuron but in this group of neurons involved during the acquisition of this knowledge.

That is why it is still hoped that histochemistry and the electron microscope may provide new information which may one day provide the solution to a problem not yet solved.

These observations on the physiological basis of the engrammation of experience and on the structural organization of knowledge will enable a better understanding of the various types of amnesia encountered in human medicine.

## 2) Physiological loss of memory and Affective Amnesias

Is it not common to all us to be surprised in remembering an old souvenir extremely clearly that we had thought long forgotten? A methodically devised psychological investigation manages to revive so many souvenirs that a psychoanalyst once remarked: "nothing is forgotten". In actual fact, much of our knowledge that has been perfectly well learnt becomes completely and definitely effaced. The experiments of Ebbinghaus<sup>7</sup> and Jost<sup>9</sup> at the end of the last century have been confirmed scores of times, i. e. physiological loss of memory is a reality.

Consequently how can one explain why and how certain experiences are retained whereas others are not? the maintenance of information above the threshold of forgetting is an active phenomenon in which repetition plays a fundamental part. It is this which ensures the functional emergence of a very great number of marked mnesic circuits underlying our multiple knowledge and which, in the interplay of the various arrangements imposed by subsequent development, are the object of reactivations that reinforce and maintain them. However, we must go one step further and ask ourselves why it is that certain souvenirs are repeated?

This leads us to the study of motivations: if the souvenirs are well fixed and repeated they are for some reason or another important to the person. From the first months of life onwards a certain ex-

perience of the body and the outside world becomes engrammed. The vital role played by this first structuration is well known and it will serve as a basis for reference for new acquisitions, determining the choice of what will be apprehended and retained among all the new items of information derived from the environment. Therefore under the double constraint of what has already been engrammed and the changes produced by new experiences, the engrams on which our knowledge and affective life are based, are gradually built up.

Although certain structures of the brain appear to play an important part in emotional life, it is difficult to visualize how any one behaviour can be purely affective and it seems more likely that each fragment of experience consists of an affective factor on the same basis as a cognitive and expressive factor. This implies the probable participation of these structures in the elaboration of a very large number of meta-circuits whose form of expression is then sensory-affective-motor. Consequently the more aspects one knows of such and such an object or situation the richer the emotional experience attached to this object or situation and that finally tastes and distastes are the intrinsic characteristics of knowledge and not the exclusive products of a few emotional centers functioning separately. It follows that we are able to apprehend new information more easily if we find references to it in our previously established engrams, this structural continuum being reflected from the psychological point of view by the fact that our present and future interests depend on the past. Furthermore the more a notion is experienced the greater are the chances that it will escape being forgotten insofar as the recollection of one or other of its aspects leads —because of the structural links between them— to the reactivation of other meta-circuits supporting the other aspects of this knowledge.

This attempt to establish a relationship between the psychological content and neuronal support of souvenirs enables a

better understanding of the mechanism involved in certain disorders of memory such as those encountered during various psychiatric conditions.

We shall not dwell on the false amnesias of the anxious nor on the paucity of intellectual acquisitions during certain psychotic states (e.g. during attacks of melancholia, lack of interest, psychotropic drugs and electroshock occurring to a greater or lesser degree), but rather emphasize the amnesias observed during various neurotic conditions in which forgetting appears as an active phenomenon. These amnesias are essentially characterized by disorders of recall, certain souvenirs seem to have been totally forgotten by the subject who can no longer recollect them and who professes not to remember them when he is told about them. Although they sometimes affect the whole fund of personal memories, these amnesias are more often than not elective, i. e. limited to certain subjects that constitute the dark spot in the memory of the subject. However these elements are not in fact erased from memory but, on the contrary, perfectly preserved as may be seen when they reappear either spontaneously or during the course of psychotherapy. In these cases there are no traumatic or functional modifications of the physiological mechanisms taking part in the mnesic processes but a change in utilization, vitiated though still usable, of the normally functioning mnesic system. It seems as if, under the influence of contradictory factors, memories were organized in such a way that the circuits supporting the repressed elements are no longer accessible to stimulations which would normally evoke them. In fact it is most likely that this cutting out is incomplete as proved by the role of "these forgotten factors" in the constitution and maintenance of neurosis, and furthermore by their quality and richness when brought to light during psycho-analytical treatment. It is evident that this is only one particular case but it too shows the importance of previous engrams in the organization of our knowledge and behaviour.

### 3) Organic Amnesias

The study of subjects suffering from localized lesions of the brain, the observation of the effects of certain neurosurgical resections in Man, as well as certain experimental data obtained from animals, have shown that although any alteration of the brain disturbs the mnesic potential of the subject to a certain extent, the nature of this disturbance differs according to the localization of the lesion. Systematic examinations by means of appropriate tests make it possible to compare amnesias produced by cortical lesions with those resulting from the involvement of certain axial formations. Owing to recent research work this distinction, which was long felt to be valid by certain authors, now holds a definite place.

Let us briefly outline the essential clinical characteristics:

#### — *Cortical amnesias.*

The destruction of part of the cortex is nearly always accompanied in pathological conditions by the destruction of the neighbouring sub-cortical zone, together with the association fibres that enter, leave or cross it, and always alters to some extent the functional capacity of the brain. They cause a loss in the acquisitions and this is the reason why Jean Delay<sup>4,5</sup> called deficiencies of this type neurological amnesias. The study of these patients by means of appropriate tests shows that there is not only destruction of a certain number of past acquisitions, by that furthermore, the capacity for new learning is affected. Hence *any cortical lesion that disturbs a function, subsequently disturbs the memory of this function* is so far that what was engrammed in this region has been destroyed and that new engrams are reduced or even impossible.

What are the clinical characteristics of these cortical amnesias?

Memory tests clearly show:

1) A deficiency in the apprehension of a situation. This is marked by the inability of these subjects to grasp rapidly and retain



simultaneously for a certain time as many facts as they were able to before the occurrence of their lesion; this was easily revealed by means of various classical psychological tests. The latter demonstrated a slow apprehension of the situation which is understood little by little by a laborious process, which finally results in a reduced understanding, whereas in the same period of time and in the same situation a normal subject would have been capable of a far greater apprehension.

2) But these notions acquired slowly and laboriously are retained, as can easily be verified by tests repeated over a period of time; the retention curve was barely less than that of normal subjects.

Hence, cortical amnesia is characterized by the difficulties in memorizing but without there being "gradual forgetting".

The cortex is rather highly specialized and it is known that the deficiency varies according to the zone destroyed: if the lesions are predominantly situated in the primary sensory or motor areas then one witnesses motor or sensory deficiencies; if they involve the association areas then, depending on the region and the hemisphere affected, there are gnostic, praxic or language disorders. Inevitably one finds again the same specialized distribution in the deficiency of memorizing since this runs parallel to the functional deficiency. Depending on the site of the lesion the memory deficiencies of cortical amnesia will predominantly affect the audio-visual activities when the lesions are situated in the left hemisphere or the gnostic-praxic activities when the lesions are situated in the right hemisphere.

A loss of the frontal cortex which renders the subject incapable of any complex mental activity without, however, depriving him of his habits of movement and language, renders him incapable of acquiring any elaborate or complex new learning whereas he remains capable of acquiring a few simple verbal or praxic notions.

Thus word, gesture and souvenirs seem to be acquired elements having their own cerebral representation but all of them

obey to the same psycho-chemical requirements in order to be able to retain their functional individuality. Nevertheless, certain functional conditions may differentiate word and gesture on the one hand from souvenirs on the other.

Indeed, the former are to a certain extent directly related to experience through the intermediary of conversation and action. In view of the requirements of the outside world it will not be long before the movements and speech assume a certain form in terms of certain social criteria. Words and movements thus acquire a certain fixity; each time they are used, the meta-circuits supporting them are reactivated and consequently ensure a rapidity and perfection of motion and speech which then become almost second nature.

This cannot be said of souvenirs which may be considered to be the "motivated conservation of unusual experiences" and consequently they cannot be reactivated as a whole sufficiently often to be maintained above the threshold of physiological forgetting; this would account for their labile nature.

Here we may find the explanation of the relative frailty of souvenirs in comparison to current verbal or praxic experiences, which as we know, are less likely to be forgotten.

#### — *Axial amnesias.*

We shall not insist on the problem of amnesias resulting from an injury of the brain-stem or the posterior hypothalamus accompanied by a decrease in vigilance, even coma, but it is clear that the integrity of these formations is indispensable for the acquisition of souvenirs as is demonstrated in the case of the total amnesia corresponding to the period of coma.

But more important still are the amnesias caused by lesions of the circuit of Papez. It is known that this circuit consisting of phylogenetically old formations links the temporal cortex to the frontal cortex of the same hemisphere via the hippocampus, the pillars of the fornix, the mamillary body, Vicq d'Azyr's bundle, the

anterior nucleus of the thalamus, and the gyrus cingulus; the association fibres of the fornix connect it with the homologous formation in the other hemisphere. Papez<sup>14-15</sup> considered this circuit as the support of emotional life and it is now thought to be indispensable for the process of memorizing and recalling souvenirs. In fact, the wide range of this system, its close topographical relationships with other formations, often make it impossible to distinguish its functional role in traumatic, tumoral or degenerative lesions. Nevertheless, it has been possible to prove its role in the mnesic process by isolated injuries to some of its elements: e. g. the elective modification of the mamillary body due to thiamine deficiency (Gamper 1928; Delay, Brion and Elissalde<sup>6</sup> 1958); resection of the hippocampus during certain neurosurgical attempts to treat temporal epilepsy (Scoville and Milner<sup>18</sup>; Penfield and Milner<sup>16</sup> 1958). Paradoxically, experiments in Man in this field preceded animal experiments; the research experiments of Cordeau, Rasmussen and Stiepen<sup>3</sup> in 1961 and those of Thompson, Langer and Rich in 1964<sup>19</sup> furnished proof that in animals, a bilateral resection of the hippocampus or the destruction of the mamillary bodies decrease both the effects of recent conditioning and the possibilities of further conditioning.

It is during the deficiency encephalopathies observed in chronic alcoholics that the clinical picture of this type of amnesia is most frequently encountered, as was observed by Korsakoff<sup>11</sup> in 1887. It is above all characterized by "gradual forgetting". In some patients it is massive, ludicrous and manifests itself after a few minutes: the patient forgets the meal he has just eaten, the visitor he has just received, the examination he has just undergone; there is complete loss of memory.

The test of the hidden object easily proves this fact. Thus, Andre, a jovial alcoholic of 54 years of age, suffering from slight polyneuritis and pronounced disorders of memory, is unable to find the bottle of wine that was hidden in front of him a few minutes previously. When he is re-

minded of the nature of the hidden object, his search becomes more attentive but remains just as unsuccessful and is soon abandoned because he has forgotten what he is looking for and even that he was trying to find something.

In other cases the forgetting is less rapid but it can easily be shown, with memory tests, that loss of memory is slight after 15 minutes, but becomes accentuated after an hour and almost total after 24 hours or at the end of a week.

In certain alcoholics this gradual forgetting is in contrast with a good conservation of apprehension, the patient is easily able to repeat 7 figures or correctly perform a praxo-gnostic efficiency test.

In these cases, gradual forgetting constitutes the major symptom, the patient being incapable of retaining what he had apprehended correctly.

In actual fact these purely hippocampo-mamillary amnesias are not very frequent and in chronic alcoholics, with or without neurological lesions, there is frequently an overall reduction in the higher functions together with a very moderate capacity for memorization; the patient being able to learn very little and laboriously—but due to the effect of this *overall amnesia*—will even forget this. Our memory tests demonstrated that in the case of these alcoholics the retention curve was reduced far more rapidly in normal subjects or even than subjects suffering from cortical lesions, no matter what origin they had. We again found the same symptoms: i. e. a deficiency in immediate apprehension and gradual forgetting, in subjects showing primary or secondary dementia, and hence it would seem to reflect an overall cerebral involvement.

It should be added that, although the distinction between affective amnesia and organic amnesia can now be considered to be a clinical fact, it is not unfrequent, nevertheless, to find that these two mechanisms become intricate, producing *mixed organico-affective amnesias* that are difficult to analyse. Thus, a patient suffering from brain injury will not relate a

past souvenir that is painful or shameful to him whereas he would tell of more recent memories whilst another neurotic patient might develop an organic amnesia

secondary to an alcoholic encephalopathy produced as a result of drinking in order to forget.

## S U M M A R Y

It is important for present day neurologists and psychiatrists to be able to situate the problems of memory and amnesia within the more general framework of acquisitions and the conservation of experience which, throughout our lifetime, ensure the individuality of our behaviour and our personal knowledge.

All these different acquisitions are situated in the brain supported by neuronal networks, each item of which (or meta-

circuit) records a certain experience lived and the whole of which constitutes a structure that is certainly complex but perfectly well defined for any one individual.

One can consider that the different types of amnesia encountered in human clinical medicine are due either to disorders in the organization of our knowledge (affective amnesias), or to lesions of the brain which cut out our thesorus or cause disorders in its utilization (organic amnesias).

## R E S U M E N

Es importante para el neurólogo y el psiquiatra actuales tener la posibilidad de situar los problemas de la memoria y de las amnesias dentro del cuadro más general de la adquisición y la retención de las experiencias que a lo largo de toda nuestra vida aseguran la individualidad de nuestro comportamiento y de nuestros conocimientos.

Todas esas adquisiciones se encuentran en el cerebro y se deben a la acción de redes neuronales, de las que cada fragmento

(o meta-circuito) registra una determinada experiencia vivida y cuya totalidad constituye una estructura por cierto muy compleja pero perfectamente definida para cada individuo.

Se puede considerar que los diversos tipos de amnesia hallados en la clínica humana se deben o bien a un disturbio de la organización de los conocimientos (amnesias afectivas), o bien a lesiones cerebrales que quiebran nuestro thesorus o perturban su utilización (amnesias orgánicas).

## R É S U M É

Il est important pour le neurologue et le psychiatre contemporains de s'autoriser à situer les problèmes de la mémoire et des amnésies dans le cadre plus général de l'acquisition et de la conservation des expériences qui, tout au long de notre vie, assurent la singularité de nos performances et de nos connaissances personnelles.

Toutes ces acquisitions reposent dans notre cerveau supportées par des réseaux neuroniques dont chaque fragment (ou mé-

tacircuit) double une certaine expérience vécue et dont l'ensemble constitue une structure certes très complexe mais parfaitement déterminée pour un individu donné.

On peut considérer que les divers types d'amnésies rencontrés en clinique humaine sont dûs soit à un trouble de l'organisation de nos connaissances (amnésies affectives), soit à des lésions cérébrales qui amputent nos thésorus ou en perturbent l'utilisation (amnésies organiques).



## ZUSAMMENFASSUNG

Es ist wichtig für die heutigen Neurologen und Psychiater, die Möglichkeit zu haben, die Probleme des Gedächtnis und der Amnesien in dem weiteren Rahmen der Anschaffung und der Erhaltung der Experimente einzuschliessen, Experimente die, die Eigenart unserer persönlichen Leistungen und Kenntnissen während unseres ganzen Lebens sichern.

Alle diese Anschaffungen liegen in unserem Gehirn, von neuronischen Netzen (neuronal networks) gestützt, wovon jeder Teil (oder meta-circuit) ein gewisses er-

lebtes Experiment dupliziert und dessen Ganzes eine sehr komplexe, aber völlig festgelegte Struktur für ein bestimmtes Individuum bildet.

Man kann behaupten, dass die verschiedenen klinisch beobachteten Amnesientypen entweder durch eine Störung der inneren Bildung unserer Kenntnissen (affektive Amnesien) entstehen, oder durch Gehirnläsionen, die unser Thesaurus amputieren oder seine Anwendung zerstören (organische Amnesien).

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# Some Observations on the Psychological Mechanisms Impaired in the Amnesic Syndrome

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Mental disorders associated with injuries or disease of the brain provide an invaluable source of information about normal mental function. They have been studied extensively for the mapping of cerebral areas or systems in relation to specific behavioral functions, and indeed also serve for delineating those functions. Traditional taxonomies of psychological functions have evolved without reference to the neurological systems and processes that constitute the conditions of their operation. Such concepts as, e. g., attention, intelligence, memory, motivation, perception, although commonly understood and useful for communication in every day conversation, are not perfect constructs for a scientific analysis of behavior. They are too comprehensive to correspond to a specific functional system in the organism, and yet not self-contained enough to exclude overlap or interaction across their boundaries. Mental disturbances also tend to be diffuse, and cognitive disorders are almost invariably manifest in several taxonomically distinct functions.

The exceptional interest of the amnesic syndrome derives from the fact that it presents, by and large, a highly specific cognitive deficit. Memory and learning are gravely impaired, but cognitive processes that do not involve the application of stored

information seem to be virtually undamaged. While the most striking anomalies appear in the cognitive sphere, there are also abnormalities in affect, and the patient's loss of spontaneity is quite particularly notable, so that several students of the amnesic syndrome have presented it as primarily a disorder in the vegetative functions and vital layers of personality<sup>3, 6, 11, 12</sup>. Several, seemingly unrelated symptoms define the amnesic syndrome, and their pattern varies quite considerably, although not in any systematic accordance with the etiology of the disease. Patients, whose illness progresses through a first stage of Wernicke's encephalopathy, may differ among themselves in their chronic mental derangement as much as they differ from patients who develop the amnesic syndrome without an initial prolonged phase of confusion and peripheral neuropathy.

It seems probable that the pattern of the symptoms is determined by the site of injury along a functionally continuous circuit in the brain. Heranyi<sup>10</sup> thought that the more frontal the lesion on the Papez<sup>17</sup> circuit, the more prominent will be the loss of initiative and the dyskinesia, while damage restricted to the region of the mammillary bodies produces a "classic Korsakoff syndrome". Theories of this type

are valuable heuristically but must be treated as tentative. Lesions in the temporal lobe, for example, have produced severe amnesic impairment combined with apathy<sup>5, 8, 9, 25</sup>, and Adams<sup>1</sup> has observed a case of extensive lesions in the mammillary bodies of a patient whom he followed right up to death, and in whom he had noted no memory disorder. The quest of relating behavioral changes to specific sites in the brain is luring and well worth further pursuit, but there are also other trails for the investigator to follow. The psychologist has already learned a great deal from the amnesic syndrome when he observes that a comparatively isolated cognitive derangement is regularly accompanied by abnormally reduced spontaneity, and usually also by some anomalies in affect.

#### Lack of Initiative

It should be stressed that the joint manifestation of cognitive and of affective-conative disorders is by no means restricted to Korsakoff patients with a history of alcoholism. A post-encephalitic patient, whom the author has studied in collaboration with Dr. Raymond D. Adams, displayed some remarkable and quite severe amnesic disorders, and was also troubled by a sudden loss of initiative, by difficulties in maintaining his accustomed affectionate relations with his family, and concern about the future. For this young man soon became aware of this memory disturbance, and had good reason to fear that he might be unable to resume his duties as a breadwinner. With other amnesic patients it usually takes a long time before they express such concerns or display any sign of insight into their condition. Korsakoff patients, with a record of alcoholism and associated nutritional deficiency, typically arrive at it in the chronic phase of their illness. Some of them, however, may never fully emerge from the confusional state that marks the early phase of their disease, and that also seems to protect them from the bleak and fright-

ening image that a realistic assessment of their condition would unveil.

The author has had the opportunity to follow two young men for some 30 months, beginning about three weeks after operation by Dr. William H. Sweet and Dr. R. Thomas Ballantyne Jr. for a subarachnoid hemorrhage originating from an aneurysm in the anterior communicating artery. For a short while after operation these young men confabulated without restraint of reason or veracity, and contemplated their condition, including an extremely severe amnesic disturbance, with casual cheerfulness. Confabulation and disorientation were the first symptoms to clear up, closely followed by the mood of inappropriate and indiscriminating heartiness. Their retrograde amnesia also receded satisfactorily, but their severely impaired capacity for new learning has been improving very slowly, and is still a long way from its preoperative level.

While the clinical history of these two men is remarkably similar—they were operated on within two days from one another and shared a room in the hospital for several weeks—the courses of their further progress diverged somewhat after a few months. One of them regained after a while some power to hold new information, and could go back to work, albeit in a less demanding job than he had originally held. He also passed through a brief period of unusual irritability after his return home and, while for a long time he discussed his sexual impotence (another symptom apparently associated with the amnesic and mood disturbance) as nonchalantly as the other patient does to this day, more recently he has become quite depressed over this chronic incapacity. Otherwise, he is still placid and agreeable, willing enough to pursue the daily routine but apparently not ready to venture far beyond its boundaries, and appears to be untroubled by the residual memory defect. The other man is quite content to stay at home and help with whatever work there is about the house or, if asked, to join friends in some outdoor activity, but never



initiates such plans. He knows that some decision must be reached about work, and discusses the matter rationally but without visible concern. His oldest son keeps annoying him, taking advantage of the patient's affability, and it was a sign of real progress that after two years the father for once managed to follow up a threat by actually spanking the boy. Until that time his anger never lasted long enough to carry the act from the stage of warning into execution.

Our observations on the mood, initiative, and affective responses of chronically hospitalized alcoholic Korsakoff patients are apt to be blurred by an expectancy of dullness, and by the fact that the monotonous routine of a custodial institution offers far fewer occasions for spirited display than does even a conventional domestic surrounding. It is, nevertheless, remarkable that these patients should evince so little desire to engage in hobbies or in social interaction. The author has discussed his observations on the mood and affect of these patients in some detail elsewhere<sup>22</sup>, and here only wishes to summarize them. A display of emotion is unusual in these patients, except for a mood of detachment that could be taken for depression, and for a rare outburst of anger which, however, abates with unwonted rapidity. With few exceptions, old-established personal relationships seem to have lost their erstwhile significance, and no meaningful new relationships emerge in the institutional setting. An experiment has demonstrated that these patients cannot even sustain an informal conversation among themselves, past an opening comment. Since, however, some of these changes in personality apply with much less force to other types of amnesic patients, they may be attributed to the institutional climate as well as to the neurological derangement.

### Memory Disturbance

The impairment of learning and memory in the amnesic syndrome has been des-

cribed and analyzed exhaustively in the literature. Several investigators<sup>7, 14, 15, 16, 24</sup> have proposed that personal experiences are forgotten more rapidly and completely than the impersonal type of information encountered in the experimental laboratory, although in Kohnstamm<sup>13</sup> there was at least one advocate of the opposite view. It is certainly more remarkable that a woman should have no recollection of her marriage that had occupied a goodly stretch of her adult life, or of a recent accident that has left her with a painful wound, than that she should forget a name or a list of meaningless words within a few seconds. There also seems to be strong support for the view under consideration in reports about amnesic patients whose overt behavior reveals their recognition of a medical procedure, but who disclaim ever having been subjected to that treatment, or in the case described by Williams and Smith<sup>27</sup>. Their patient was hospitalized with tuberculous meningitis shortly after

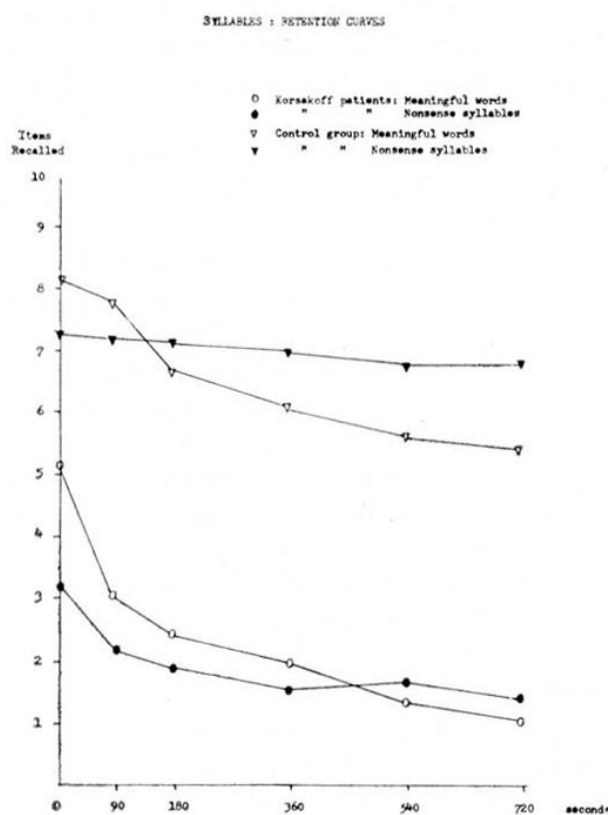


Fig. 1

taking a training course. Some time after his discharge from the hospital, he received a group photograph of the men who had taken part in that course, and he named them all correctly but could not remember where he had met them.

Isolated associations —between the instrument and its application or between a portrait and a name— are likely to persist intact from the period affected by retrograde amnesia, or may even be established during the period of anterograde amnesia, but cannot be fitted into their context. It should be noted, though, that amnesic patients are also apt to perseverate with false associations, nor should one overrate their achievements of learning in laboratory experiments. Figure 1 and Figure 2 illustrate the steep initial slopes of forgetting curves within seconds for lists of ten monosyllabic words or nonsense syllables, and within hours for a short story of 97 words, divided into 24 sections for recall. Figure 3

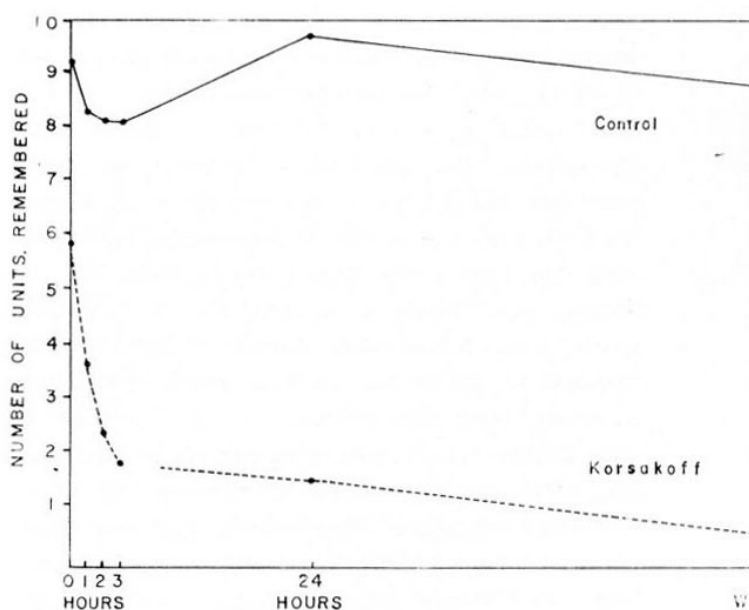


Fig. 2

shows the results of an experiment administered under four conditions, in which

## IMMEDIATE AND DELAYED RECALL OF A SENTENCE OF TEN WORDS

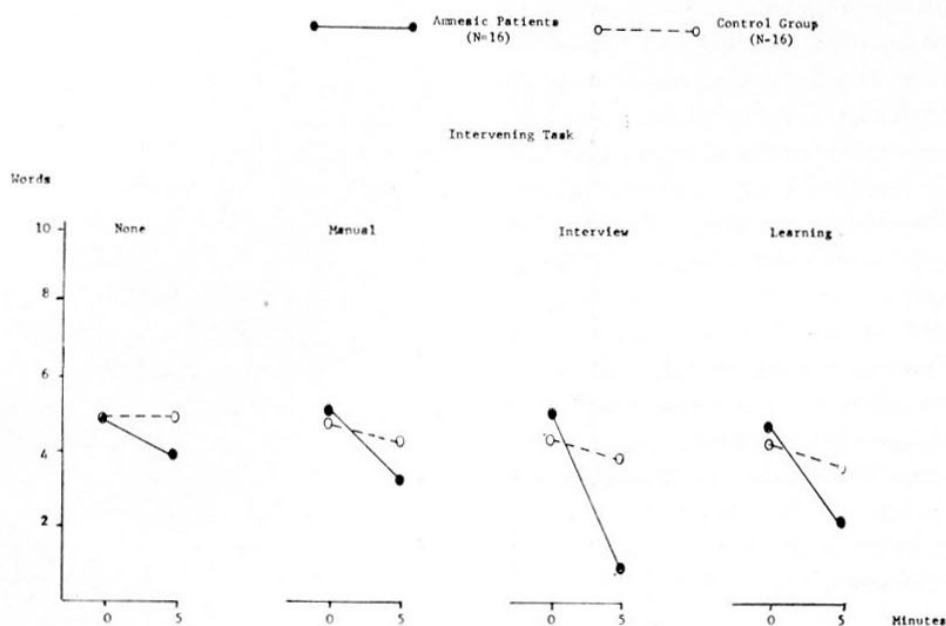


Fig. 3

recall for sentences of ten words was tested immediately and after a retention period of five minutes. While the lists of words and syllables were viewed in print for three minutes, the sentences and the story were presented only once and read aloud by the experimenter. With both the story and the lists of words the Korsakoff patients performed far below the control group in immediate recall, and also proceeded to forget at a faster rate. With the sentence, the two groups did not differ in immediate recall but differed in the expected direction after a period of five minutes.

Even though the retention period was unoccupied by any other activity, the amnesic patients could not hold all that they remembered of the sentence on first testing. With an interpolated task they forgot still more, and they retained least when the intervening activity consisted of an informal interview probing some affectively neutral information about the patient's childhood. The interview interfered with retention more potently than a second learning task because it disrupted the patients' task-set more completely. Unlike normal persons, amnesic patients cannot pick up the thread they have dropped; without some external impetus they cannot change their set or orientation to a problem, to a situation, or to the immediate sensory input. Most of the perceptual defects demonstrated in these patients, by means of laboratory experiments<sup>22</sup>, stem from their inability to shift the focus of attention between two objects, between two aspects of the same pattern, or from their incapacity to test one and then apply a different criterion to the same item of sensory input.

In one experiment lists of nouns were presented from a recorded audio tape, to each subject at his level of intelligibility. Given a long and randomly assorted list, half the words would be reproduced correctly, but from a list compiled to represent a familiar conceptual class, e. g., names of animals or of parts of the body, the score would rise considerably higher than 50 %. This was true of the amnesic patients as well as of the control subjects,

especially if they knew in advance what class of words to expect. Their real test only came with a list of 36 words, of which twelve were drawn from one category, twelve from another, and twelve from as many different classes. Figure 4 shows that the control subjects recognized more than half the words from both classified lists; just under ten of one and between eight and nine of the other. The Korsakoff patients benefited from one of the alternate expectancies but the second did not help their performance. The number of words correctly reported from the second classified list, like that from the mixed list which conflicted with expectancy, dropped below fifty percent. Evidently, the amnesic

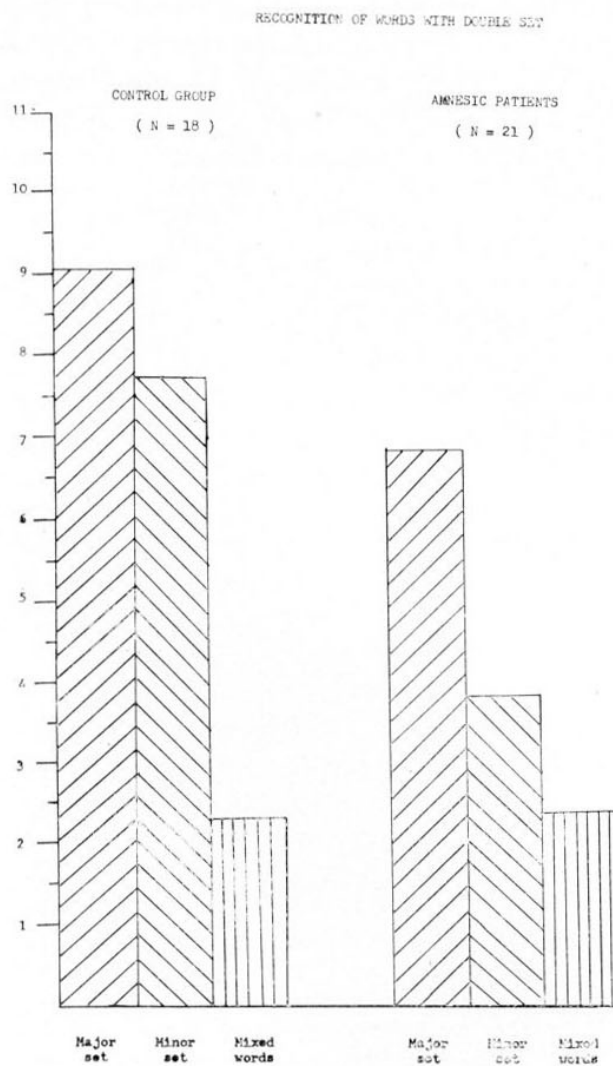


Fig. 4



patients were unable to operate with alternate sets, to switch to and from one to the other.

This inflexibility is apparent in motor performance and problem solving situations as well as in tests of perception and memory. Writing the letter S in the standard direction, a group of 20 Korsakoff patients performed no differently from a control group. Writing the same letter in reverse, however, these patients could barely reach half the level set by the control group, as shown in Figure 5. Given the task to discover the criterion, the defining attribute of arbitrary category of figures, Korsakoff patients will try the same approach again and again, even though it has failed and they can remember its failure. Their initial approach is apt to be concrete rather than abstract, and consequently they seem to persevere with a concrete orientation, but given the appropriate set they are quite able to perform the mental operations required for abstraction.

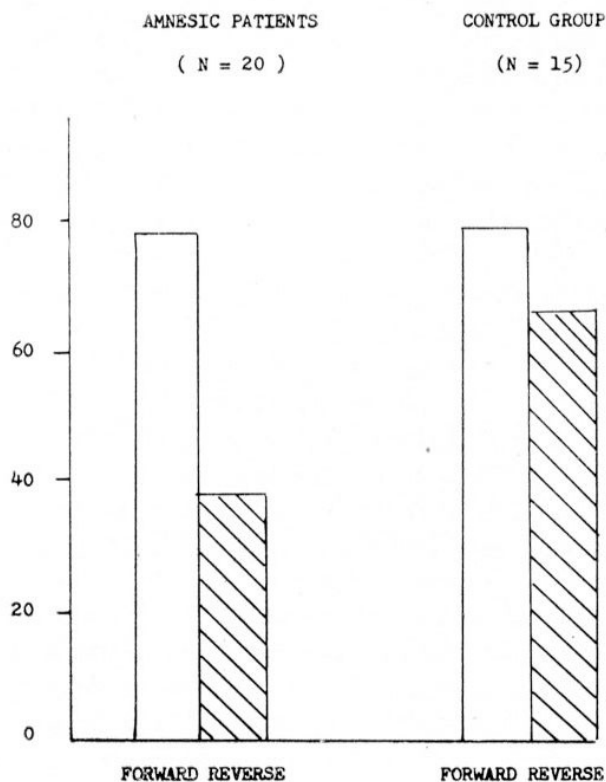


Fig. 5

Their thought processes are not markedly impaired and their judgment is usually quite realistic. Exceptions to this rule occur most commonly when the patient misjudges the temporal setting of an event. This is the typical example of confabulation, a symptom characteristic of the early confusional stage of the disease but much rarer in the chronic phase. A misperception of the momentary situation easily leads to the recasting of its occupants into strange roles, so that a familiar fellow patient is recognized as an old army pal, or the white-coated doctor as a physician encountered at some other hospital in the past. Confusions of this kind are apt to persist but often fluctuate freely and are easily demolished. The absence of a stable setting in reality offers a perfect occasion for paranoic delusions but these, though they do occur, are not a particularly common feature of the amnesic syndrome.

#### Uncertainty, contextual isolation and defective execution of plans

Recollections devoid of a firm temporal anchorage are, in this respect, like hallucinatory<sup>19</sup> or dream<sup>20, 26</sup> memories, with which they also share a quality of intuitive uncertainty. Pick<sup>18</sup> attributed the confabulations observed in amnesic patients to their loss of the ability to doubt, but in fact these patients express uncertainty and a readiness to be corrected in many situations that would not admit doubt in healthy persons. What normal man would confess to incertitude as to whether he was still married or divorced; what normal woman would be satisfied with a guess about the number of children she had borne? Contradictions on matters of such immediate personal concern, as in regard to one's age or occupation, arise because the object of self-reference is labile, now recognized as it was at one stage in the personal history, now as it appeared at another stage. That continuity which is the cardinal attribute of the self seems to be experientially unavailable to the amnesic patient. Not only does he exist in cross

section of life, without a longitudinal dimension, as Sollier<sup>21</sup> remarked, his successive moves in an action sequence are disjointed, as if they did not belong to the same comprehensive plan.

This fragmentation is particularly marked in regard to memories, which can often be elicited by pointed though uninformative questions. By that method, the author succeeded in obtaining from a patient a detailed and exact account about the fatal accident that had befallen his brother, even though at the time the patient was acting as if he had no recollection of that loss<sup>23</sup>. The accident had occurred several months prior to the patient's illness but the interview also extended to information about another event in the patient's family which had occurred after the onset of his illness. The most remarkable observation in that interview was not the patient's ability to furnish accurate answers to every question, but his subsequent unawareness of his performance. He believed that all the information that had passed between the interviewer and himself had been supplied by the former. The same interview was repeated half an hour later, with the same results: the patient answered the questions correctly, and again believed that he had been the recipient of all the information given.

Several varieties of paramnesia have been reported but none that corresponds to this instance, in which recall was both factually accurate and within the correct context, but not registered as a performance in remembering. How can one account for the absence of the experiential quality of an accomplishment, for mistaking the active for the passive role, after a minimal delay and in regard to a subject matter of such profound personal significance? Very likely the performance was in fact not experienced as an active one. Because he gave each item of information in response to a specific question, the patient may have felt as if he were a passive participant in a program imposed on him. Although he still had to search for the correct answers, he did not have to initiate those plans of

search that are necessary for remembering, and the proprietary sensation of remembering something may indeed arise with the setting up of such plans.

Students of the amnesic syndrome never accepted a model of losing discrete items of information as a satisfactory explanation of the memory disturbance. That theory is no more convincing in its modern guise, as a defect in storage in contrast to the acquisition or retrieval of memories. The patient who can name the persons on the photograph but does not remember where he met them<sup>27</sup>, or is upset at the sight of the E. S. T. apparatus without recalling his seeing it before<sup>2</sup>, or avoids shaking hands with the interviewer for fear he might stab her with a pin, but is unaware that this is what had happened to her a few minutes earlier<sup>4</sup>, is impaired, not in the storage of information, but in relating the information stored to its context. Storage and acquisition, long and short term memory, seem alike to be impaired in the amnesic syndrome. It is reasonable to assume that the damage is in a mechanism that is necessary for the completion of both operations: putting into storage for future recall and extracting from storage. Both operations, to be effective, depend on the execution of plans, and there is ample evidence that amnesic patients are impaired in setting up, in resuming after interruption, and in carrying through to completion plans of action that need to be checked and may demand revisions in response to contingencies.

The retrieval of discrete items of information, as well as the registration of such items for future reference, involves operations of search and test for the correct fit, which must be kept up until successful closure has been reached. Although their initial response may be quite adequate, amnesic patients cannot complete these operations if they involve the selection between alternate courses, or demand sequential responses to conditional cues. Their memory fails unless retrieval can follow the most direct route, as happens with overlearned skills, e. g., in the use of language. If, how-

ever, the required information is not immediately available, the necessity to search further or in other directions, to check the correctness of fit, is apt to disrupt their plan. They stop before reaching closure, possibly also because they do not recognize the misfit of their answer, although the chronic patient usually stops with an admission of ignorance.

Similarly, these patients do not complete the operations necessary for registering new information —be that a personal experience or some formally presented message— with all the cross references necessary for its subsequent retrieval on request. These operations extend further than an

appropriate perceptual response to stimuli that impinge on the receptors, and they may not be involved in simple associative learning, for most amnesic patients do manage to learn something at that level. The model here proposed to account for retrograde and anterograde amnesia certainly fits in closely with, and is influenced by, the observations about the lack of initiative in amnesic patients. It implies that the basic impairment is conative rather than cognitive, but the fact that the amnesic syndrome can be manifested in various patterns of its constituent symptoms allows for alternative theories.

## S U M M A R Y

The amnesic syndrome is discussed primarily in regard to the information it furnishes on the psychological mechanisms impaired in this disease. Since the syndrome is associated with lesions in certain specific areas of the brain, it is inferred that those areas are involved in the normal functioning of the mental processes affected. A lack of initiative is as characteristic a symptom of the amnesic syndrome as are retrograde and anterograde amnesia. Experimental results show that the rate of forgetting in amnesic patients is steep, as is also their forgetting of personally rele-

vant information, and that they have special difficulties in alternating task sets and in resuming performance after interruption. Clinical observations are listed to illustrate certain characteristic anomalies in remembering that can occur even though the patient gives the correct responses. It is proposed that both the registration of new information for future recall and the recall of previously learned information involves certain action plans, and that the basic defect in the amnesic syndrome is an inability to initiate, to resume after interruption, and to complete action plans.

## R E S U M E N

El síndrome amnésico es considerado al principio con relación a la información que proporciona acerca de los mecanismos psicológicos alterados en esta enfermedad.

Dado que el síndrome está asociado a lesiones situadas en determinadas áreas del cerebro, se infiere que estas últimas están involucradas en el funcionamiento normal de los procesos mentales afectados. La falta de iniciativa es un síntoma tan característico del síndrome amnésico como lo son la amnesia retrógrada y anterógrada. Los

resultados de la experimentación demuestran que en los pacientes con amnesia la proporción de olvido es muy pronunciada, así como también lo es el olvido de datos personales importantes; además evidencian particular dificultad en alternar tareas distintas y en retomar la tarea después de una interrupción.

Las observaciones clínicas son enumeradas para ilustrar ciertas anomalías características del recuerdo que pueden presentarse aún cuando el paciente responda en



forma correcta a las preguntas formuladas. Se plantea el hecho de que tanto el registro de datos nuevos a ser recordados en el futuro como el recuerdo de datos previamente adquiridos implican la existencia de ciertos planes de acción; de aquí

que la deficiencia básica en el síndrome amnésico reside en la incapacidad para iniciar planes de acción, retomarlos luego de una interrupción y realizarlos en su totalidad.

## R É S U M É

Nous présentons le syndrome amnésique premièrement parcequ'il nous fournit de l'information sur les mécanismes psychologiques qui sont dérangés dans cette maladie.

Etant donné que ce syndrome est associé avec des lésions dans certains parties du cerveau, il est déduit que ces parties comportent la fonction normale du processus mental qui est affecté. Un certain défaut d'initiative est un symptôme caractéristique du syndrome amnésique, de même que l'amnésie rétrograde et antérograde le sont.

Des résultats obtenus expérimentalement montrent que chez les malades amnésiques le degré d'oubli est très haut, de même que l'oubli de l'information d'importance pour eux, et il paraît qu'ils ont des difficultés

particulières à faire alternativement des tâches diverses, et à les recommencer après une interruption.

Nous enregistrons des observations cliniques pour illustrer certaines anomalies caractéristiques du rappel que nous constatons, quoique le malade nous donne des réponses qui sont exactes. Nous sommes d'opinion que l'information qui est enregistrée pour le rappel futur, ainsi que le rappel de l'information qui a été déjà apprise, entraînent certains plans d'action, et que le défaut le plus important du syndrome amnésique est l'incapacité du malade de prendre l'initiative, de renouer des plans d'action après une interruption, et de les accomplir.

## Z U S A M M E N F A S S U N G

Es wird das Amnesiesyndrom zunächst in Hinblick auf die Kenntnisse, die es über die bei dieser Krankheit betroffenen psychologischen Mechanismen liefert, besprochen.

Da das Syndrom mit Läsionen gewisser spezifischer Zonen des Gehirns vergesellschaftet ist, nimmt man an, dass diese Zonen bei der normalen Funktion der affektierten mentalen Prozesse betroffen sind. Das Fehlen der Initiative ist ein ebenso charakteristisches Symptom des Amnesiesyndroms wie die retrograde und antero-grade Amnesie. Experimentelle Ergebnisse zeigen, dass das Vergessen bei amnesischen Patienten hochgradig ist, ebenso wie das Vergessen persönlich wichtiger Informationen und dass sie besondere Schwierigkei-

ten haben bei alternierenden Aufgaben und beim Wiederaufnehmen von Handlungen nach ihrer Unterbrechung.

Es werden klinische Beobachtungen geschildert, um gewisse charakteristische Anomalieheiten beim Ennennen zu illustrieren, die vorkommen können, obwohl der Patient korrekte Antworten gibt. Es wird angenommen, dass sowohl die Registrierung neuer Information für zukünftige Erinnerungen und die Erinnerungen an vorher gelernte Informationen gewisse Aktionsplanungen betreffen, und dass der grundlegende Defekt beim Amnesiesyndrom in der Unfähigkeit besteht, die Aktionspläne zu beginnen, nach Unterbrechung wiederaufzunehmen und durchzuführen.

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# Aspects Anatomo-Pathologiques des Amnésies

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La complexité clinique des troubles de la mémoire laisse entrevoir une grande variété possible de lésions anatomiques responsables de tels troubles et explique que dans de nombreux cas la pathologie en soit encore inconnue ou seulement soupçonnée ou éventuellement multiple.

Schématiquement, on peut distinguer dans le domaine clinique trois grandes variétés de troubles amnésiques organiques:

1 -- Les amnésies de fixation vraies, qui sont généralement lacunaires. Elles groupent les amnésies post-commotionnelles, post-comitiales, post-confusionnelles et post-sismothérapeutiques.

2 -- L'amnésie de mémoration ou syndrome de Korsakoff.

3 -- Les amnésies plus ou moins globales des processus démentiels.

La pathologie des cas du 1er groupe est mal connue, du fait de leur évolution habituellement favorable et de la rareté des vérifications anatomiques. De plus, dans les cas vérifiés, il n'est pas sûr que l'on puisse attribuer aux lésions retrouvées un rôle significatif dans la genèse du trouble mnésique.

Les lésions des cas du 3ème groupe sont parfaitement connues, mais leur diffusion habituelle les rend difficilement interprétables et leur retire toute valeur localisatrice.

Seule la pathologie du 2ème groupe est claire et précise et les lésions ont ici une topographie très particulière, d'un grand intérêt physiopathologique, qui peut éclairer partiellement la pathologie des 2 autres

groupes. Nous envisagerons donc en premier lieu l'anatomie pathologique des amnésies korsakowiennes.

## A -- PATHOLOGIE DU SYNDROME DE KORSAKOFF

Si le syndrome de Korsakoff, décrit initialement dans ses étiologies carentielles, garde une unité clinique caractéristique comportant l'association d'une amnésie des faits récents avec préservation des faits anciens, d'une désorientation temporo-spatiale, d'une fabulation de richesse variable et de fausses reconnaissances, son étiologie est par contre multiple et, à côté de la forme alcoolique classique, on en connaît des variétés de causes tumorales, vasculaires, neuro-chirurgicales, traumatiques, et enfin de causes rares ou même exceptionnelles.

Les lésions anatomiques rencontrées dans ces diverses étiologies ont un type histologique et une répartition topographique, qui varient selon les étiologies en cause tout en gardant pour chacune d'entre elles une ou plusieurs localisations très particulières.

Nous étudierons la pathologie des diverses étiologies du syndrome par ordre de fréquence approximatif.

1. *Forme classique: le syndrome de Korsakoff alcoolique*<sup>8, 15</sup> (fig. 1 à 4).

Le premier connu et le plus fréquent, le syndrome de Korsakoff alcoolique a une



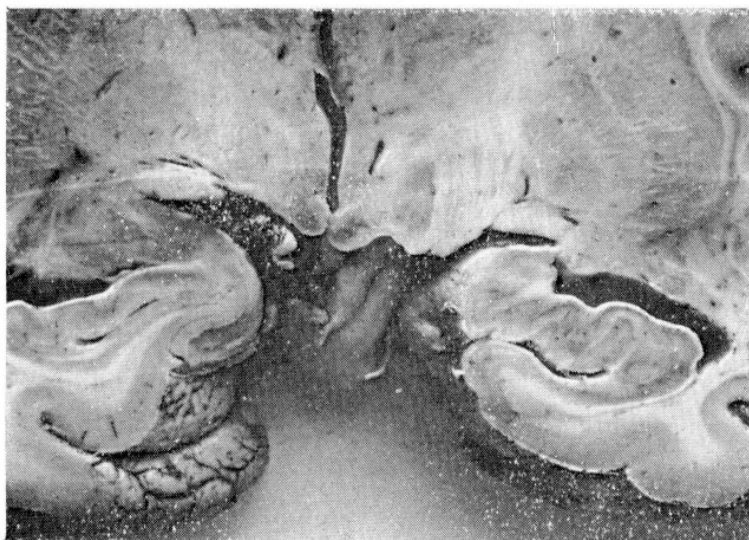


Fig. 1

pathologie schématique qui est celle de l'encéphalopathie de Wernicke. Encéphalopathie de Wernicke et syndrome de Korsakoff sont, en effet, les deux aspects évolutifs, le premier aigu, le deuxième sub-aigu ou chronique, d'un même processus lié à une carence en vitamine B1, elle-même conséquence indirecte de l'éthylisme chronique par la conjonction d'une dénutrition grave et d'une cirrhose hépatique.

Le type lésionnel est ici double et comporte d'une part des proliférations gliales et vasculaires, d'autre part des altérations neuronales revêtant le type de la dégénérescence rétrograde.



Fig. 2

Ces lésions sont situées dans diverses structures autour de l'Aqueduc de Sylvius entre le IIIème et le IVème ventricules. Elles sont bilatérales et symétriques et elles touchent constamment, dans l'encéphalopathie de Wernicke, les corps mamillaires (noyau principal), les noyaux réticulés périaqueducaux, le noyau dorsal du vague et plus inconstamment d'autres formations comme les noyaux du pont. Associés dans certaines structures (noyau du X, noyaux réticulés), ces types lésionnels restent isolés dans d'autres: prolifération glio-vasculaire pure sans lésions neuronales dans les corps mamillaires, dégénérescences rétrogrades isolées dans les noyaux du pont. Fait

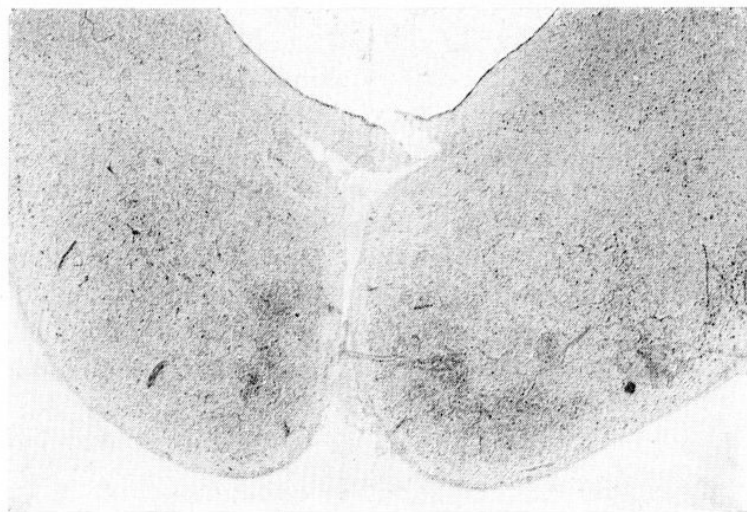


Fig. 3

négatif important, tous les noyaux somatomoteurs, et notamment les noyaux oculomoteurs, sont intacts.

Le syndrome de Korsakoff alcoolique peut réaliser anatomiquement l'ensemble de la répartition topographique lésionnelle que nous venons de décrire et qui constitue le tableau anatomique complet de l'encéphalopathie de Wernicke, et ceci n'est pas étonnant puisque ces maladies sont deux stades évolutifs d'un même processus et qu'on y observe souvent une phase aiguë terminale. Mais certains cas de syndrome de Korsakoff ont des lésions plus limitées dont le dénominateur commun est l'atteinte du noyau principal du corps ma-

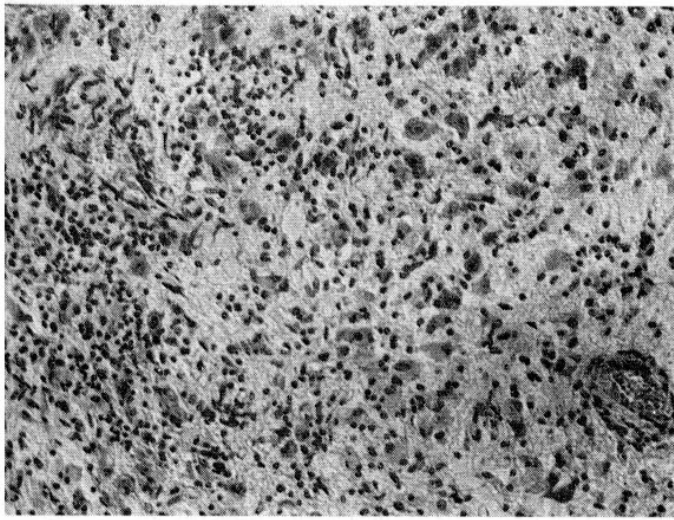


Fig. 4

millaire, démontrée par Gamper en 1928. Ce noyau est parfois même seul touché dans quelques observations privilégiées<sup>7</sup>. Il en résulte que la lésion bilatérale, glio-vasculaire du corps mamillaire est actuellement considérée comme responsable du trouble mnésique particulier du syndrome de Korsakoff.

## 2. Syndromes de Korsakoff d'origine carentielle<sup>8, 15</sup>.

D'autres carences thiaminiques d'origine non alcoolique peuvent engendrer un syndrome de Korsakoff. De tels cas avaient déjà été décrits par Korsakoff lui-même, mais leur nature carentielle ne fut prouvée que dans les années 1935-1945. Le facteur causal est ici une dénutrition qui a

été observée initialement dans des suppurations prolongées ou des suites opératoires troublées à une époque dépourvue de réanimation efficace, ou encore dans des cas de vomissements incoercibles de la grossesse. Ultérieurement on a décrit toute une série de syndromes de Korsakoff carentiels non alcooliques dans des cancers digestifs, chez des gastrectomisés, dans des carences de guerre en Extrême-Orient.

Les lésions anatomiques sont identiques à celles du groupe précédent, avec la même constance de l'atteinte bilatérale du corps mamillaire principal.



Fig. 6

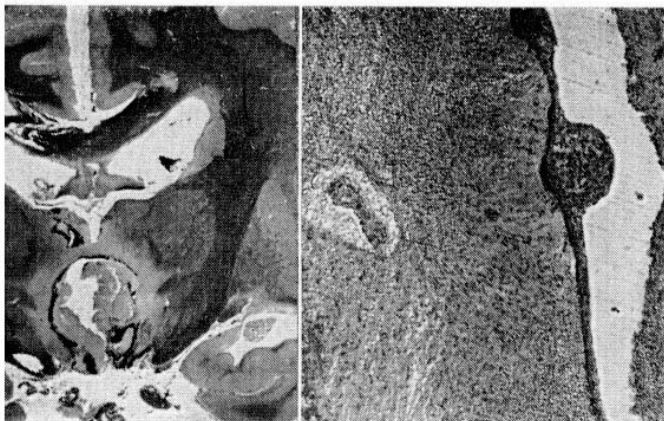


Fig. 5

## 3. Syndromes de Korsakoff d'origine tumorale<sup>11</sup> (fig. 5, 6, 7).

Les tumeurs responsables d'un syndrome de Korsakoff sont de types divers, mais ont une répartition topographiques très précise. Dans les 56 cas de la littérature qui sont à la fois cliniquement certains et anatomiquement vérifiés, il s'agit toujours de tumeurs profondes et médianes, siégeant: soit dans le plancher du IIIème ventricule ou dans le IIIème ventricule, détruisant ou comprimant les corps mamillaires, les for-



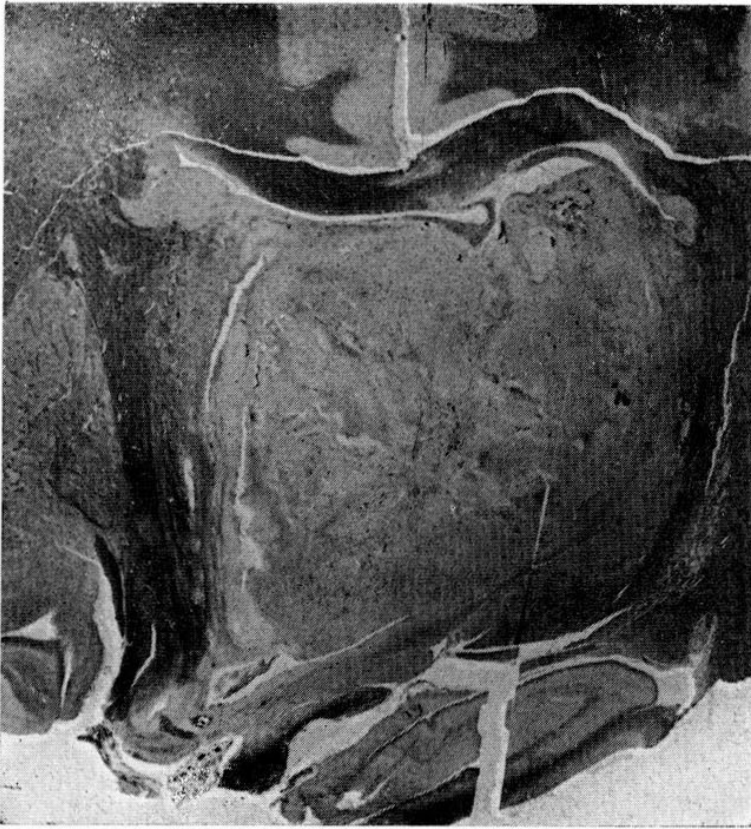


Fig. 7

nix ou les thalamus; soit dans le septum, coupant les 2 fornix; soit dans la partie interne des lobes frontaux, détruisant une partie des 2 gyrus cingulaires. De toutes ces tumeurs, la plus fréquente est le craniopharyngiome de l'adulte à développement postérieur, qui représente un tiers des cas de syndrome de Korsakoff d'étiologie tumorale et qui agit habituellement par compression mamillaire directe.

#### 4. *Syndromes de Korsakoff d'origine vasculaire*<sup>9, 10</sup>.

Leur connaissance est relativement ancienne, mais leur interprétation exacte, récente<sup>10</sup>. Le nombre de cas anatomo-cliniques rapportés en est très restreint, mais leur étude est capitale sur le plan des localisations. Schématiquement, les lésions sont toujours bilatérales, mais il faut distinguer les formes symétriques et les formes asymétriques.

On connaît 7 formes symétriques:

La plus ancienne est une forme bioccipitale décrite par Dide et Botcazo, qui a

comporté un syndrome de Korsakoff, une hémianopsie double, une alexie et une astéréognosie droite, et qui montrait anatomiquement un ramollissement ancien bilatéral du lobe lingual, donc du territoire de l'artère cérébrale postérieure; à droite, la lésion débordait sur les circonvolutions temporales inférieures adjacentes; à gauche, la partie postéro-externe du thalamus était lésée; aucun renseignement n'est donné sur l'état de la fimbria.

Deux observations très anciennes également, celles de Mabile et Pitre et de Zacher, comportent la première un syndrome de Korsakoff d'évolution prolongée, lié à deux ramollissements symétriques de la substance blanche frontale sous-cingulaire, et la deuxième une amnésie antérograde par lésion bilatérale de la substance blanche pré-frontale.

Parmi les quatre dernières observations, plus récentes, celles de Glees et Griffith,

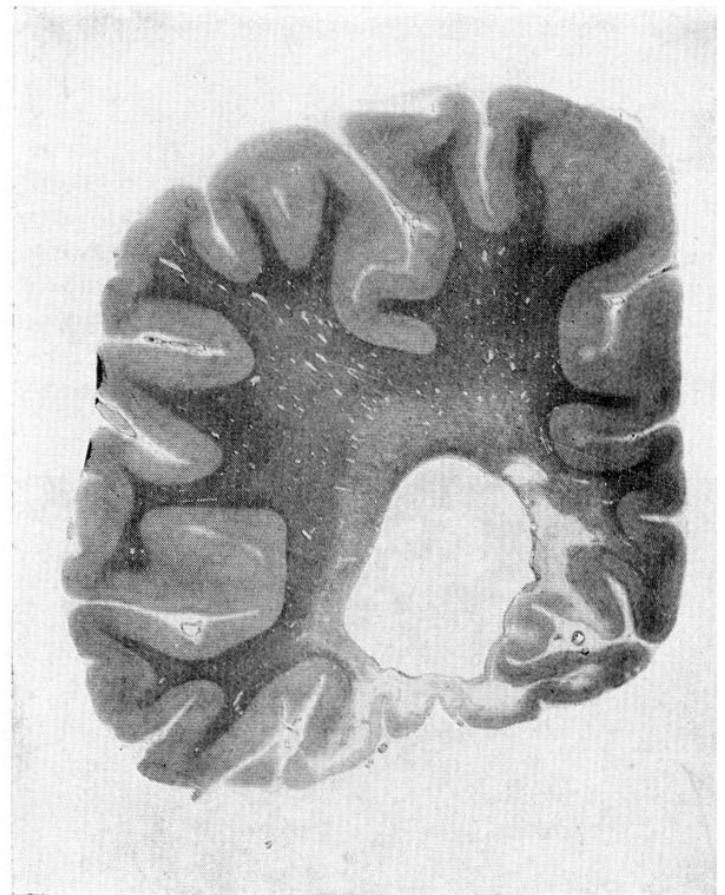


Fig. 8



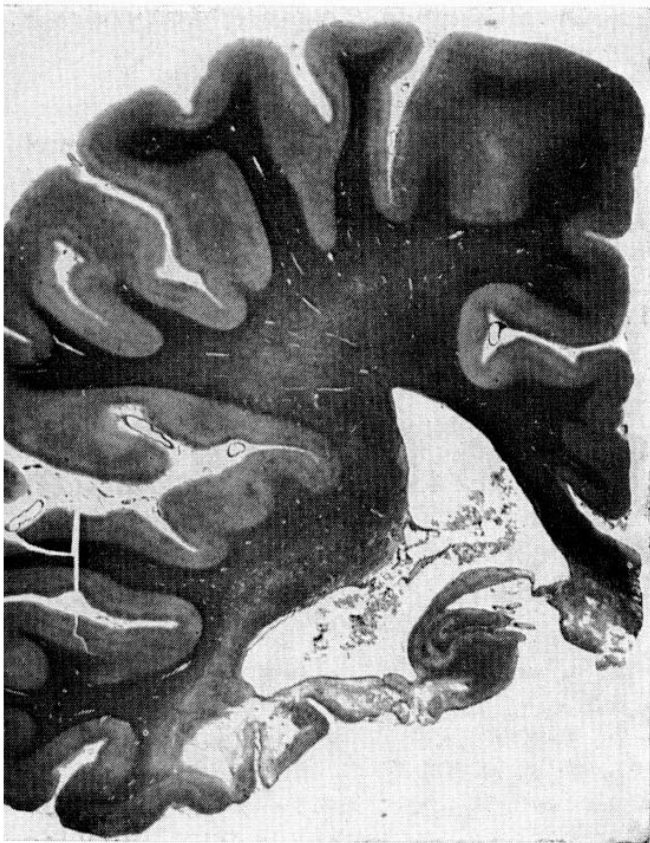


Fig. 9

de Victor Angevine Mancali et Fischer et de Nomura et Ishikawa ont un point commun: l'existence de ramollissements anciens bilatéraux des deux cornes d'Ammon, avec ou sans dégénérescence secondaire du fornix et des corps mamillaires. La quatrième, observation 1 de Castaigne et Coll., comporte une section bilatérale des faisceaux de Vicq d'Azyr par un ramollissement bithalamique.

Deux formes asymétriques ont été publiées: l'une par Dide et Pezet, l'autre par nous en collaboration avec Delay, Escourolle et Melle Marquès<sup>9</sup> (fig. 8 à 11). Cette dernière comporte un ramollissement occipital du territoire de l'artère cérébrale postérieure gauche, avec atteinte de la fimbria et atrophie secondaire unilatérale du fornix et du corps mamillaire; à droite, on trouve exclusivement un ramollissement limité thalamique, interrompant le faisceau de Vicq d'Azyr. Le cas de Dide et Pezet montrait également un ramollissement organisé occipital interne gauche et un foyer

vasculaire dans la couche optique droite, mais la localisation exacte de ce dernier n'est malheureusement pas précisée.

Un 3ème cas inédit de Boudin est encore plus asymétrique. Il comportait cliniquement un premier accident vasculaire ayant laissé une hémianopsie latérale homonyme droite isolée et un deuxième accident ayant entraîné un tableau complexe associant syndrome korsakowien, syndrome frontal, dyspraxie du membre supérieur gauche avec perte d'incitation motrice de ce membre, sans aucune apraxie à droite. Anatomiquement, on a trouvé un ramollissement ancien occipito-temporal inférieur gauche avec atteinte de la fimbria et atrophie mamillaire gauche secondaire, et une lésion droite plus récente détruisant tout le territoire de l'artère cérébrale antérieure droite, en englobant notamment le cingulum droit.

##### 5. *Syndromes de Korsakoff neuro-chirurgicaux.*

Sous cette rubrique, nous avons groupé des cas secondaires à des interventions de psycho-chirurgie ou de chirurgie des mouvements anormaux. Ils restent peu nombreux et sont heureusement plus souvent transitoires que permanents.

Les premiers décrits et les plus connus sont les cas de Scoville et Milner, qui réa-



Fig. 10



Fig. 11

lisèrent des syndromes de Korsakoff permanents, très intenses, mais pratiquement exempts de fabulation, à la suite d'exérèse bilatérale de la corne d'Ammon chez des sujets épileptiques ou schizophrènes.

Les autres cas concernent d'autres localisations. Ce sont d'une part les chronotaxis ou confusions du temps, syndromes de Korsakoff transitoires décrits par Spiegel, Wycis, Orchinik et Freed, au cours de coagulations du noyau dorso-médian ayant pu déborder sur le noyau antérieur du thalamus. Ce sont d'autre part de syndromes korsakowiens passagers, marqués essentiellement par une assez riche fabulation, décrits par Whitty et Lewin dans des cingulectomies bilatérales. C'est enfin un cas unique de Hassler et Riechert de coagulation bilatérale du fornix, où les 2 coagulations ont été faites à un an d'intervalle et où la coagulation du 2ème fornix a déclenché le syndrome.

#### 6. *Syndromes de Korsakoff post-traumatiques* <sup>23</sup>.

Ces syndromes observés après certains traumatismes crâniens graves sont très purs cliniquement, mais leur évolution est habituellement favorable et on n'en connaît qu'un cas anatomique, celui d'Evrard, Divry et Molders qui montrait des lésions fronto-orbitaires bilatérales.

#### 7. *Syndromes de Korsakoff d'étiologie rare ou exceptionnelle* <sup>12, 23</sup> (fig. 12).

Dans cette rubrique, rentrent des cas disparates, parfois uniques. Ce sont par exemple les syndromes de Korsakoff d'étiologie encéphalitique <sup>23</sup> dont la plupart sont cliniques mais dont un est pathologique, celui de Conrad et Ule qui était une encéphalite nécrosante avec lésions bitemporales, internes, atrophie secondaire des fornix et des corps mamillaires. On peut citer également les syndromes de Korsakoff dus à des processus granulomateux bitemporaux, qui ont été rapportés par Bertrand et Gruner.

Un cas unique de syndrome de Korsakoff post-comitial, après état de mal épileptique déclenché par une corticothérapie intensive pour asthme subintrant, a été rapporté récemment par Delay et Coll. <sup>12</sup> et comportait une lésion bilatérale limitée au champ de Sommer et de type anoxique. Son in-

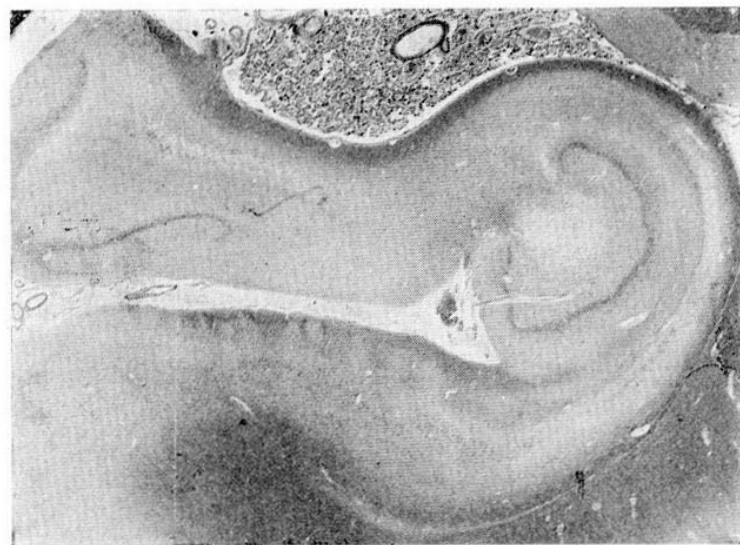


Fig. 12



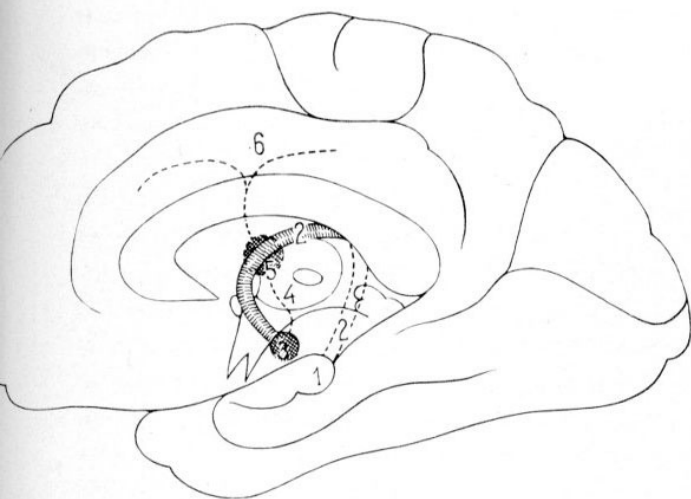


Fig. 13

térêt est de montrer d'un côté une dégénérescence secondaire du corps mamillaire et du noyau antérieur du thalamus, mais surtout de révéler des lésions bilatérales intenses du noyau latéral dorsal du thalamus probablement secondaires à la lésion ammonique.

Enfin dans les rares formes thalamiques pures du syndrome de Creutzfeldt-Jacob, on observe une démence avec troubles mnésiques prédominants, notamment dans un cas personnel rapporté avec Garcin et Khochneviss<sup>19</sup> où l'atteinte mnésique était de type korsakowien et où la lésion thalamique détruisait totalement les deux noyaux antérieurs et les deux noyaux latéral dorsal du thalamus.

#### 8. *Regroupement topographique* (fig. 13).

De la multiplicité des localisations lésionnelles rencontrées dans les diverses étiologies du syndrome de Korsakoff se dégage la notion d'un système de structures formant un circuit bilatéral et symétrique, qui a été décrit initialement par Papez comme circuit de l'émotion avec des arguments critiquables, mais qui a été rattaché ultérieurement par Benedek et Juba à la pathologie de la mémoire. Cette opinion de Benedek et Juba a été amplement confirmée par les diverses localisations lésionnelles que nous venons de décrire et actuellement on a acquis la notion d'un

circuit dont l'intégrité bilatérale est nécessaire au bon fonctionnement des mécanismes de mémoration. Inversement la lésion de ce circuit entraînera un syndrome de Korsakoff à condition qu'elle soit bilatérale. L'atteinte mamillaire bilatérale et isolée de l'alcoolisme chronique engendre le syndrome le plus pur et le plus caractéristique. Les autres sièges peuvent donner des tableaux plus atypiques, soit par confusion surajoutée qui masque le syndrome et c'est le cas d'un certain nombre de tumeurs profondes, soit par prédominance d'un groupe de symptômes et ce semble être le cas pour les formes ammoniques où la fabulation manque souvent, ou pour les formes cingulaires où la fabulation semble au contraire prédominante alors que l'amnésie y reste plus discrète. La bilateralité des lésions paraît être un facteur capital mais non leur symétrie; des lésions asymétriques, comme celles de certains syndromes vasculaires, entraînent des syndromes de Korsakoff analogues à ceux des lésions symétriques; au contraire, les lésions unilatérales de quel côté qu'elles soient ne déclenchent habituellement pas de trouble mnésique.

Le circuit de Papez est, rappelons-le, un circuit hémisphérique interne limbo-limbique réunissant la corne d'Ammon au cingulum par l'intermédiaire de toute une série de structures profondes. Il comprend

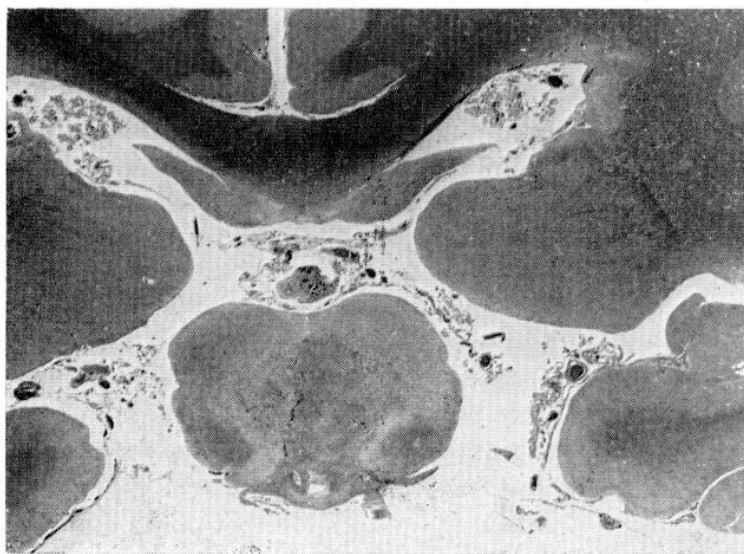


Fig. 14





Fig. 15

successivement la corne d'Ammon, et particulièrement le champ de Sommer, la fimbria, le fornix le noyau principal du corps mamillaire, le faisceau de Vicq d'Azyr, le noyau antérieur du thalamus et le cingulum (aire 23 et 24).

Il faut y adjoindre, comme l'ont montré Locke, Angevine et Yakovlev et comme semble le prouver notre cas de syndrome de Korsakoff post-comital<sup>12</sup>, un deuxième circuit parallèle partant de la corne d'Ammon, passant par le fornix mais projetant directement sur le noyau latéral dorsal et sur le noyau antérieur du thalamus puis se poursuivant vers le cingulum. Le noyau latéral dorsal ne reçoit que des fibres directes de la corne d'Ammon tandis que le noyau antérieur du thalamus reçoit un faible contingent de fibres directes et un contingent beaucoup plus important de fibres indirectes ayant fait un relai mamillaire. L'intégration éventuelle du noyau latéral dorsal aux circuits des mécanismes mnésiques serait un fait important, car la physiologie de ce noyau est jusqu'ici inconnue.

## B — PATHOLOGIE DES AMNESIES DE FIXATION VRAIES

A l'opposé de la pathologie parfois compliquée mais toujours schématique et ac-

tuellement bien connue du syndrome de Korsakoff, celle des amnésies lacunaires est très mal connue et elle est pratiquement superposable à celle des confusions mentales.

Les lésions élémentaires sont aussi variées que les processus étiologiques en cause et seules leurs localisations possibles présentent quelque intérêt. Il pourra s'agir de lésions corticales diffuses ou éventuellement de troubles fonctionnels corticaux diffus sans lésion anatomique décelable, et on sait qu'un dysfonctionnement cortical global a souvent été invoqué comme facteur nécessaire dans la genèse de la confusion mentale, celle-ci comportant comme séquelle une amnésie lacunaire.

Parmi les lésions corticales plus localisées, il faut retenir les atteintes frontales, qui engendrent volontiers des troubles de l'attention conditionnant secondairement une amnésie de fixation, et les atteintes pariéto occipitales qui s'accompagnent assez souvent, dans leur phase aiguë, de confusion mentale et d'amnésie de fixation résiduelle.

Dans les étages sous-corticaux, deux sièges sont à retenir:

— en premier lieu, le thalamus dont les lésions engendrent parfois des con-



Fig. 16

fusions mentales, probablement par l'intermédiaire d'une altération des mécanismes de vigilance et sans que l'on connaisse leur localisation exacte ni la nécessité ou non d'une lésion bilatérale; ce fait est bien connu dans les tumeurs thalamiques et nous avons vu que la confusion vient parfois se superposer au syndrome de Korsakoff au point d'en masquer l'existence; de même les démences thalamiques d'origine vasculaire, individualisées par Schuster puis par Grünthal, comportent habituellement une confusion mentale et des troubles de la mémoire de fixation, comme l'ont montré les observations d'Angelergues, Ajuriaguerra et Hècaen et de Castaigne et Coll. (observation 2); la lésion est ici un ramollissement thalamique postérieur bilatéral ne touchant pas directement le circuit de Papez mais détruisant divers noyaux intra-laminaires.

— en second lieu, le pédoncule cérébral, dont le rôle dans le déclenchement d'une confusion mentale et d'une amnésie lacunaire résiduelle est connu depuis l'encéphalite de Von Economo, où hypersomnie et confusion étaient des signes majeurs, et depuis les travaux sur l'hallucinoïse pédonculaire de Jean Lhermitte<sup>24, 25</sup>. Un cas personnel inédit illustre ce rôle du pédoncule: il s'agissait d'une confusion mentale fébrile, durable, liée à un petit tubercule du toit du pédoncule, sans aucune autre lésion histologique encéphalique (fig. 14).

L'imprécision et le caractère fragmentaire de ces localisations soulignent bien notre manque de connaissances sur la structure exacte des mécanismes régissant les fonctions de vigilance, dont le fléchissement engendre la confusion mentale et éventuellement l'amnésie lacunaire résiduelle. Il est vraisemblable que ces structures sont complexes et siègent à des étages divers, qu'elles comportent des interactions multiples qui expliquent et la multiplicité des localisations et les discussions qui en découlent. Il n'existe, en tous cas, aucun argument actuel pour y intégrer le circuit de Papez et pour lui faire jouer un rôle préférentiel dans la genèse des amnésies de fixation vraie.

## C — PATHOLOGIE DES AMNESIES DEMENTIELLES<sup>10</sup>

Ces amnésies se rencontrent dans les processus démentiels séniles et leur pathologie est parfaitement connue mais généralement trop diffuse pour permettre des conclusions valables sur le plan topographique.

L'étude pathologique doit être envisagée séparément pour les 2 types cliniques principaux de démence sénile: presbyophrénie et démence globale.

La presbyophrénie réalise cliniquement l'association d'un syndrome de Korsakoff typique et d'une détérioration démentielle. Anatomiquement, il s'agit habituellement de démence sénile pure ou dégénérative et exceptionnellement de démence vasculaire pure. La démence sénile pure comporte les lésions élémentaires classiques associant un semis de plaques séniles corticales diffuses et des lésions cellulaires de dégénérescence neuro-fibrillaire (D.N.F.) et de dégénérescence granulo-vasculaire (D.G.V.), localisées dans la corne d'Ammon, une prédominance franche des lésions dans T5 sous forme de nombreuses plaques séniles et enfin la présence constante de plaques séniles en nombre variable dans les 2 corps mamilaires (fig. 15 et 16).

Des rares cas de démence vasculaire pure, certains sont ininterprétables faute de renseignements anatomiques précis (cas de Fischer cités par Rouby); les autres ont été analysés précédemment dans le chapitre des syndromes de Korsakoff d'étiologie vasculaire.

La démence sénile globale comporte une détérioration mentale profonde et une amnésie globale, où tout a sombré y compris la mémoire des faits anciens. Anatomiquement, il s'agit soit de démence sénile pure, soit de démence mixte, vasculaire et dégénérative. Dans tous les cas, les lésions de type dégénératif (P.S. dans le cortex; P.S., D.N.F. et D.G.V. dans la corne d'Ammon) sont diffuses, mais, là encore, on trouve un renforcement des plaques séniles dans la circonvolution temporale interne et

la présence de plaques séniles dans les corps mamillaires.

Il n'existe donc pas de différence anatomique fondamentale entre les 2 processus et, dans les 2 cas, on trouve un renforcement des lésions dans les portions corticales du circuit de Papez et dans les corps mamillaires. Ce fait s'explique si l'on reconsidère la pathologie du syndrome de Korsakoff proprement dit. En effet une lésion bilatérale du circuit de Papez engendre un syndrome de Korsakoff. Dans sa forme la plus pure, le syndrome de Korsakoff alcoolique dépend d'une atteinte bilatérale des corps mamillaires. A côté de cette forme pure, il existe, chez les alcooliques, des syndromes de Korsakoff avec détérioration démentielle qui associent anatomiquement, à la lésion bi-mamillaire responsable du trouble mnésique, des lésions corticales de types divers, parfois vasculaires, expliquant la détérioration surajoutée. Un processus analogue semble intervenir dans les démences séniles, où l'atteinte de T5 et des corps mamillaires paraît constante; si les lésions sont d'intensité modérée, il en résulte le tableau de la presbyophrénie; si les lésions sont plus intenses, on observera une démence globale, qui empêchera l'extériorisation d'éléments korsakowiens potentiels sous-jacents; l'appréciation de cette intensité lésionnelle est toutefois pratiquement impossible, du fait de la

diffusion des lésions et de l'impossibilité de mesures précises de leur intensité.

\* \* \*

En conclusion de cette revue de l'anatomie pathologique des amnésies, 3 données émergent:

1. Le syndrome de Korsakoff a des lésions précises mais multiples, variant selon les étiologies diverses du syndrome. Leur topographie bien particulière conduit à la notion d'un circuit limbo-limbique centré par les corps mamillaires, circuit dont la lésion bilatérale, symétrique ou non, engendre le syndrome et dont l'intégrité bilatérale est nécessaire au bon fonctionnement de la mémoire des faits récents.

2. Les amnésies lacunaires ou de fixation ont une pathologie encore obscure, probablement superposable à celle des confusions mentales et comportant vraisemblablement des lésions de sièges divers: corticales diffuses ou plus ou moins localisées, thalamiques ou pédonculaires.

3. Les amnésies des démences séniles ont une pathologie bien connue mais diffuse, englobant, au même titre que d'autres régions corticales, les circonvolutions limbiques, et touchant également les corps mamillaires, mais ne permettant pas de distinction anatomique absolue entre la presbyophrénie, associant amnésie de type korsakowien et démence, et la démence globale avec troubles mnésiques diffus.

## R É S U M É

Nos connaissances de l'anatomie pathologique des amnésies sont de qualité inégale.

1. Les amnésies lacunaires ou de fixation vraie ont une pathologie peu connue, probablement superposable à celle de la confusion mentale et dépendant vraisemblablement de lésions diverses: corticales, thalamiques ou pédonculaires.

2. Le syndrome de Korsakoff ou amnésie de mémorisation a des lésions très précises mais variables, dans leur siège et leur type, selon l'étiologie en cause: lésions mamillaires des formes carentielles; com-

pression des corps mamillaires, des fornix, des thalamus internes ou des régions frontales internes dans les processus tumoraux; ramollissements symétriques bi-occipito-temporaux, bitemporaux ou bifrontaux, et très rarement lésions vasculaires asymétriques mais toujours bilatérales dans les étiologies vasculaires; lésions biammoniques ou bicingulaires dans les formes dites neuro-chirurgicales; lésions diverses temporales ou thalamiques dans quelques étiologies exceptionnelles. La comparaison de ces topographies lésionnelles a abouti à la notion d'un circuit limbo-limbique centré



par les corps mamillaires, circuit dont la lésion bilatérale, symétrique ou non, engendre le syndrome et dont l'intégrité bilatérale est nécessaire au bon fonctionnement de la mémoire des faits récents.

3. Les amnésies des démences séniles ont une pathologie corticale diffuse, touchant, entre autres, les circonvolutions lim-

biques et les corps mamillaires. Ces lésions sont sensiblement équivalentes dans les formes presbyophréniques à symptomatologie korsakowienne prédominante et dans les formes globalement amnésiques, qui sont, de ce fait, impossibles à différencier sur le plan anatomique.

## RESUMEN

Nuestros conocimientos acerca de la anatomía patológica de las amnesias son desiguales.

1. Las amnesias lacunares o de fijación verdadera tienen una patología poco conocida, que puede superponerse a la de la confusión mental y que depende en realidad de lesiones diversas: corticales, talámicas o pedunculares.

2. El síndrome de Korsakoff o amnesia de memorización responde a lesiones bien definidas pero variables en cuanto a su localización y a su naturaleza de acuerdo a la etiología en cuestión: lesiones mamilares con formas carenciales; compresión de los cuerpos mamilares, de los fornix, de los tálamos internos o de las regiones frontales internas que sufren procesos tumorales; reblandecimientos simétricos bi-occipito-temporales o bifrontales, y muy rara vez lesiones vasculares asimétricas pero siempre bilaterales en las etiologías vasculares; lesiones biamónicas o bicingulares

en las formas llamadas neuroquirúrgicas; lesiones diversas temporales o talámicas en algunas etiologías excepcionales. La comparación de estas topografías lesionales ha culminado con la noción de un circuito limbo-límbico centrado en los cuerpos mamilares; la lesión bilateral de este circuito, simétrica o no, engendra el síndrome ya que su integridad bilateral es necesaria para el buen funcionamiento de la memoria para hechos recientes.

3. Las amnesias de las demencias seniles tienen una patología cortical difusa, que implica entre otras cosas las circunvoluciones límbicas y los cuerpos mamilares. Estas lesiones son llamativamente equivalentes en las formas presbiofrénicas con sintomatología similar a la del síndrome de Korsakoff predominante y en las formas amnésicas globales, que de hecho son imposibles de diferenciar en el plano anatómico.

## SUMMARY

Our knowledge concerning anatomopathology of amnesia is not uniform.

1. Lacunar amnesias or amnesias of real fixation have a not very well known pathology, which might be similar to pathology of mental confusion and which depends on different kinds of lesions: cortical, thalamic or peduncular.

2. The syndrome of Korsakoff or memorization amnesia is due to well defined lesions which may vary in their localization and in their nature according to their

aetiology: mamillar lesions in their carential forms; compression of corpi mamillari, fornix, intern thalami or of inner frontal regions in tumoral processes; symmetrical bi-occipito-temporal, bitemporal or bifrontal softening, and hardly ever, assymetrical vascular lesions, but always bilateral in vascular aetiologies; biamonic or bicingular lesions in the so called neurosurgical forms; diverse temporal or thalamic lesions in some exceptional aetiologies. Comparison of these lesional topographies has lead to

the notion of a limbo-limbic circuit in the corpi mamillari; bilateral lesion of this circuit, symmetrical or not, causes the syndrome as bilateral integrity is necessary for good functioning of memory for recent facts.

3. Amnesias of dementia senilis have a diffuse cortical pathology which affects,

among others, limbic convolutions and corpi mamillari. These lesions are surprisingly equivalent to presbyophrenic forms with a similar symptomatology to the one of the dominant syndrome of Korsakoff and in global amnesic forms, which in fact are impossible to be differentiated in the anatomical sense.

## ZUSAMMENFASSUNG

Unsere pathologisch-anatomischen Kenntnisse ueber die Amnesie sind ungleichmaessig:

1. Die lakunaeren Amnesien oder die Amnesien mit wirklicher Fixation haben eine wenig bekannte Anatomie, die wahrscheinlich mit der der mentalen Konfusion identisch und wahrscheinlich von verschieden gelagerten Laesionen abhaengig ist, die cortical, thalamisch oder pedunculaer sein koennen.

2. Das Korsakoffsche Syndrom oder die Erinnerungsamnesie ist durch sehr praezise aber verschiedenartige Laesionen bedingt, sowohl bezueglich ihres Sitzes als auch des Typs, je nach der Aetiologie: mamillaere Laesionen bei den carentiellen Formen; Kompression der corpi mamillari, des Fornix, der Thalami interni oder der internen frontalen Regionen bei tumoralen Prozessen; symmetrische bi-occipito-temporale, bitemporale oder bifrontale Erweichungsherde und sehr selten asymmetrische aber immer bilaterale vasculaere Laesionen bei der vasculaeren Aetiologie;

biammonaere oder bicingulaere Laesionen bei den sogenannten neuro-chirurgischen Formen; verschiedene temporale oder thalamische Laesionen bei einigen Formen aussergewoehnlicher Aetiologie. Der Vergleich der Topographie dieser Laesionen hat zum Begriff eines limbo-limbischen Kreislaufes gefuehrt, dessen Zentrum die corpi mamillari sind und bei dem die bilaterale Laesion, ob sie nun symmetrisch ist oder nicht, das Syndrom entstehen laesst, und dessen bilaterale Integritaet notwendig ist, damit die Erinnerung an juengst vergangene Dinge gut funktioniert.

3. Die Amnesien der Dementia senilis haben eine diffuse corticale Pathologie, die, unter anderem, die limbischen Zirkumvolutionen und die mamillaere Koerper betrifft. Diese Laesionen sind weitgehend gleichwertig bei den presbyophrenischen Formen und vorwiegend Korsakoffaehnlicher Symptomathologie und bei den global amnestischen Formen, die in diesem Falle nicht auf dem Gebiete der Anatomie zu differenzieren sind.

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# Clinical and Psychometrical Study of a Patient with Memory Disturbances

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All kind of knowledge is acquired, and vacuity of information of a new born baby is opposed to the acquisition of a series of cognitive elements.

This is possible thanks to memory, that aptitude of the brain to memorize, to keep and rememorate information.

Memory is not an independent faculty, it is closely related to function of the different cerebral activities. It is therefore difficult to study it isolated from them, but it is possible to study each one of the activities from the viewpoint of memory analyzing in what way it has been apprehended, kept and rememorated.

The product of the meeting of the child with the outer world is characterized by the elaboration in his brain of a certain degree of order which reflects — in all the stages of life — the experience which bore it at some level of the considered scale.

It is possible to conceive that there exists a code between what every individual knows about the outer world and the organization of a functional structure in his brain. This progressively elaborated functional structure is the duplicate of that experience, the support of its cognitive contents; it is to that experience that the brain will always refer the data of the world where it acts, which will only be recognized through that same experience.

Every disturbance — localized or diffuse — of the brain will be reflected on the cognitive contents, which will not only

diminish, but will also see its possibilities of expanding and getting richer diminish.

We conceive that the balance of a subject with memory disturbances includes necessarily the evaluation of the damage of his ancient knowledge, of his mnesic stock, and also the appreciation of the possibility to learn and keep new information.

The memory disturbances may be the major symptom in some cases; in other cases it may be one element of a more complex deficit. In all the cases the examination of the patient should be practical and should follow a certain order which should always be the same.

In this way it is possible:

- 1) To make an exact balance of the stated mnesic disturbances by studying knowledge and remembrances acquired in the past and by appreciating the possibilities of learning and keeping new data.
- 2) To follow their evolution by comparing successive balances.
- 3) To compare with efficacy this observation to others performed in the same conditions of examination.

## I. STUDY OF WHAT THE PATIENT HAS KEPT FROM THE PAST

Can the cognitive contents of anybody be evaluated?

This would theoretically imply to know all the circumstances of the subject's life from the day he was born, which is of course impossible. The doctor has to limit himself to an inquiry with the purpose of learning if the patient has conserved a certain amount of information of the past which he should have acquired. One is therefore lead to apply two kinds of tests:

1) Standardized test on general notions (Wechsler-Bellevue type) in which a certain amount of verbal, praxic, conceptual and cultural knowledge is analyzed and compared with the knowledge contained in large homogeneous series statistically established.

This allows to bring to evidence global or partial deficits. When the results are inferior to the average of the equivalent category, it is possible to determine the impoverishment of knowledge acquired in the past.

2) Test adapted to the subject considering his own past. The best way of studying the past of a subject is to ask him on the development of his own life. A systematical examination stressing particularly the different periods of life in which the subject has been deeply impressed due to practical or affective reasons (first remembrances of school life, first contacts with the teacher, circumstances in which he met his future life partner, first days of military service, etc.). These successive stages are carefully followed by the inquiry and allow to obtain a "history of life" that constitutes a very interesting document on the memory of past facts.

After analyzing this account it is possible to state precisely:

— Exuberance or scarcity of remembrances.

— Characteristics of the account: spontaneous evocation following chronological order or otherwise bits of remembrances chaotically evoked, incapacity of a spontaneous account.

— If the disturbance of remembering concerns specially *evocation*: the remembrance appears after whispering, or after

putting the subject in the situation. Or if the disturbance affects *recognition*.

In this case the patient does not recognize as him own the remembrance of a fact of his past life.

— Chronology of memory disturbances.

Sometimes they affect the whole past and only some mnestic rests subsist concerning certain remembrances of childhood or certain events that particularly impressed the patient.

In some cases memory disturbances respect a part of the past.

The extension of the proved gaps may vary and may cover a few minutes in some cases, several days in others and even years in others. They are the consequence of two different — sometimes associated — mechanisms:

In certain cases they are related to a memory defect related to gradual forgetting which leads to anterograd amnesia. It is most evident that in these cases the patient is unable to evoke a period in which he has not fixed anything.

In other cases there are perfectly well remembered events, remembered some time before, that gradually disappear. These are the cases of organic dementias, in which the cognitive contents disappear progressively. A particular kind of retrograd amnesia may be observed in patients with cranial traumatism. It affects a few minutes — sometimes a few hours — preceding the accident. This may find an explanation in the fact that the events taking place during this period cannot be remembered — and as a consequence could not have been fixed — due to coma.

The existence of some thematical gaps affecting electively certain events while other simultaneous events are fixed, can sometimes also be proved. Conscious or unconscious motivations allowing such a scotomisation have to be investigated.

— Behavior of the patient towards his memory defect: recognition of the mnestic disturbance, indifference to it or ignorance of the same with compensatory phenomena: fabulation and false recognition.

## II. STUDY OF ACQUISITION AND CONSERVATION OF NEW INFORMATION

### 1) Clinical tests

Many tests can be imagined and we use the following:

a) *Test of the bidden object.* A patient is shown an object he has to describe which is afterwards hidden while he is present in his room. After 10 minutes, one hour and 24 hours he is asked to tell the name of the object and the place where it is hidden.

b) *Test of the watch.* A watch is presented to the patient while he is told the mark of the watch, where and when it has been bought, its price and the name of its owner. The watch is then presented to the patient after 10 minutes, one hour and 24 hours and the data he kept are studied.

These tests or even the retention of a sentence model or learning the names of physicians and nurses constitute a first approach to judge the ease of retention but they are not sufficient to quantify the importance of the trouble and to analyze its chief modalities. As a matter of fact, a thorough balance of the mnestic disturbances is necessary, carried out during a rather long time and demanding the cooperation of trained physicians and psychologists.

### 2) Psychometrical tests

Numerous tests used in psychology can be useful to study memory; differed evoking of certain visual retention tests can also be studied: Benton Test Bender; Figure of Rey.

It is important to have a battery of memory tests containing the different perspectives in which memory is exerted:

Learning of visual and verbal material and their retention during shorter or longer periods of time.

Among these relatively few test batteries some have been standardized on a normal population: the Wechsler memory scale<sup>3</sup> and the "Profil de rendement mnésique"

("Profile of mnestic efficiency") of André Rey<sup>1</sup>. We shall remind the reader about the principles indicating the precise descriptions given by the authors.

The *WECHSLER MEMORY SCALE* contains 7 sub-tests covering different sectors:

Simple questions of *information* and *orientation*

Tests of *mental control* (counting from 20 to 0; 3 by 3)

Test of *logical memory* (repeating of a story stressing the retained ideas)

Test of *immediate memory* (repetition of numbers)

Test of *reproduction of illustrations*

Tests of *learning of words*; (the task consists in associating 2 words):

"easy" words belonging to the same semantic field:

money — gold                      baby — cries

"difficult" words: cabbage — feather

This test stresses the immediate apprehension and retention of verbal and visual material. It has the purpose of being directly comparable to the I.Q. obtained with the Wechsler-Bellevue scale. It has been devised to allow the comparison of what concerns a memory disturbance and what concerns defects of other intellectual functions.

But this test is limited to the study of immediate evocation because it does not include differed evocations in the time span. Learning acquired during a restricted period is not opposed to forgetting.

The "*Profile of mnestic efficiency*" of Rey (P.M.E.) contains a series of drawings whose design gets more and more complex up to those where no identification is possible and where only remembering allows to find the drawing. This test allows to analyze a certain number of factors:

— a factor of structuring of data (it is necessary to understand the successive simplifications of the same picture).

— a mnestic factor: the remembrance of the first perception helps to the recognition of the data.



Besides it allows to study the importance between recognition and evocation, the rate of evocation, the phenomenon of retroactive inhibition.

Nevertheless we point out:

- its timely limited profit (one single differed evocation takes place 15 minutes after the first evocation).
- The fact that its author has the purpose of studying the mnestic efficiency once the disturbance of symbolic functions has been eliminated: aphasia and agnosia.

*The BATTERY OF MEMORY TESTS* we use at the Albert Chenevier Hospital has the purpose of replacing the study of memory in the systematic study of intellectual functions.

Which are the possibilities of a subject to increase his cognitive capital? How can his shortcomings in this aspect be analyzed?

In this sense we have privileged certain aspects:

— In first place our tests contain differed evocations; the maximum between the first and last evocation is of one week. Other investigations performed after one hour and 24 hours allow to establish a *forgetting curve*.

— We have tried to cover the main fields of intellectual activity: therefore the mnestic possibilities are always analyzed in connection with a specific aspect (language; praxognosias).

— Finally we have analyzed independently: immediate memory ability, immediate evocation, learning, retention.

Some tests in our battery are classic: Span I, words of Rey. Others have been devised in our service. The latter have been tried on normal subjects; but our standardization is at present still limited to some ages and some cultural levels. This is why we are giving them as a reference and not as absolute criteria. Furthermore, we are only able to give the averages, without index of dispersion.

*Tests used:*

We shall only enumerate them, their

description, their sequence and results *being indicated separately*.

## 1) Tests with verbal responses

*Auditive entry:* Span I; Span I bis; Words of Rey; Story.

*Visual and auditive entry:* Kim test.

## 2) Test with motor responses

*Visual entry:* Test of 7/24.

## Analysis of Different Aspects in Memory

We have enumerated the different operations participating in the acquisition of information, no matter of its nature (verbal, praxognosic). Four questions may arise:

1 — When a limited number of information is presented once to a subject, how much from it can he keep simultaneously and errorless? This maximum, determined by the subject, defines the *ability of immediate memory*.

2 — In case of a *non limited amount of information*, how much of it does the subject keep after one presentation? This amount may vary; it depends on the proposed quantity of information and of the time during which it is presented. In this test a real structuring and a reorganization of the whole material play an important role.

We group these multiple tasks under the name of *immediate evocation*.

3 — How can the subject improve his initial apprehension by new material presentations. We distinguish two modalities in this *learning*:

— partial learning where the number of repetitions is limited; the obtained performance is the measure of the learning.

— a total learning up to 100, where only the number of repetitions varies and where the required performance is constant.

4 — How long does this information persist? Basing on successive discharge of

formerly presented material, we establish a study of retention (forgetting curve).

A new classification of the used tests can be given following these four aspects:

|                             |                                                       |
|-----------------------------|-------------------------------------------------------|
| Ability of immediate memory | — Span I (immediate verbal memory)<br>— Visual span * |
| Immediate evocation         | — Kim<br>— Story<br>First evocation                   |
| Learning                    | — Span I bis; words of Rey<br>— 7/24                  |
| Retention                   | — Kim<br>— Story<br>Forgetting curve                  |

These tests allow to study simultaneously the way in which new data are apprehended when the task belongs to the verbal or visual field and also the way in which they are kept by the subject.

a) The study of ability of immediate memory, of immediate evocation and of learning allow to analyze and quantify the way in which the subject apprehends and fixes new data in memory.

They constitute also a new way of abording the instrumental possibilities: verbal or practognosic playing a role in these tests.

It is interesting to point out that these tests allow to bring to evidence very slight instrumental disturbances and that they particularly allow to appreciate the existence of latent aphasia.

While most of the complex tests necessary to detect a slight language deficit involve cultural factors or conceptual possibilities going beyond the strict field of language, verbal memory tests, accessible to everybody are very sensitive to discrete verbal deficit.

Immediate evocation of a lowered history, limited verbal learning have appeared to us as the first signs of a minimal verbal deficit or have objectivated the last sequelae of aphasia.

b) No matter of the modality — excellent or mediocre of memory, learning curves and specially retention curves allow

to follow the flow of these acquisitions: their retention or their eventual forgetting.

Using this battery of memory tests in patients with different types of brain lesions it is stated that many tables can be performed.

Summarily it can be stated:

a) an immediate memory deficit and possibilities of immediate evocation, slowness and mediocrity of learning are associated, following the rule, to deficit of function in cases of evident or latent aphasia and also in apraxignosia. These instrumental amnesias are closely connected with the damage of functions.

If the apprehension of new data is poor, retention of this hardly acquired material is generally good. These features define *amnesia of cortical origin*.

b) The relative conservation of ability of immediate memory opposed to the existence of gradual forgetting parallel to damage of learning and retention are hardly ever observed. This formula characterizes *amnesia of hippocampo-mamillar origin*, observed when a bilateral lesion interrupts the Papez circuit.

\* The evocation following the first presentation of board VII of 7/24 we have used as a mesure of the ability of immediate visual memory revealed itself rather undiscriminative. Other tests, specially those using tachytoscope may be used.

c) More frequent is the simultaneous damage of immediate memory, of immediate evocation, of learning and of retention characterizing *global amnesia* which, according to the distribution of the lesions may be of cortical or hippocampomamillar dominance and evidenced by the particular importance of gradual forgetting. Tests allow to appreciate its degree.

Psychiatric examination and personality tests will often be important complements of this investigation. It is necessary to point out that on the contrary the appliance of a mnestic test battery allows to establish the proof of organic origin of a complex mental syndrome in certain cases, particularly in the appreciating of sequelae of a patient with cranial traumatism.

Systematic examination of the mnestic possibilities following a well established protocole is thus inscribed in the study of language and praxognosias, completing the study of higher functions.

#### DESCRIPTION OF THE TESTS OF THE "MEMORY BATTERY" USED AT THE "CENTRE DE REEDUCATION DE LA MEMOIRE ET DU LANGAGE DE L'HOPITAL ALBERT CHENEVIER"

##### 1) Tests of verbal response Auditive entry

###### — *Span I* (Number test)

Ability of verbal immediate memory.  
This test consists in telling the patient to repeat lists of numbers of increasing length.

Ability of immediate memory is the amount of numbers the subject is able to repeat without alteration immediately after hearing them.

*Norms:* Obtained results in a group of 105 Medical students of an age ranging between 18 and 24 years.  
Average.....: 6,40  
(sigma: 1,27)  
Obtained results in a group of 51 normal 65 year old subjects:  
Average.....: 4,77

###### — *Span I bis*

Repetition of an increasing amount of numbers in which each evocation contains only one new number.

Example: 9,2 - 9,2,7 - 9,2,7,4 - etc...

The obtained score has to be slightly higher to the one of the ability of immediate memory because there is a progressive learning from one series to the following.

*Norms:* In a group of 91 Medical students:

88 have given sufficiently grouped results to give the average value.

Average.....: 9,06

(sigma: 2,12)

51 subjects over 65 year old:

Average.....: 5,87

###### — *Story*

A story containing 22 elements is read. The subject has to evoke it immediately, and also after one hour, 24 hours and a week without helping him to remember. There are parallel stories containing the same number of elements and allowing retest. We are giving two as an example.

*Story of the Lion:* A lion, / called Sultan, / escapes from his cage, / through the door / left unlocked by the careless guardian, / the numerous crowd of visitors / on that sunday / runs to the near buildings. / A woman, dressed in blue / drops her / one year old baby / she carried, / in her arms. / The lion takes him. / The woman comes back / and crying, / implores the lion to give her baby back / the animal stares / at her / a long time. Finally, he let the baby go / without hurting it.

*Story of Pauline Dubois:* Pauline Dubois / servant / at the S.N.C.F. / 38 year old. / came home / in the subway / on friday night / at Bastille station / a little / man / wearing glasses / takes away her bag / containing 200 francs / deprived of her money / she has not been able to pay her rent / and she became afraid of being evicted / with her three children, / Her friends / and mates / have contributed / and have given her / the collected 250 francs.



**Instruction:** I will tell you a story, pay attention to it; then you will repeat it to me as exactly as possible.

**Annotation:** includes:

The number of elements evoked at each discharge.

The time in which each evocation takes place.

**Norms:**

- 1) Medical Students:  
Average of immediate evocation:  
14.2
- 2) Normal subjects from 50 to 64 years of age (N.C. 2,3,4)  
Immediate 1 hour 24 hours 1 week  
11,8 11 10,9 10,8
- 3) Normal subjects over 65 years old (N.C. 2,3,4)  
9,5 9,07 9,1 8,4

— Words of Rey

This test is taken from André Rey.<sup>2</sup>

- 1 — A list of words is read 5 times to the subject, and is followed each time by evocation. Here are some proposed lists.

|          |           |           |
|----------|-----------|-----------|
| Violin   | Cousin    | Banana    |
| Tree     | Ear       | Tool      |
| Tie      | Knife     | Hunter    |
| Ham      | Staircase | Bucket    |
| Suitcase | Dog       | Champagne |

**Instruction:** I shall read you a list of words. Pay attention and when I finish you shall tell me all the words you remember. It is not necessary to remember them in the same order, you will tell them just as they come to your mind. You have to tell me as much as possible.

**Annotation:**

Correct words

Wrong words

Double words (repeated twice)

Time: Evocation in 1 minute with a mark every 10"

- 2 — I will once more read the same words and when I finish, you will repeat all the words you know; the first time you told me ... words; you will now be able to tell me more;

you will repeat those from the first time and add some new.

Same annotation.

Time: 1'30"

This procedure is repeated up to a total of 5 evocations.

### 3 — Recognition test

A story containing all the words of the list is read to the subject, who has to recognize them as he hears them. Here is the story corresponding to the list of words:

The dog (1) of the blind musician (2) who played the violin (3) near the staircase (4) of the bridge (5) kept his master's (6) dinner (7) and bread (8) and ham (9) in the suitcase (10) standing behind the wall (11) among the pebbles (12), and old tools (13); banana (14) peel (15) and rusty buckets (16). He raised his ears (17) and showed his teeth (18) while he saw far away in the field (19) near the big tree (20) of the forest (21) both cousins (22) of the hunter (23) who, the gun (24) on the shoulder (25) and a knife (26) in their hand (27) approached without a hat (28) and without a tie (29) singing a song (30).

**Annotate:** the number of recognized words.

**Norms:** (given by a Rey)

Note: total of 5 evocations.

### A. Young adults

| Centiles | Manual | Intellect. | Students |
|----------|--------|------------|----------|
| 10       | 47     | 53         | 53       |
| 25       | 50     | 57         | 61       |
| 50       | 56     | 63         | 65       |
| 75       | 62     | 65         | 67       |
| 100      | 66     | 69         | 73       |

### B. Aged subjects

| Centiles | primary | Intellectual |
|----------|---------|--------------|
| 10       | 22      | 24           |
| 25       | 24      | 28           |
| 50       | 36      | 42           |
| 75       | 42      | 48           |
| 100      | 52      | 68           |

**2) Tests with verbal response****Visual and auditive entry****— KIM**

20 familiar objects are presented on a tray to the subject during 1'30". The subject has to name all the objects and then, once the tray has been retired, he has to evoke them.

Then these objects are once more presented to the subject while the well evoked objects are enounced and then the non-evoked ones. The retention of this material is examined after one hour, 24 hours and 1 week, without a new presentation of the material.

*Annotation:* immediately.

— The number of evoked objects after one hour, 24 hours and 1 week.

— The time needed for each evocation. (The time to consider is the one of the last evoked object).

**Norms:**

1) Normal between 50 and 64 years old (N.C. 2,3,4 )

| Immediate | 1 hour | 24 hours | 1 week |
|-----------|--------|----------|--------|
| 13,2      | 14,6   | 14       | 13     |

2) Normal over 65 years old (N.C. 2,3,4)

|      |      |      |      |
|------|------|------|------|
| 11,4 | 12,8 | 12,2 | 12,5 |
|------|------|------|------|

**3) Test with motor response****— 7/24 test**

This test consists in reproducing a form constituted by 7 chips disposed at random on a draughtboard of 24 divisions.

The model is presented to the subject and, after taking it away, he is given 9 chips and an empty draughtboard and he is told to reproduce it. (Fig. 1).

This figures is presented in sections of 10" up to a total learning. The number of presentations is limited to 15. Successive discharges (and re-learning in case of wrong reproduction) take place after delays of 5 minutes, 1/2 hour and 24 hours.

*Annotation:* for each phase.

— the number of correctly disposed chips in the first evocation (it can be used to measure the immediate memory — visual span).

— the number of presentations necessary to a total learning. If the model has not been learned after 15 presentations, a simplified board including 5 chips disposed on a similar draughtboard is presented after a week (board V); in case of failure, after another delay, a board including 3 chips (board III) is presented.

**Norms:**

Normal between 41 and 79 years old.  
Average of age = 58 years (N.C. 2,3).

| Board VII  | Evocation<br>Average<br>number | Learning<br>timely<br>average |
|------------|--------------------------------|-------------------------------|
| Immediate  | 4                              | 49"                           |
| 5 minutes  | 6,16                           | 6"                            |
| 30 seconds | 6,25                           | 4"                            |
| 24 hours   | 6,06                           | 6"                            |

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**S U M M A R Y**

As memory, far from being an isolated faculty, is closely related to different aspects of brain function, every disturbance — localized or diffuse — will act on it, not only reducing the cognitive contents but also reducing its future possibilities of expansion. It is therefore of utmost impor-

tance in the examination of patients with memory disturbances to evaluate precisely the damage of ancient knowledge, their mnesic stock, and also to appreciate the possibilities of learning and keeping new information.

In order to obtain these data the exa-

mination has to follow an invariably strict order:

1) Study of what the patient remembers from his past by using two kinds of tests:

a) Tests of the Wechsler - Bellevue type, which allow to state if the damage is global or only affects a certain function. The results of these tests measure the impoverishment of knowledge in relation to the average values.

b) Test adapted to the patient based on his own past, which allows to learn about: exuberance or scarcity of remembrances, features of his narration (spontaneous and chronologically correct evocation, bits of remembrances chaotically evoked or incapacity to give a spontaneous account), if the damage affects mainly evocation or recognition and the chronology of memory disturbances.

2) Study of Acquisition and Remembering of New Information by applying clinical and psychometrical tests. Among the psychometrical tests the authors recall the Wechsler memory scale and Rey's Profile of mnesic efficiency. Detailed information is given about a battery of tests successfully used by the authors at the "Centre de Rééducation de la Mémoire et du Langage de l'Hôpital Albert Chenevier", which replaces the study of memory within the systematic study of intellectual function. This battery used in patients with brain lesions is a valuable aid to distinguish different pathological pictures: amnesia of cortical or hippocampo-mammillar origin and global amnesia.

The paper is complemented by the description of the different tests constituting this battery and mentions the obtained results.

## RESUMEN

La memoria, lejos de ser una facultad aislada, está íntimamente relacionada con diferentes aspectos de la actividad cerebral, por lo tanto toda perturbación —localizada o difusa— actúa sobre ella reduciendo el bagaje cognitivo y reduciendo también sus futuras posibilidades de expansión. Esto hace que sea sumamente importante en el examen de los pacientes con trastornos de la memoria la evaluación exacta del deterioro de los conocimientos anteriores y la apreciación de las posibilidades de adquirir y retener información nueva.

Para obtener estos datos el examen debe ceñirse invariablemente a un orden estricto:

1) Estudio de lo que el paciente recuerda de su pasado, mediante la aplicación de dos tipos de tests:

a) Tests del tipo Wechsler-Bellevue, que permiten determinar si el deterioro es global o afecta una sola función. Los resultados de estos tests miden el empobrecimiento de los conocimientos con respecto a los valores promedio.

b) Test adaptado al paciente teniendo en cuenta su propio pasado, que permite

determinar: la riqueza o pobreza de sus recuerdos, las características de su narración (espontánea y cronológicamente correcta, trozos de recuerdo caóticamente evocados o incapacidad de hacer un relato espontáneo), si el deterioro afecta más a la evocación o al reconocimiento y la cronología de los disturbios de la memoria.

2) Estudio de la adquisición y retención de información nueva mediante la aplicación de tests clínicos y psicométricos.

Entre los tests psicométricos los autores mencionan la escala de memoria de Wechsler y el perfil de rendimiento de memoria de Rey. Dan información detallada sobre una batería de tests usada con éxito por ellos en el "Centre de Rééducation de la Mémoire et du Langage de l'Hopital Albert Chenevier", que reubica el estudio de la memoria dentro del estudio sistemático de la función intelectual. Aplicada en pacientes con lesiones cerebrales, esta batería de tests demostró ser una ayuda valiosa para distinguir diferentes cuadros: amnesia de origen cortical o hipocampo-mamilar y amnesia global.



Este trabajo es complementado por la descripción de las diferentes pruebas que

componen esta batería y contiene los resultados obtenidos.

## R É S U M É

Comme loin d'être une faculté indépendante, la mémoire est étroitement liée à de différents aspects des activités cérébrales, toute perturbation —localisée ou diffuse— retentit sur elle, non seulement en amoindrissant le bagage cognitif, mais aussi en diminuant les possibilités de sa future expansion. Il est donc extrêmement important d'évaluer avec précision dans les patients présentant des troubles de la mémoire l'atteinte de leurs connaissances anciennes, de leur stock mnésique, ainsi que d'apprécier les possibilités d'apprentissage et de retention d'informations nouvelles.

Pour obtenir ces données l'examen du patient doit suivre un protocole précis et toujours le même:

1) Etude de ce que le malade a retenu de son passé au moyen de deux sortes de questionnaires:

a) Questionnaire du type Wechsler-Bellevue, permettant de déterminer s'il s'agit d'un déficit global ou s'il porte sur une seule fonction. Les résultats de ce questionnaire mesurent l'appauvrissement des connaissances par rapport à la moyenne.

b) Questionnaire adapté au patient en tenant compte de son propre passé, qui permet de préciser: la richesse ou pauvreté

des souvenirs, les qualités du récit (évacuation spontanée et chronologiquement correcte, bribes de souvenirs évoquées chaotiquement ou bien incapacité de la construction spontanée du récit), si le trouble porte surtout sur l'évocation ou sur la reconnaissance.

2) Etude de l'acquisition et de la conservation d'informations nouvelles au moyen d'épreuves cliniques et psychométriques.

Parmi les épreuves psychométriques les auteurs rappellent l'échelle de mémoire de Wechsler et le profil de rendements mnésiques de Rey. Ils donnent une information détaillée sur une batterie d'épreuves appliquée par eux avec succès au "Centre de Rééducation de la Mémoire et du Langage de l'Hôpital Albert Chenevier", qui replace l'étude de la mémoire dans l'étude systématique des fonctions intellectuelles. Cette batterie s'est révélée comme aide efficace chez des patients atteints de divers types de lésions cérébrales pour distinguer: l'amnésie de memoration d'origine corticale ou hippocampo-mamillaire et l'amnésie globale.

Le travail est complété par un annexe contenant la description des différentes épreuves ainsi que les résultats obtenus.

## Z U S A M M E N F A S S U N G

Da das Gedächtnis nicht nur keine isolierte Fähigkeit ist, sondern eng verbunden mit den verschiedenen Aspekten der Hirnfunktion, wird jede lokalisierte oder diffuse Störung auf diese ihre Wirkung ausüben. Dabei wird nicht nur der cognitive Inhalt reduziert sondern auch die künftigen Möglichkeiten einer Erweiterung des Gedächtnisses. Es ist deshalb bei der Untersuchung von Patienten mit Gedächtnisstörungen äußerst wichtig,

genau den Schaden an alten Kenntnissen zu bestimmen und auch die Möglichkeiten des Lernens und des Behaltens neuer Information abzuschätzen.

Um diese Daten zu erhalten, muss die Untersuchung nach einer strikten und unveränderlichen Ordnung vor sich gehen:

1) Die Untersuchung dessen, an was der Patient sich aus der Vergangenheit erinnert, unter Zuhilfnahme von zwei Arten von Tests:

a) Tests vom Typ Wechsler-Bellevue, welcher erlaubt festzustellen ob der Schaden global ist oder nur eine gewisse Funktion umfasst. Die Resultate dieser Tests zeigen den Grad der Verarmung an Kenntnissen im Verhaeltnis zu Durchschnittswerten.

b) Einen zum Patienten passenden Test, der sich auf seine eigene Vergangenheit stuetzt und welcher erlaubt zu erfahren: ueber die Fuelle oder Armut an Gedaechnissbildern, Zuege aus seiner Erzaehlung (die spontane und chronologisch Korrekte Erinnerung, Stuecke von chaotisch hervorgerufenen Gedaechnis oder die Unfaehigkeit eine spontane Erzaehlung auszufuehren), wenn der Schaden die Erinnerungsstoerungen.

2) Das Studium der Erwerbung und der Erinnerung neuer Information mit Hilfe von klinischen und psychometrischen Tests. Unter den psychometrischen Tests

erinnern die Autoren an die Wechslersche Erinnerungsskala und Rey's Profil der mnestischen Effizienz. Es wird ins einzelne informiert ueber die Test-Serie die mit Erfolg von den Autoren benutzt worden ist im "Centre de Rééducation de la Mémoire et du Langage de l'Hôpital Albert Chenevier", welches verdraengt hat das Studium der Erinnerung innerhalb einer systematischen Untersuchung der intellektuellen Funktion. Diese "Batterie" wird bei Patienten mit Hirnverletzungen benutzt und ist eine wertvolle Hilfe um verschiedene pathologische Bilder zu unterscheiden: Amnesia kortikalen oder hippocampomamillaeren Ursprungs und die globale Amnesie.

Die Arbeit ist erweitert durch die Beschreibung der verschiedenen Tests dieser "Batterie" und die Besprechung der Resultate.

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# The Traumatic Amnesias

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One of the characteristic features of cerebral concussion of either slight or severe degree, is that the trauma causes a disturbance of consciousness. Loss of awareness of the environment is therefore common after most types of head injury, and it is evident that during this period of confusion events which occur are subsequently not remembered. In this regard the effects of *commotio cerebri* resemble the effects of anaesthesia, electric convulsion therapy (E.C.T.), or any illness such as meningitis or subarachnoid haemorrhage which impairs brain mechanisms. It is not necessary for the patient to be comatose for memory mechanisms to be blocked, for there may be comparatively normal behaviour and activity after concussion, say during a game of football, and yet the player after recovery finds a gap in his memory for perhaps some hours following the injury.

Modern research techniques on the forces which disturb the brain during closed head injury indicate that when the skull is subjected to high accelerations, often amounting to 300 or 400 G for a few milliseconds, the brain is subjected to distorting waves of pressure which cause areas of displacement one on another, which must clearly cause substantial damage to nerve cell processes at the time. These distorting effects are more severe where the brain is most bulky, in the cerebral hemispheres. It is often assumed that injury to certain areas of the brain-stem causes the unconsciousness in concussion, but it

is just as likely that the widespread damage to the large areas of the cerebral hemispheres will lead to generalised suppression of brain mechanisms, not only in the cerebral hemispheres but also in the brain-stem. It is reasonable, therefore, to look on the common type of head injury, which may be referred to as *acceleration concussion*<sup>1</sup>, as causing a widespread interference with function in all parts of the brain due to direct distorting forces affecting a vast number of cerebral neurones.

## Post-traumatic amnesia (P.T.A.)

One of the characteristics of damage to any part of the nervous system, whether it be peripheral nerve, spinal cord, or brain, is that the severity of the injury can often be related to the time taken for function to return to normal<sup>6</sup>. Thus, as far as the brain is concerned, the duration of disturbed function provides important evidence regarding the severity of concussive injuries, and as disturbed function of the brain is associated with an inability to remember current events, then in retrospect the duration of "memory gap" after the time of the injury provides useful evidence regarding the severity of brain injury<sup>7</sup>. Thus, even long after an injury, the patient will usually be able to indicate that he recovered awareness of his environment a few hours, or a few days or weeks after the date of the injury, and that since this



"waking up" his memory of current events has been satisfactory and sequential<sup>8</sup>.

In the handling of patients recovering from acceleration concussion therefore, it is important to identify the time at which the patient becomes fully aware of his environment and is storing current events for future use. The degree to which consciousness has recovered does not necessarily emerge clearly from casual conversation and it is important to ask the patients about events during the preceding hours, for it is only when current events are being stored for future use that the period of post-traumatic amnesia has come to an end. In uncomplicated cases, once full awareness of the environment has returned recovery proceeds uneventfully, but should any complication of the head injury develop, then there may, after a lucid interval which is later remembered, be a subsequent period of confusion with amnesia which is due to the complication. It is of course well known that repeated assessment of the degree of consciousness provides an essential part of the care of any recovering case of head injury in relation to the recognition of dangerous developments such as cerebral compression by haemorrhage.

During the period of confusion, not only are new memories not established, but the recall of old memories may be very faulty, particularly of the more recent events before the injury. Thus a recalling mechanism may be vigorous but inaccurate and wild confabulation and even false accusations with regard to the injury may appear<sup>9</sup>.

Penetrating injuries to the skull and brain are very different from the closed head injuries of the acceleration concussion type, from the point of view of the resulting loss of consciousness and amnesia. War wounds illustrate the differences very clearly. These penetrating wounds cut into the cerebral hemispheres and often cause extensive areas of brain destruction with perhaps an immediate hemiplegia, aphasia or hemianopia, without necessarily causing

any impairment of consciousness. Thus 25 per cent of soldiers with a penetrating brain wound<sup>10</sup> remained fully conscious and had no traumatic amnesia. In this instance the head is not subjected to any violent accelerations.

### **Retrograde amnesia (R.A.)**

After recovery from closed head injury due to acceleration concussion there is therefore a gap in memory for the events which occurred during the period of confusion, but in addition there is almost always a short gap in memory for events which occurred immediately before the injury. This R.A. may be very short, simply a matter of one or two seconds, but in some instances may extend for minutes, hours, or even days. The genuine traumatic R.A. can only be accurately assessed if the patient was awake and alert at the time of the injury: obviously if he were asleep this would lead to an unexpectedly long R.A. A short R.A. is therefore a very common feature not only of cerebral concussion but also of E.C.T. The phenomenon of R.A. is now considered to illustrate an essential feature of the memory process, namely one that points to the establishment of a memory trace being dependent on a continuing process after the event, which requires normality of brain function for a substantial period. The establishment of a memory is therefore a continuing process, which requires to go on for a period, presumably by a repetitive process, after the event has passed. The establishment of a memory thus often tends to strengthen as time passes, so that the most vulnerable memories tend to be of events which occurred just before an accident, and that the most resistant memories are among those which occurred long before an accident. The relative vulnerability of recent memories and experiences is a very striking feature of many patients with moderate or severe concussion, so that after recovery it is found that not only is there a short complete period of retrograde amnesia but

episodes that occurred within a few days or weeks before the injury are much less firmly recalled than those which occurred at a longer period before the injury<sup>12</sup>. This often seems to happen without reference to the importance of the memory to the individual. Both the retrograde amnesia phenomenon therefore, and the vulnerability of recent memories support the view that the establishment of a memory trace requires a substantial period of time if the memory is to survive the effects of a cerebral concussion. Some may attempt to explain these amnesic features as being due to a failure of recall, and this certainly sometimes plays a part, but the evidence now is overwhelming that both R. A. and the amnesia for recent events are mainly due to a failure to establish a proper "memory trace".

Of special interest in relation to retrograde amnesia is the pattern of the recall of past memories during a period of traumatic confusion prior to full recovery. At this stage the patient often thinks himself to be years younger and to be in a place where he lived long ago. The recent years may be entirely forgotten in this phase and it is only slowly, perhaps over a period of days or weeks, that the retrograde amnesia shrinks from a period of years to perhaps a period of minutes, as full recovery occurs. This remarkable feature of traumatic confusion is hard to explain, for it is difficult to understand how a recalling system can be so much more effective for remote memories. It could however be supposed that the strengthening of some memories with the passage of time may continue for years so as to render them less disturbed after concussion than are more recent experiences.

The time factor concerned with the establishment of a memory trace has also been studied in animals by administering E.C.T. after "training runs"<sup>2</sup>. Such experiments demonstrate that E.C.T. given soon after the training run (5-10 mins) prevents learning but if given an hour after the training run there is no such interference with learning.

### Hallucinations of events in the amnesic period

Of particular interest from the point of view of memory mechanisms is the occasional occurrence during recovery from severe head injury with substantial amnesia, of short-lasting hallucinations of some event which was clearly related to factors causing the injury<sup>9</sup>. These short-lasting hallucinations occur soon after recovery of full consciousness and last only for a few seconds. They occur when the patient's thoughts are apparently at rest and fully relaxed and they are not associated with any accompanying emotional reaction such as fear or apprehension. Such hallucinations may for example consist of the sudden image of a galloping horse or the huge tyre of a motor vehicle bearing down on the individual, and on questioning it becomes apparent that the accident was in fact associated with a vivid visual experience of this kind just before the injury occurred. It seems likely that this half memory represents a repetitive process which was set up in the occipital region of the brain at the time of the injury but was prevented by the injury from establishing itself as a proper memory with its associated emotional concomitants. It is of great interest to compare this with the rare phenomenon of visual perseveration<sup>3</sup>, which sometimes occurs after focal lesions of the occipital lobe. In this phenomenon, previously seen objects remain clearly visible after they have gone, and interfere with reception of other visual information. This seems to be a repetitive process of a slightly different kind, but both give a glimpse of the remarkable mechanisms which seem to be associated with perception and the storage of items observed or experienced.

### The physiology of memory

It is important to view these traumatic memory disorders against the background of other evidence regarding the anatomy and physiology of memory processes. It is

reasonable to emphasize the recent evidence that one system of the brain influences the threshold of another<sup>4</sup>. The alerting system from the reticular formation of the brain-stem is the best known of these, but there is plenty of evidence, both clinical and experimental, to indicate that parts of the limbic system of the brain play an essential part in the establishment of memories. This is well seen in the disappearance of the ability to memorize in Korsakoff's syndrome, caused either by Wernicke's encephalopathy, by encephalitis affecting both temporal lobes, or by the bilateral surgical excision of both temporal lobes. These clinical syndromes damage the limbic system on both sides of the brain and are all associated with a loss of the capacity to establish new memories. This, therefore, may be a system which is particularly vulnerable in concussion and which when injured gives rise to such a great variety of disorders of memory and recall<sup>11</sup>.

### Traumatic Korsakoff's Syndrome

In some cases with severe closed head injury there is a long period of traumatic psychosis of the Korsakoff variety which continues for weeks or months. Another type of injury which can lead to this Kor-

sakoff's syndrome occurred in a few instances of brain wound from the second world war, where a small metal fragment penetrated the brain and, without doing much damage on the way in, crossed the mid-line in the region of the third ventricle and led to a severe and often permanent Korsakoff type of disorder<sup>5</sup>. In both these examples there is no evidence of gross damage to the cerebral hemispheres and yet a severe inability to establish memory traces occurs and may be permanent. This confirms the observations of clinical disease which indicate that the deeply placed limbic structures of the brain are an essential feature of memorizing. In some way, therefore, the limbic system is intimately connected with the on-going physiological process which seems to be essential for the formation of memory traces. Further, this mechanism, which seems essential to the establishment of memory traces, does not apparently remain so essential for the recall of remote memories which were established before the limbic system was damaged. Here are several mysteries which are still unsolved, for the relationship between establishing memories and recalling memories, and in recognizing a true memory when it appears, are closely interconnected and yet distinct from each other.

## S U M M A R Y

The impairment or loss of consciousness resulting from cerebral concussion causes the patient after recovery to be aware of a period of post-traumatic and of retrograde amnesia. Memory of events shortly before the injury may also be affected in

that there is a relative vulnerability of recent memories. The significance of the traumatic amnesias is considered in relation to recent advances in knowledge concerning the physiology of memory and learning.

## R E S U M E N

La perturbación de la conciencia o pérdida del conocimiento que resultan de la conmoción cerebral hacen que el paciente

al recobrase pase por un período de amnesia post-traumática y retrógrada. Puede estar afectada igualmente la memoria re-



ferente a acontecimientos sucedidos inmediatamente antes del accidente ya que los recuerdos recientes son sumamente vulnerables. La significación de las amnesias

traumáticas es considerada en este trabajo en relación a progresos recientes en el conocimiento de la fisiología de la memoria y del aprendizaje.

## R É S U M É

En reprenant conscience après une commotion cérébrale, le traumatisé se rend compte d'une période d'amnésie retrograde et antérograde. Il peut souffrir aussi un déficit mnésique portant sur les semaines qui ont précédé l'accident, car les souvenirs

récents sont relativement vulnérables. L'article discute l'importance des amnésies traumatiques par rapport aux découvertes récentes sur la base physiologique de la mémoire et de l'apprentissage.

## Z U S A M M E N F A S S U N G

Nach der Genesung erfährt der Patient eine Periode von posttraumatischer und rückläufiger Amnesie, die durch den Verlust des Bewusstseins als Folge der Gehirnerschütterung hervorgerufen wird. Das Erinnerungsvermögen an Ereignisse, die innerhalb von wenigen Wochen vor der Verletzung stattfanden, kann auch beruehrt

werden, da eine relative Verwundbarkeit von frischen Erinnerungen besteht. Die Bedeutung der traumatischen Amnesien wird mit Bezugnahme auf kürzliche Fortschritte in der Wissenschaft der Physiologie des Gedächtnisses und des Lernens betrachtet.

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# Amnésies Affectives

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L'amnésie hystérique a connu au XIX<sup>ème</sup> siècle une grande vogue, aussi bien dans les ouvrages scientifiques — pour ne citer que les observations les plus célèbres, rappelons les travaux de Azam (1887), de Pitres (1891), de Morton Prince (1898) et de Pierre Janet — que dans les oeuvres littéraires — de Balzac à Giraudoux, — longue est la liste de ceux qui ont utilisé, comme prétexte à une situation dramatique, l'amnésie consécutive à un choc affectif ou émotionnel. Les travaux de S. Freud<sup>1</sup> ont cependant modifié fondamentalement l'approche des difficultés de remémoration, et ont permis une étude scientifique de ce qui était considéré auparavant comme un mystère de la nature. Pendant la même période, si les conditions d'évocation des souvenirs étaient mieux étudiées chez des patients présentant les organisations mentales les plus diverses, les cas de grande amnésie hystérique regressaient en nombre, de la même façon que tous les grands syndrômes du même ordre.

Aujourd'hui, le terme "affectif" est lui-même d'un usage malaisé en nosologie psychiatrique. Le fonctionnement psychique dans son ensemble est soumis à l'action des forces inconscientes, dont les effets peuvent être observés dans l'activité mentale normale comme dans les états pathologiques, quelles que soient leurs conditions d'apparition. Ces pulsions inconscientes, et leurs dérivés ou leurs représentants, constituent ce que le langage courant désigne sous le terme d'"affectivité", terme à la fois trop restrictif et trop vague pour être très utile.

Tout le monde reconnaît aujourd'hui que dans les désordres et les réorganisations mentales consécutives aux lésions cérébrales les plus certaines et les mieux connues, l'organisation inconsciente et le passé vécu du sujet doivent être pris en considération, pour expliquer un certain ordre électif d'échecs et de réussites, aussi bien dans l'activité spontanée qu'au cours de rééducation. Il en résulte une impossibilité de limiter l'étude des "amnésies" affectives aux seules difficultés pour lesquelles on supposerait une "genèse" purement psychique, car cela impliquerait l'hypothèse d'une différence de nature qu'un examen objectif des faits cliniques ne justifie pas entièrement.

Enfin, il nous faut souligner une autre difficulté pour une sémantique rigoureuse. Les travaux et les théories de S. Freud<sup>1</sup> ont montré le rôle du passé dans l'activité psychique du présent, et l'importance que peut prendre la possibilité ou l'impossibilité de retrouver un désir infantile, d'en reconnaître le caractère ancien. La capacité de remémoration et ses limitations sont indissociables de la vie mentale dans son sens le plus étendu, sans parler de son rôle dans les activités symboliques qui nécessitent l'utilisation d'un code. La capacité de fixer ou d'évoquer des représentations ou des affects anciens, et d'en reconnaître le caractère passé ne peut être considérée comme une fonction élémentaire, puisque cette activité se retrouve dans la plupart des activités mentales conscientes, et que toutes les activités mentales ont une in-

fluence sur elle. De ce fait, le déficit mnésique ne peut être séparé d'autres altérations de la mémoire, fréquemment observées chez les sujets réputés normaux et chez les névrosés comme chez les psychotiques de façon plus spectaculaire.

Henri Ey<sup>3</sup>, dans sa perspective neo-Jacksonnienne, distingue "les troubles où prédominent les troubles négatifs (amnésies et dysmnésies) et ceux où prédominent les troubles positifs (hypermnésies, illusions de la mémoire et paramnésies) alors que Jean Delay<sup>2</sup> oppose, dans un esprit assez voisin, les amnésies démentielles aux libérations "de la mémoire autistique" qui conduit au délire. La théorie psychanalytique ne permet pas de distinguer des troubles positifs ou négatifs (distinction nécessitant une hypothèse psychologique téléologique). La difficulté d'évoquer certains souvenirs (qui entraîneraient du déplaisir ou de l'angoisse en devenant conscients), et la réorganisation fantasmatique du passé vécu sont indissociables de la vie mentale la plus habituelle.

Nous étudierons successivement: 1<sup>o</sup>—Amnésie et remémoration en dehors des états pathologiques caractérisés. 2<sup>o</sup>—Les amnésies et dysmnésies des structures névrotiques. 3<sup>o</sup>—La mémoire et ses variations dans d'autres structures psychopathologiques. 4<sup>o</sup>—Quelques aspects particuliers des troubles de la mémoire chez des malades atteints de lésions cérébrales et chez les vieillards.

# I. Amnésies et remémoration en dehors des états pathologiques caractérisés

Nous devons à S. Freud<sup>4</sup> une connaissance précise de ces phénomènes, dont l'étude constitue une partie importante de la métapsychologie psychanalytique. Nous n'en rappellerons ici que l'essentiel nécessaire à la compréhension des dysfonctionnements mnésiques pathologiques.

Deux éléments retiendront principalement notre attention:

## a) *L'amnésie infantile*

C'est une constatation que chacun peut faire sur lui-même que l'on ne garde que des souvenirs lacunaires des premières années de sa vie. Ce fait a paru pendant des siècles tout-a-fait anodin, et relié à la faiblesse naturelle de l'esprit enfantin. Cette explication simpliste ne tient pas compte des aspects spécifiques de cette amnésie. C'est qu'en effet, dès la deuxième année de sa vie, l'enfant se montre parfaitement capable de fixer et d'évoquer des souvenirs, de dénommer objets, lieux et personnes entrevues parfois brièvement, et ceci au bout d'un intervalle de temps parfois fort long. Ce n'est qu'après quelques années que l'on s'aperçoit que l'enfant devenu grand a oublié des événements et des circonstances de sa vie, qu'il avait été, les années précédentes, parfaitement capable de remémorer.

Ce phénomène d'oubli n'est en effet nullement progressif. Il survient à un certain moment de la vie de l'enfant, et il n'est pas indifférent de savoir ce qui se passe, précisément quand l'enfant cesse de reconnaître comme sienne une histoire qui pourtant jusqu'alors était suffisamment organisée et cohérente pour qu'on soit en droit de la supposer définitivement fixée.

Les psychanalystes rattachent cet oubli au refoulement consécutif à l'identification au parent du même sexe, à l'acmé du complexe d'Œdipe. Cette identification donne une forme cohérente à l'ensemble de pulsions libidinales et destructrices qui constituent le sur-moi. Le refoulement des désirs œdipiens entraîne le refoulement de toute représentation s'y rattachant, et l'émergence dans le champ de la conscience de souvenirs ainsi "marqués" provoque autant d'angoisse que la résurgence du désir réprimé.

Une telle explication surprendra toujours celui qui souhaite nier l'expérience psychanalytique. Mais même ce lecteur reconnaîtra qu'elle rend mieux compte de l'aspect actif et précis de l'amnésie infantile, que l'hypothèse de la débilité de la mémoire infantile. D'autant plus que les



variations de l'amnésie infantile sont souvent en rapport, comme nous le verrons plus loin, avec la structure du sujet.

b) *L'amnésie et la remémoration dans la formation des rêves, des actes manqués et des "symptômes" de la vie quotidienne. Préconscient et inconscient.*

C'est l'analyse des rêves qui a conduit S. Freud<sup>4</sup> à prendre en considération l'amnésie infantile (La signification des Rêves - 1899). Par la méthode des associations libres, première ébauche de la technique psychanalytique, S. Freud<sup>4</sup> découvrit que le rêve, derrière son texte inintelligible à première vue, avait un sens qu'il était possible de rétablir. Ce contenu latent implique la réalisation d'un désir enfantin *oublié*, réalisation que prend parfois une forme compliquée sous l'effet des pulsions antagonistes. Il s'agit alors de compromis, satisfactions partielles des pulsions, que l'on retrouve aussi bien dans la formation des lapsus, oublis, actes manqués divers — décrits par S. Freud dans la "Psychologie de la Vie Quotidienne" (1901) — que dans la formation des symptômes névrotiques ou psychotiques.

Nous en retiendrons, pour ce qui nous intéresse ici qu'un désir, une peur ou un déplaisir, un événement lié à un affect désagréable, ou à un plaisir interdit peuvent être oubliés, mais que leur disparition en tant que représentation consciente n'entraîne pas la suppression de leur effet sur la vie mentale. Plus la repression est intense, plus ses effets indirects sont importants. La représentation ou l'affect oubliés sont remplacés par une autre représentation, sur laquelle le désir ou la crainte peuvent être déplacés, — c'est ce qui se passe, par exemple, dans le rêve —, par un autre fragment du discours du malade dans une cure psychanalytique, ou par un comportement répétitif.

Le souvenir oublié que le sujet peut retrouver au cours du travail de remémoration d'une cure psychanalytique ont été qualifiés par S. Freud de préconscients, par opposition aux éléments du psychisme qui

ne peuvent jamais devenir conscients et qui ne sont connus qu'à travers le discours conscient, et qui constituent l'inconscient proprement dit.

Parfois, un souvenir-écran peut avoir une valeur particulière pour le sujet, sans qu'il en connaisse pour autant les implications. Au cours d'une cure psychanalytique, il n'est pas rare de constater qu'un tel souvenir est renforcé par des affects provenant de souvenirs oubliés, qui sont ainsi "représentés" par le souvenir écran. Quand il s'agit d'un souvenir de cette nature remontant à l'enfance, il arrive souvent que l'on puisse démontrer qu'il ne correspond à aucun événement réel, mais qu'il est le produit d'une élaboration fantasmatique du passé. (cf. Freud<sup>4</sup> "l'Homme aux Loups" - 1918).

Savoir si l'on peut remonter au delà des souvenirs écrans, si un malade peut se remémorer les événements vécus dont les effets ultérieurs se reconnaissent par l'analyse structurale de souvenirs plus récents, est une des questions fondamentales et des plus controversées de la théorie psychanalytique. Outre le très important article de Freud "Remémoration, Répétition et Elaboration" (1914), il est intéressant de comparer à ce sujet les opinions de Mélanie Klein et de ses élèves, avec ceux des psychanalystes de l'école de New-York (cf. E. Kris,<sup>5</sup> la Remémoration de souvenirs d'enfance en Psychanalyse, 1955).

La même qualité fantasmatique peut être retrouvée dans des souvenirs concernant des événements fortement investis. Au cours d'une recherche que nous effectuons actuellement avec S. Lebovici sur l'évolution des psychoses infantiles, nous avons eu la curiosité de faire reprendre l'anamnèse de cas que nous suivons de très près depuis plus de 15 ans. Le récit des parents s'est révélé le plus souvent en contradiction flagrante avec les faits dont nous avons été témoins, et qui étaient consignés dans les observations des malades. S'il faut parfois tenir compte d'arrefacts possibles d'observation qui peuvent induire en erreur les observateurs, il n'en reste pas moins que

l'étude de la fantasmatisation par les parents de l'histoire de leurs enfants est une façon très intéressante d'observer des amnésies électives, ainsi que des confabulations parfois surprenantes. On en apprend ainsi beaucoup sur l'inconscient du narrateur, et on peut ainsi vérifier l'hypothèse que les renseignements anamnétiques recueillis en psychiatrie — qu'il s'agisse d'enfants ou d'adultes, — n'ont pas de grande valeur objective.

Ainsi la possibilité ou l'impossibilité de remémorer, dont on ne peut dissocier les produits dérivés du refoulement actif qui en est responsable, forment une paire contrastée qui constitue une dimension importante de la vie psychique.

## II. Les amnésies des structures névrotiques

a) La grande hystérie a fourni longtemps le modèle exclusif de l'amnésie affective ou psychogène, comme en témoigne encore un sous-titre de l'Etude N° 9 d'Henri Ey<sup>3</sup>.

Deux types d'amnésie sont classiquement décrits sous la dénomination d'amnésie hystérique :

— L'amnésie des malades présentant des troubles spectaculaires de la personnalité, du type personnalités multiples. C'est le cas de la célèbre Felida décrite par Azam, et la non moins illustre Miss Beauchamp de Morton Prince en est un exemple cité dans les traités classiques. Au cours des "états seconds" (ou multiples dans l'observation de Morton Prince), le sujet ne semble disposer que d'une série particulière de souvenirs, apparemment oubliés quand il est dans l'autre état. Il n'y a guère d'observation récente de cas de ce type, qui ont disparu avec les grandes manifestations d'hystérie. Il est cependant utile de rappeler que c'est en essayant de comprendre scientifiquement la signification de souvenirs retrouvés par les hystériques sous hypnose, c'est à dire dans un "état second" provoqué, que Breuer et Freud, puis S. Freud

tout seul, découvrirent les lois de l'inconscient.

— Très différente en apparence est l'amnésie d'évocation (ou retrograde) de ces malades que l'on découvre un jour ayant perdu tout souvenir biographique. Les observations de ce type sont fort nombreuses. Généralement les souvenirs reviennent par groupe, à partir de quelque élément inducteur.

Ces formes d'amnésie provoquaient généralement dans les sociétés de psychiatrie ou elles ne manquaient pas d'être présentées, l'éternelle discussion pour savoir si de tels malades, qui par ailleurs n'étaient atteints d'aucun trouble neurologique, ni d'aucune affection mentale suffisamment spectaculaire pour correspondre à une nomenclature classique, étaient hystériques ou simulateurs. Et jamais les tenants de la simulation ne se demandaient pourquoi des sujets réputés non-malades mentaux se livraient à une simulation tellement contraire à leurs intérêts les plus évidents. En fait la plupart des observations de la période classique, telles celles citées par Henri Ey<sup>3</sup> dans son étude, sont beaucoup plus orientées vers la démonstration de la réalité du trouble, que sur l'étude de la personne du malade et de sa biographie. Ce qui reste sûr, c'est qu'il faut être psychotique pour que des bénéfices primaires ou secondaires inconscients puissent justifier une telle simulation.

Car toute la discussion hystérie-simulation est viciée par la psychiatrie de guerre et par les expertises de compagnies d'assurance. C'est cependant en pratique de guerre et en traumatologie que sont observés les cas d'amnésie post-commotionnelles, de nature très voisine des précédentes, et qui firent les grands succès de la narcose pendant la dernière guerre mondiale.

b) Beaucoup plus fréquentes sont les amnésies partielles et transitoires observées chez les malades présentant par ailleurs des symptômes hystériques moins spectaculaires. Dès 1905, S. Freud (Etude d'un cas d'hystérie Dora) insistait sur les grandes difficultés mnésiques des hystériques, et sur leur incapacité à retracer tant l'histoire de

leur maladie que celle de leur vie. Il s'étonnait de la précision de certaines observations publiées dans la littérature, étant donné qu'on ne peut se faire une idée à peu près cohérente qu'après avoir écouté le malade pendant de nombreuses séances. Les raisons de cette difficulté mnésique se trouvent dans le refoulement lui-même et dans la signification que prend pour le patient son dialogue avec le médecin, d'emblée investi sur un mode transférentiel. Freud pensait même que l'on pouvait éliminer le diagnostic d'hystérie si le malade était d'emblée capable d'un récit cohérent et ne présentant pas de grave lacune.

Cependant, on trouve des difficultés d'évocation de souvenirs dans d'autres structures névrotiques que la structure hystérique.

Chez les malades présentant une névrose obsessionnelle, il n'est pas rare de constater un étonnant allongement de la période d'amnésie infantile. Plusieurs de mes patients présentant des structures de ce type, affirmaient, alors qu'ils n'avaient pas atteint la quarantaine, n'avoir aucun souvenir de leur enfance ni de leur adolescence. Ils ne pouvaient retrouver la suite cohérente des événements qu'ils avaient vécu qu'à partir de l'âge de seize ans pour une, dix-huit ans pour un autre. Pour la première, cette amnésie n'était en fait pas totale, et en faisant un effort, elle retrouvait des éléments qui lui permettait de retrouver une suite apparemment correcte d'événements. Cet exercice lui paraissait tout-à-fait artificiel, et sans intérêt, puisque de toute façon, elle était véritablement incapable de se souvenir de quoi que ce soit qu'elle ait elle-même pensé ou ressenti. C'est le refoulement de tout affect qui lui donnait ce sentiment d'amnésie complète d'une aussi longue période de sa vie. Pour le deuxième malade, la nature de l'amnésie infantile, encore plus prolongée, que pour la première, se révéla différente au cours de l'analyse. Des événements lui revinrent à la mémoire, mais à chaque fois au milieu d'un contexte particulièrement angoissant. Ce patient avait en fait longtemps souffert, d'une façon consciente, de beaucoup d'in-

suffisances de la part de sa mère. Son enfance et son adolescence s'étaient déroulées sur un mode très dépressif. Il avait fini par effectuer un véritable travail de deuil à l'égard de cette mère, pour qui il ne manifesta plus qu'une indifférence polie. En même temps que des symptômes compulsifs apparaissaient, il devint un jeune homme plein d'allant, mais se mit à rejeter avec horreur tout ce qui pouvait lui rappeler le long état dépressif dont il venait de sortir. Le travail de remémoration fut très pénible pour lui, et souvent il manifesta une grande répulsion à continuer l'exploration d'un champ qu'il avait eu tant de mal à clore.

D'une manière plus générale, les mécanismes d'isolation, qui prennent une grande importance dans la névrose obsessionnelle, perturbent la remémoration et provoquent des effets amnésiques aussi importants parfois que dans l'hystérie, bien que d'une nature différente. Ici, ce n'est plus l'ordre du récit qui est perturbé, mais sa substance même, réduite à un squelette. Les représentations étant séparés des affects, et ces derniers étant sévèrement refoulés, aucun élément ne paraît plus significatif au patient. Il ne peut alors raconter sa vie qu'en se repérant sur des détails matériels, et affirme ne se souvenir de rien d'autre.

D'autres obsédés ont une possibilité apparemment normale d'évoquer des souvenirs, mais ceux-ci semblent avoir perdu toute relation avec leur vie présente. Une série comme l'autre étant isolée des affects qui pouvaient s'y rattacher, tout rapprochement entre les deux devient un exercice purement artificiel. Mais ces malades se rapprochent de quelques autres que nous allons évoquer au paragraphe suivant.

c) Ce tour d'horizon rapide des difficultés mnésiques des structures névrotiques serait incomplet, si nous ne citons les perturbations mnésiques liées à un déplacement des investissements intellectuels.

Le sentiment de "déjà vu" a été observé depuis l'antiquité, puisqu'on attribue à Pythagore l'opinion qu'il fallait y voir une preuve de la metempsychose. On trouvera dans l'étude d'Henri Ey<sup>3</sup>, une bibliogra-



phie abondante des observations anciennes. Comme le faisait remarquer S. Freud<sup>4</sup>, deux types d'explication peuvent être alors employés. Les premières ne veulent voir dans la fausse reconnaissance que l'erreur, le déficit dont il convient de rechercher la cause. Freud cite un auteur du XIX<sup>ème</sup> siècle, Wigan (1860), qui utilisait déjà l'hypothèse d'un asynchronie des deux hémisphères cérébraux. D'autres explications impliquent l'hypothèse d'un fait ancien réel et oublié, provoquant le sentiment de déjà vu (Grasset — 1904 — cité par Freud). La psychanalyse permet une étude précise des conditions d'apparition de ce phénomène. Dans la deuxième édition de "La Psychopathologie de la Vie Quotidienne", Freud étudie le cas d'une jeune fille qui avait connu cette impression étrange au cours de la maladie d'une jeune camarade. L'analyse montra que pendant une maladie grave de son propre frère, la patiente avait vivement refoulé des souhaits de mort à l'égard de celui-ci, ce qui empêcha ensuite l'analogie entre les deux situations de devenir consciente. En 1913, le même auteur publia un travail sur un cas de fausse reconnaissance fréquemment observé depuis au cours du traitement psychanalytique: il s'agit de l'impression fausse, que certains patients ressentent, d'avoir déjà raconté un événement dont ils font, en fait, le récit pour la première fois. Un tel phénomène se produit quand une représentation est refoulée au cours d'une séance alors qu'elle était sur le point de devenir consciente.

### III. La mémoire et ses variations dans d'autres structures pathologiques

Nous n'insisterons ici que sur quelques points particuliers, qui permettent, par opposition, de mieux cerner les troubles de la mémoire d'ordre névrotique.

— Il n'est pas rare de constater que certains malades, présentent une structure psychosomatique, telle qu'elle a été décrite en France par F. Marty, M. Fain, M. de M'Uzan et Ch. David<sup>6</sup>, ont une très grande facilité pour retrouver l'histoire anecdotique de

leur vie. Mais ce récit, fourmillant de détails non significatifs, ne semble pas les impliquer: toute la tension conflictuelle se liquidant dans le processus somatique, on ne retrouve aucune de ces altérations du récit considérées par Freud comme pathognomoniques de l'hystérie.

— S'il est classique de citer les amnésies électives qui font partie intégrante des délires des schizophrènes, ainsi que les fausses reconnaissances et le sentiment délirant de déjà vu et de déjà vécu, il est nécessaire de souligner qu'au cours de traitements psychothérapiques prolongés, ces malades font preuve d'une implacable mémoire en ce qui concerne les événements de leur vie, et les sentiments qui s'y rattachent.

### IV. Quelques aspects particuliers des troubles de la mémoire chez les malades atteints de lésions cérébrales et chez les vieillards

On ne saurait traiter "d'amnésies affectives" sans faire mention du rôle de ce que l'on désigne, en théorie psychanalytique, sous le nom d'investissements et de contre-investissements (cathexis), dans le caractère électif de ce qui paraît perdu au cours des amnésies rétrogrades. C'est ainsi que Barbizet et coll.<sup>1</sup>, dans un récent article consacré au syndrome amnesique des traumatisés crâniens, montrent que, dans des cas où l'on peut supposer une atteinte élective du système hippocampomammillaire, la conservation du souvenir est fonction de l'affect qui s'y rattache. De telles indications peuvent avoir un très grand intérêt pour la conduite des rééducations, en particulier en traumatologie, où l'analyse de la signification prise par l'accident comme par ses séquelles est essentielle pour comprendre les vicissitudes de la récupération fonctionnelle.

Enfin, rappelons que si le tableau clinique des malades atteints de démence sénile comporte une amnésie rétrograde portant sur les faits les plus récents, si le tableau clinique de la presbyophrénie comporte une amnésie de fixation assez impression-

nante, contrastant avec une assez bonne possibilité d'évocation, s'il est indiscutable que des lésions artériopathiques du cerveau donnent des troubles mnésiques accompagnés souvent de difficultés d'ordre aphaso-apraxo-agnosiques, il n'en reste pas moins que ce modèle a longtemps permis une attitude démissionnaire des médecins et des psychiatres devant le problème des personnes âgées. Pour beaucoup d'entre elles, les difficultés de fixation sont dues à un état dépressif chronique, caractérisé par un retrait d'intérêt, un "desinvestissement", dirions-nous, pour ce qui se passe autour d'eux. On ne retient que ce qui prend un sens dans son propre système de coordonnés. Autant faut-il en avoir un. Deux or-

dres de faits jouent dans le sens de ce desinvestissement: la situation même du sujet qui a fini de jouer son rôle (rôle d'amoureux ou de parent, rôle professionnel), l'organisation personnelle du sujet qui avait équilibré sa vie d'adulte jeune grâce à des compromis précaires, et qui a perdu tout espoir de satisfaction narcissique régressive quand s'est effondré le fragile échafaudage qui avait fait jusqu'alors illusion. L'allongement de la durée générale de la vie met à l'ordre du jour la psychothérapie et la sociothérapie des vieillards, et ses premiers résultats montrent que dans beaucoup de cas, les difficultés de fixation ne sont nullement irréversibles.

### R É S U M É

Si les amnésies constituent un tableau clinique parfois bien individualisé, la mémoire n'en est pas pour autant une fonction isolable, dont on pourrait étudier de façon linéaire les déficits. De même, le concept "affectif" a aujourd'hui un sens trop global pour être utilisé dans un sens pathogénique ou causaliste. Par contre il est utile d'étudier les modalités de remé-

moration, ses facilités et ses difficultés, ses limites, en cernant ainsi la définition même de l'inconscient, dans les différentes structures mentales, qu'il s'agisse de névroses, de psychoses, ou de réorganisations après des lésions cérébrales. Les troubles mnésiques des vieillards méritent d'être étudiés sous ce même angle.

### R E S U M E N

Aunque las amnesias constituyen a veces cuadros clínicos bien individualizados, la memoria no es sin embargo una función aislable, cuyos disturbios pueden estudiarse en forma lineal. Del mismo modo, el concepto de "afectividad" tiene actualmente un sentido demasiado amplio para poder ser utilizado en el plano de la patología o de la etiología. Por el contrario es útil estudiar las modalidades de la rememoración,

sus ventajas y sus dificultades, sus límites, circunscribiendo así la definición, aún de lo inconsciente, en las diferentes estructuras mentales, ya se trate de neurosis, psicosis o de reorganizaciones posteriores a lesiones cerebrales. Las alteraciones de la memoria en las personas de edad avanzada deben ser consideradas también desde este ángulo.

### S U M M A R Y

Even if amnesia sometimes evidences a well individualized clinical picture, this fact does not allow to consider memory as

an isolated function whose disturbances can be studied in a linear sense. At the same time the concept "affective" has ac-

quired a too broad meaning to be used in pathogenesis or aetiology. Contrarily to this, it is useful to study the forms of remembrance, its facilities and difficulties, its limits, in order to circumscribe the definition, even of unconsciousness, in the

different mental structures of every case: neurosis, psychosis or reorganization after lesions of the brain. The mnesic disturbances in old people have to be investigated from the same viewpoint.

## ZUSAMMENFASSUNG

Wenn die Amnesien zuweilen ein gut individualisiertes klinisches Bild darstellen, so ist das Gedächtnis deshalb noch keine isolierte Funktion, and der man die lineare Form des Defizits studieren könnte. Gleichweise hat der Begriff "affektiv" heute eine zu globale Bedeutung, um in einem pathogenetischen oder aetiologischen Sinne benutzt zu werden. Dagegen ist es möglich, die Formen des Wiedererinnerns

seine Erleichterungen und seine Schwierigkeiten, und seine Grenzen zu studieren, um so die Definition des unbewussten selbst zu umschreiben, bei den verschiedenen mentalen Strukturen, ob es sich um Neurosen, Psychosen oder Reorganisationen nach zerebralen Laesionen handelt. Die Gedächtnisstörungen der Greise müssen unter demselben Gesichtspunkt studiert werden.

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### 4TH SYMPOSIUM OF THE FULTON SOCIETY

September 20 — 1969

HILTON HOTEL — N. Y. C.

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Uruguay



# Personality

## Dr. HANS HOFF

A man's biography is a living testimony of what a country and epoch or environment can make on a personality that is trying to fulfil its aim, the growth toward his entelechy.

Hans Hoff has many reasons to thank living for: To be born in the romantic atmosphere of the old Vienna. Where music was raining over the

country like a magic mist, elating and tuning a pace of creation in science. Feeling and thinking grows harmoniously in a country where art and science meet at its highest level.

All his experiences of early life were woven with the setting of the Austro-Hungarian Empire. Music, brilliancy, and the gayity of its gorgeous ceremonial display. He could dream of the Emperor and the Empress leading the way down the great stairways followed by the multicolored courtiers.

The rooms with lavish colored marbles and the mirrors like terse lakes reflecting men's passing glory, statues with frozen movements of static beauty and ceilings peopled with beautiful mythological scenes. And the horse-shoe shaped entrance stairways jealously flanked by picketing guards in uniform, who let pass the court official in ceremonial costumes.



### HIS FATHER AND GRANDFATHER

William James said once that he regards Humanity going upwards like in a big escalator and the people relentlessly going from infancy to old age.

And no one sees the same panorama, no one has the same experience. In this lies the secret of human uniqueness.

To disclose a personality one enriches his life by sharing another people's experience.

Dr. Hoff was born in 1897 in Vienna.

Vienna: It is the time of the Austro-Hungarian Empire. A doctor's child is tense with expectancy. Somebody has knocked at the door late at night. He knows that a drama may be hidden in this sudden sound that blotted the silent night. There are hushed voices with a tinge of despair. His bedroom was next to the hall, where the people were waiting, and it was familiar to him to hear somebody pleading with his father to go to save a life. Being a pedantic man, his father, only went along after taking good care of his appearance. The beard was neatly done and his countenance was in perfect order when at last he was ready to see the patient.

It may be that this wasn't just a way to take care of his flattering image, but part of the therapeutic effect of a doctor coming to see his patient regardless of the time and occasion in a relaxed, selfcontrolled way. Little Hans fought with sleep trying to be awake, counting the minutes for his father's return. And this was not the only occasion that his father's going out late at night kept him awake. There were other occasions when his father left home to attend the Imperial Ball.

This was a very important event that occurred every year. This distinction was due to the fact that he was the doctor to the lower escort of the imperial Court and therefore invited to it. His mother remained at home since only noble families were allowed to attend the Ball...

But there was another reason for keeping little Hans awake that to entertain his fancy, and that was the sweets that the chef of the Imperial Household used to send him, invariably in the same pocket of his father's overcoat. The excitement that preceded the preparation of the Imperial Ball, the impatient waiting for the sweets packed in paper with the Imperial crest, were of prime importance in his early childhood.

He recalled one of his grandparents as a grand-seigneur who deeply, impressed him because he seemed to know everybody, specially the great composer Gustav Mahler, at that time the Director of the Viennese Opera.

The other was a well-known veterinary, who used to treat also human beings, and who wrote a book of prescriptions in Latin, for human use. Emperor Franz Joseph distinguished him with his personal friendship to the point of paying tuition for his son's (Hans Hoff's father) education.

Thus Hans Hoff grew in an atmosphere of art, science and witnessed the pompous way of living of Imperial Vienna.

The World War I disrupted the normalcy of his adolescent's days.

He entered the Army, and when the turmoil was over, he began to study ardently, wishing to recover the precious time lost in preparing himself for the future.

Thus there were no holidays or vacation for him in order to do three semesters in one year. After four years he was rewarded with the title of Doctor in General Medicine.

Dr. Hoff, recalled that there was a time when he had thought of becoming an officer, but the defeat of the Austro Hungarian Army dissolved with it his illusion to become part of it.

Thus destiny spared him the torture of doubt; it was clear for him that it was his way to become a doctor.

At the beginning of his career his aim was to be a surgeon, specially a gynecologist. Recalling this fact, he attributed this wish to a famous picture widely known at that time that showed a noted surgeon Professor Werthein pushing the Death away from a young woman on the operation table. Nothing was more inspiring than this heroic gesture. So he, decided to become a young volunteer to Dr. Halban whom it was presumed would succeed in Dr. Werthein's chair. But at the last moment the Ministry refused to appoint Dr. Halban; so, Dr. Hoff was obliged to have another choice.

Prof. Wagner Jauregg, who had at that time the Department of Psychiatry under his command, was engaged in doing research on the malaria treatment of general paralysis; and even though he wasn't a great teacher, his powerful personality impressed Dr. Hoff who asked him to be accepted as a young collaborator.

Soon he realized that the kind of investigation that Dr. Jauregg was doing was less attractive to him than that carried on at the Institute for Experimental Pathology, and more of his spare time was dedicated to it where by he became acquainted with its operation in animals.

At that time Psychiatry seemed to be too vague in comparison with Neurology which provided ground for logical thoughts.

Then one year later he became acquainted with a dynamic personality of the neurological world, Schilder, a brilliant mind, full of new ideas, inspired and inspiring. Schilder made young Hoff love the neurological sciences. He, along with Wagner-Jauregg and Pötzl formed a triumvirate whose sparkling virtues of quite different qualities left each one a lesson of the mental approach to science.

Wagner Jauregg, cold, exact, detached, was a truthful general doctor dry and dispassionate. Schilder, bubbling with creativeness, was so full of ideas and phantasy that it was hard to bring him back to the ground of reality. Pötzl, was a living testimony which is for a great mind difficult to remain confined in the narrow field of a speciality and he mastered different branches of science from mathematics to biology including neurophysiology, neuroanatomy and physics.

The complex and varied influence of these three men different in many ways but equal in the love of ideals that burned bright in the quest of science were of prime importance in the forming years of a prospective neurologist.

Pötzl and Schilder made a wonderful team, combining their special abilities from which emerged a dynamic neurology with the aim of getting a better understanding of the psychological behavior of the insane.

## WORLD WAR II

Thus destiny favoured Dr. Hoff with wonderful teachers, wise and learned with whom he published, as a young scientist, numerous papers. As the time passed by and with his stores repleted of knowledge, Dr. Hoff sowed the seeds of enlightenment in different parts of the world.

He was Professor in Baghdad, at the Columbia University in New York, Professor of Neurology and Neuropathology at the University of Vienna and finally he became Orifesar in Neurology and Psychiatry at the University of Vienna.

In everyone of these occasions he had to deliver an inaugural lecture. But the one that stirred in him the deepest emotions was the last one deli-



vered in 1950 as a newly appointed full Professor of Neurology and Psychiatry in Vienna. All of a sudden came to his memory when he was like one of the audience, a young student wishing with all his heart to occupy the place where he stood now. He had to clear up his voice, while a strange feeling in his throat made it difficult for him to speak.

### A VERY INTERESTING CLINICAL CASE

All along his career Dr. Hoff had occasion to see many interesting cases, but the one that struck him most was that of a very wealthy Italian lady who came to see him after consulting fifty-six specialists from all over the world. Dr. Hoff made a very careful and thoughtful examination; and he said that maybe because she was in a more advanced stage of her illness, it was evident that she suffered from a Brown-Séquard Syndrome.

Dr. Hoff ordered immediately a myelography which did not show a quite clear picture on the level of the 4th cervical segment on the right side. The patient was almost dying. A very distinguished X Ray specialist, Prof. Schüller, could not decide whether it was a tumor on the level of C3/C4 on the right side or not. The patient's condition was critical. Dr. Hoff young and inexperienced took upon himself a great responsibility. The patient was in a bad condition, almost sixty, fat, making an intervention risky. But Dr. Hoff decided that a laminectomy ought to be done. The operation was performed by Prof. Schönbauer and the tumor was surprisingly easy; a meningioma almost fell into the surgeon's hands. The patient had a speedy recovery after which she ordered the following: the picture of the tumor to be sent to all the fifty-six doctors she consulted in different lands. And she cabled to her son-in-law who managed her estate during her illness, telling him that from now on she would take again everything in her own hands.

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Dr. Hoff's most important contributions to neurological topics are: Brain pathology; Brain tumors; Posture reflexes; Epilepsy.

His favourite writers in philosophy: Kierkegaard and Montaigne. — His favourite in literature: Shakespeare and Grillparzer.

Dr. Hoff has enjoyed two different periods of Vienna: the old and the new. As a child he witnessed the romantic and luxurious way of living. When power meant richness to the point that it is told there were nobles going to England, who displayed robes where diamonds were sewn on and some of them in a loose way so that they could fall to the ground as a largess.

Modern Vienna still retains traces of the past grandeur, in its baroque castles, its finely laced Gothic Churches and the onion-shaped spires of some of its cathedrals, and the coloured tiles that gayly covered Saint Stephen's roof. The ever flowing-Danube passed through its history carrying echoes of generations that chanted, fought, cried, and new rumours peopled its currents, so that the new and the old eternally mingled through beautiful Vienna.

Dr. Hoff understood and participated in both. Science, Art and History have a curious way to be entwined, in modern Vienna. Some years ago, an important scientific event gave us ground to make this affirmation. It was on the occasion of the VIII International Congress of Neurology, under Professor Hoff's Presidency.

Hofburg, with its imposing building, its massive architecture, its beautiful gardens, was the setting for this important gathering where the most brilliant minds on the field of neurology pooled their knowledge.

Mozart frozen in marble, Goethe with his bronze overcoat, Queens in metallic carriages with ever galloping horses, fauns and nymphs sensually reflected in refreshing fountains, all this was in our way to meetings and reunions.

Then came the Opera's dazzling nights, and the Children's Chorus that gave the impression that Angels could be heard by man. And the places near the Opera where you could have a wonderful dinner in a charming historical atmosphere, enjoying Viennese friendship so warm and full of anecdotes. This was the other side of the Congress.

While the Congress invaded History and in the same wonderful drawing rooms where the aristocracy seemed far and aloof, the new dignitaries of modern society, the scientists were trading their knowledge in the ancient way by exchanging goods. Somebody called them "Merchants of Light" which it was.

In the same room where the joyful music of Mozart was played and sounded as mischievous elves where tiptoeing his pizzicatos on the clavicord, new rhythms were heard with inner ear of understanding, rhythms under the Greek key of Alfa, Gamma, Beta, Delta, informing science how neurons dance a Minuetto of their own, whether in normal or upsetting times.

The Congress seems now a fine and remote world, in the kingdom of remembrance, in which Dr. Hoff's soft and kind smile greeted us with his ever gay and laughing eyes.

This is the story of a man who grew in a beautiful place, who married the girl of his dreams, whose son followed the intellectually brilliant line of ancestors, who was loved and respected. Dr. Hoff is a contemporary example of effort, endurance, and love to Science.

VICTOR SORIANO.

# Book Reviews

## THE NEUROSCIENCES

A study program planned and edited by Gardner C. Quarton - Theodor Melnechuk, Francis O. Schmitt and the Associates and staff of the Neurosciences Research Program. Published by The Rockefeller University Press, New York 1967. 962 pages.

According to the evolution of knowledge different disciplines have been integrated in the training of neurologists, making this specialization wider and more engaging and also more fascinating. Some decades ago, a neurologist, besides being a clinician, had to know about neuroanatomy and neuropathology. With the progress of neurophysiology and when it began to deal with concepts reaching man, particularly with the use of the monkey for experimentation, the neurologist had to study this discipline for a better understanding of his specialization and in order to be able to incorporate new physiopathological approaches introduced by neurophysiology. This discipline almost immediately won a place of privilege as well as electrophysiology one of its chapters of most extraordinary development, which keeps its relevance up to now.

At present the progress of basic sciences allowed to approach them to the study of the nervous system in all its manifestations, regardless of their complexity, even to the study of psychical activities such as memory and learning. Analysis and research of the nervous system with the aid of basic disciplines constitute what is called neurosciences. Information about them is absolutely necessary for all those practising not only neurological but also psychiatric disciplines. This book of almost a thousand pages is characterized by an outstanding exuberance of information furnished by a group of select specialists. It contains the following sections: 1) Components of the Nervous System; 2) Molecular Biology; 3) Molecular Biology of Brain Cells; 4) Neuronal Physiology; 5) Brain Correlates of Functional Behavioral States; 6) Brain Correlates of Learning; 7) Interdisciplinary Topics.

### 1) Components of the Nervous System

Knowledge concerning the neuron is of chief importance for the comprehension of the manifold kinds of expression of nervous function. Progress in these investigations is closely related to electron microscopy and to the exploration of isolated neurons by microelectrodes. Neurons form circuits with almost reciprocal interaction for their inte-

grative action; the features presented by them depend on the localization, the kind and the amount of contacts of endings on different parts of the same cell and also on the way in which their own endings are distributed. Physiology actually goes beyond the strictly structural boundaries and reaches the study of neural function at a molecular level.

### 2) Molecular Biology

This field is widely explored. A place of privilege is kept for the study of macromolecules, of greatest importance in the neuronal organization due to their dynamic and biologic action. Here the standing concepts and knowledge concerning memory of the species and immunological memory are stressed, leading to the comprehension of psychological memory. Particular attention is paid to immunological memory, defined as the property of the immunological system allowing a higher reactivity to the second administration of a given antigen. Certain analogies between the central nervous system and the immune system are deduced and if a selective mechanism can support the process of learning. It is concluded that the terms instruction and selection may be applied to descriptions of the same process at different levels.

It is believed that it is not very likely for macromolecules (nucleic acids and proteins) to act as a tape for storing information; the latter rather is reflected in the state of the synapses of the neural network and the proteic changes would be provoked by the action of substances released by the presynaptic axon.

### 3) Molecular Biology of Brain Cells

The studies in Molecular Neurobiology have become fundamental to understand the brain and psychological function, in its purpose to explain high brain functions with the broad participation of metabolic processes expressed in form of bioelectrical manifestations.

Following the already mentioned trend of research, immunochemical approaches to the study of the nervous system are made, which are undoubtedly somewhat limited.

Neurogenesis depends to a great extent on the features of the neurons allowing them to enter selectively into functionally critical groupings and interconnections.

In development everything happens with a certain flexibility but anyway a high degree of fixed structural order is reached.

RNA in Brain cells - Neurons and adjacent glia show, when functional balance is changed, that they are totally connected and



constitute a functional unit. Glia seems to be a metabolic stabilizer of sensitive neurons. It would participate at the richness of the RNA fraction of the neuron. This would lead to believe that glia programs the synthesis of macromolecules within the neurons. Glia would also be a modulator of the electrical properties of the neuron.

Protein of Nervous Tissue; specificity, turnover and function. It is stated that it has not been possible to correlate properties of a protein with higher mental function.

Biochemistry and ultrastructure of the membrane are thoroughly analyzed as well as membrane thermodynamics.

#### 4) Neuronal Physiology

Signals and neuronal coding is a challenging subject. Scientists reveal the different codes according to which impulses are transmitted till they reach the brain.

Synaptic and ephatic transmission deserves a thorough and well structured study. In every stage the participation of at least four interfaces, one intercellular and three intracellular, is admitted.

Concerning synaptic transmission excitatory and inhibitory agents of transmission are considered, which correspond to different kinds of secretory processes in the respective presynaptic elements.

Ephatic transmission is exclusively excitatory and it occurs due to electronic diffusion of the currents generated by active cells. Study of comparative physiology of dendrites. - Its role in the regulation of neuronal excitability and in the production of cerebral electrical activity — spontaneous and evoked—, that evidences to represent excitatory and inhibitory postsynaptic potentials generated at various loci in dendrites. An interesting fact to be pointed out is that in the central nervous system of mammals most of the dendrites do not show spike electrogenesis.

The relation between the outer world and our brain is determined at the level of receptor transduction and the first order neural encoding. Different codes are used in different sensory elements and the same code can be used in different ways on different elements.

Two kinds of synaptic mechanisms are established, the excitatory postsynaptic and the inhibitory postsynaptic.

A special kind of nervous cell is described, an inhibitory neuron and two kinds of inhibitory pathway exerting a feed forward and a feedback inhibition. It was in the hippocampus where for the first time the inhibitory interneuron —the basket cell— was identified. In the cerebellum the basket cells, Golgi and Purkinje cells,

are all inhibitory. Two principles in the synaptic process are defined: 1) one cell acts by the same transmitter at all of its synapses; 2) in all the synapses made by the axonal branches of the nervous cells the transmitters open the same kind of gates.

Neurochemical transmission evidences changes in its efficacy, which could act in alternating "switches" involved in the acquisition and storage of information for the brain. For a good neurochemical transmission these are important factors: the rate of the transmitter release, the ease with which the transmitter reaches the receptor and the responsiveness of the receptor mechanism.

In synaptic transmission acetylcholine plays a most important role. Strong evidence exists on the GABA function in this sense. Glutamate action is questionable.

Biogenic amines and behavior. It is likely that not only one amine determines the emotional state. It is admitted that central norepinephrine plays an important role in affective and arousal states; but it is also likely that several amines act in concert on specific areas of the brain: on them an action is exerted by cognitive factors related to the past of the individual and his present situation.

Electroencephalogram is investigated by intracellular recordings in human nervous cells and by means of studies of its relation with synaptic potentials in single units. It is concluded that electroencephalogram is the result of the addition of synaptic potentials of hundreds of thousands of neurons. It is most unlikely that cerebral rhythms are used in sensory coding.

A series of problems are posed concerning the integration of evoked potentials which evidence the variety of elements which at the actual level of our knowledge may be effective in generating them: if spontaneous potential oscillation is possible in the membranes of neurons and to what extent this is significant in cortical dendrites; if effective synaptic transmissions are considered with a minimum or no PSP at all; evoked potentials derived from glia; besides it is also recalled that we deal with partial data, as only gigantic cortical neurons constituting less than 1 % of the whole have been studied, etc. It is undoubtedly necessary to be very careful in the interpretation and evaluation of evoked potentials.

At present it is tried to establish correlations between mechanisms of general and local control commanding integrative actions and certain anatomical elements in the form of circuits of cells and even parts of cells which would essentially carry out those functions: we mention sensory reception, storage, discrimination, actions of adaptation, complex performances involved in memory and learning.

### 5) Brain correlates of functional behavioral states

Modern investigation about sleep evidences the existence of hypnogenic active mechanisms related to structures whose greatest part are situated in the lower part of the brain stem.

Sleep is constituted by two different states alternating regularly: slow wave sleep (possibly related to serotonergic neurons of the Raphe system) and paradoxical sleep (characterized by rapid eye movements and in man it is accompanied by dreaming, probably under the dependency of noradrenergic neurons located in the pontine tegmentum).

In sleep no generalized reduction of the total amount of discharge is observed; in the stage of paradoxical sleep there is an augmented activity of many neurons.

The way in which the organism reacts towards the surrounding is contained in the same organism and this is conditioned by sensory input. The organism leads its actions towards targets which mean an inner satisfaction. Emotional behavior is subordinated to reticular activation. Besides its somatic and visceral manifestations result from discharges coming from the hypothalamus up to the rostral midbrain through descending reticular system.

### 6) Brain correlates of learning

Three theories are presented to explain memory:

1) *Connexionistic theory* explaining memory as the storage of material to be remembered in form of a nervous flow circulating through neuronal circuits. In this theory two aspects are distinguished: on one hand it indicates that certain parts of the brain neurons have a certain organization and must be considered as a whole (non-specific theory); an isolated neuron is not able to memorize. On the other hand connexions of every nervous cell are hierarchically organized, in such a way that information is received by units of the nervous system rather than by groups of cells (specific theory).

In this connexionistic theory of learning the efficacy of synapses is most significant. It is most likely that the use improves the function of synapses and this is surely connected with learning. It is difficult to accept that memory is exclusively a function of circulating impulses because if the animal is subjected to low temperatures and electrical activity of the brain is stopped, memory does not disappear.

2) *Field theory*. — Brain function may evidence changes being independent from synaptic mechanism. For instance the phenomenon of spreading depression slowly scatters along the cerebral cortex and acquires the characteristics of a paroxysmal burst of neuronal activity, which is accompanied by direct current potential shifts and movements of ions and metabolites.

The electrical fields may produce changes in the behavior of the neuron. It is admitted that the field potentials are modulators of basic synaptic activity and that they can therefore act on the latter.

3) *Macromolecular theory*. — Information would be stored in the specific configuration of macromolecules, mainly ribonucleic acid and/or polypeptides.

It is an accepted fact that macromolecules act as reservoirs to keep information; this was clearly demonstrated by genetic information, i.e. memory of the species.

Using antimetabolites to interrupt protein-synthesis, consolidation or retention of the learnt behavior also fails. It is nevertheless prudent not to ascribe a precise significance to this fact, for if the proteic metabolism is altered it has to be also admitted that the brain is damaged in its total functioning, which does not mean that there is a specific action in memory function.

Glia would also have its part here. It is said that glia and the neuron constitute a metabolic and functional unity. Glia would be a metabolic stabilizer of the neuron. Protein synthesis in the neurons could be programmed by transfer of the ribonucleic acid from the glia. Most interesting experimental studies have been made on worms and vertebrates, particularly mice and rats, of transmission experiences or of a greater aptitude to acquire certain experiences, by means of brain extracts of animals who had previously been taught about the same experiences. Ribonucleic acid would play a role in these passages. Stimulators and inhibitors acting on memory and learning are studied. Among the former are stricnine (which would act stopping inhibitory processes in inhibitory synapses of the central nervous system or it may cause inversion from inhibition to excitation), amphetamine (which probably alters the threshold of reinforcement and mechanisms of motivation), scopolamine (having effects antagonistic to acetylcholine; it acts on cholinergic synapses due to the so called antimuscarinic effect), ion potassium (it is not known how it acts), ribonucleic acid, Trycianoaminopropene (it is believed that it increases the synthesis of RNA in the brain under certain conditions), nitrous oxide (which perhaps stops the tendency of the surrounding to interfere with the consolidation of learnt material), magnesium Pemo-line (enhanced nucleic acid synthesis by in-

creasing the activity of true RNA polymerase in vivo and in vitro).

Among the inhibitors of learning we will mention puromycin (inhibitor of protein synthesis), acetoxycycloheximide (AXM) (a potent inhibitor of protein synthesis).

### 7) Interdisciplinary Topics

They bring to the limelight several hypothesis which cut across many fields with a philosophical approach.

The monumental work achieved with the contribution of most qualified and well-known authors has to be read and consulted by all those who deal with any of the disciplines belonging to nervous system, in basic subjects, neuroanatomy, neurophysiology,

neurochemistry, and also in those disciplines related to study and treatment of the patient, neurology, psychiatry, psychology, neurosurgery, electroencephalography, neuroradiology, clinical laboratory.

The topics displayed in the book we are reviewing give rise to new viewpoints of the foundations of all the problems concerning the nervous system —normal or pathological— covering somatic and psychic aspects.

Whoever intends to go in for any of the specialties connected with the nervous system and also those who already are practising, should consider the information on neurosciences of utmost importance, and nothing better to obtain the necessary knowledge than this valuable volume.

VICTOR SORIANO.

## News

### SEVENTH INTERNATIONAL CONGRESS OF ELECTROENCEPHALOGRAPHY AND CLINICAL NEUROPHYSIOLOGY. — San Diego, Calif. USA. — Sept. 14-20, 1969.

Officers of the International Federation: Dr. Antoine Remond, President; Dr. Robert G. Naquet, Secretary; Dr. W. Storm Van Leeuwen, Treasurer.

Organizing Committee for the Congress: Dr. Herbert Jasper, President; Dr. Robert Ellingson, Treasurer; Dr. Richard Walter, Secretary. - The Neuropsychiatric Institute, 760 Westwood Plaza. - Los Angeles, Calif. 90024.

The Seventh International Congress of Electroencephalography and Clinical Neurophysiology will be held in the El Cortez Hotel, in San Diego, on September 13-19, 1969. This year will be the fortieth anniversary of the first publication of the work of Hans Berger and the fortieth anniversary of the publication of Adrian and Bronk's use of coaxial electrodes to study motor unit action potentials in man. In keeping with this date, the theme of the Congress will be:

#### "Appraisal and Perspective after forty years of the functional Exploration of the Nervous System"

The scientific program will consist of a series of didactic lectures presented by international authorities, followed by free communications relating to the subject. All of the major areas will be covered, including electroencephalography, electromyogra-

phy and other electro-diagnostic techniques. In addition, scientific demonstrations and displays will be presented covering the latest techniques in telemetering, the use of computers, and experiments performed at the time of the Congress. The program should be of interest to all investigators and clinicians in the neurosciences.

A number of social events are being planned, some of which are in connection with the 200th anniversary of the founding of the city of San Diego.

Application forms and more detailed information about the Congress can be obtained from: Richard D. Walter, M.D. Secretary, International Congress. The Neuropsychiatric Institute at U.C.L.A. 760 Westwood Plaza. Los Angeles, California 90024.

### North-Holland announces the first volumes of the

#### HANDBOOK OF CLINICAL NEUROLOGY edited by P. J. Vinken\*, G. W. Bruyn\*\*

Editorial advisory board: Raymond A. Adams, Massachusetts General Hospital; A. Biemond, University of Amsterdam; MacDonald Critchley, The National Hospital, London; Russell N. de Jong, University of Michigan, Ann Arbor; Raymond Garcin, Hôpital de la Salpêtrière, Paris; S. Refsum, University of Oslo; K. J. Zülch, Max-Planck Institute Cologne; Shigheo Okinaka, Toranomon Hospital, Tokyo.

\*Executive Chief Editor, Excerpta Medica Foundation, Consultant Neurosurgeon, University Clinics, Amsterdam. \*\*Consultant Neurologist, Netherlands Central Military Hospital, Utrecht.



**Subscription scheme:** The entire work will consist of thirty volumes, to be published over a period of about eight years.

The volumes 1 to 6 are scheduled to appear in the last quarter of 1968 and in early 1969.

All volumes can be ordered separately. A **considerable reduction** however is allowed when entering a subscription to the complete work.

Please request the extensive brochure for the Handbook.

NORTH-HOLLAND PUBLISHING COMPANY, P.O. Box 3489, Amsterdam. The Netherlands.

## THE ISRAEL SOCIETY OF NEUROPHYSIOLOGY AND ELECTROENCEPHALOGRAPHY.

A Joint Scientific Meeting of The Israel Society of Physiology and Pharmacology and The Israel Society of Neurophysiology and Electroencephalography, took place in Jerusalem in May 1968. The following papers were presented:

### SESSION I

Chairman: J. Magnes, Department of Physiology \*.

Pathways involved in caudate nucleus effects on the posterior hypothalamus.

S. Feldman and N. Dafny, Department of Nervous Diseases, Laboratory of Neurophysiology \*\*.

Effects of extrahypothalamic structures on single cell activity in the posterior hypothalamus.

N. Dafny and S. Feldman, Department of Nervous Diseases, Laboratory of Neurophysiology\*\*.

The influence of high oxygen pressure on the electrical activity of the brain.

D. Harel, D. Kerem and S. Lavy, Laboratory of Experimental Neurology, Department of Nervous Diseases \*\*.

Regulation of transmitter release at the neuromuscular synapse.

R. Rahamimoff and F. S. Dodge, Department of Biophysics, University College, London.

### SESSION II

Chairman: H. Edery, Israel Institute for Biological Research.

Psychopharmacological activity of some substances extracted from *Cannabis sativa* L. (Hashish).

Y. Grunfeld and H. Edery, Israel Institute for Biological Research, Ness-Ziona, Israel.

\* Hebrew University-Hadassah Medical School, Jerusalem.

\*\* Hadasah University Hospital, Jerusalem.

Changes in the electrical activity of the brain following pinealectomy.

I. Nir, K. Behroozi, M. Assael, I. Ivriani, F. G. Sulman, Department of Applied Pharmacology, Hebrew University School of Pharmacy and Talbieh Psychiatric Hospital, affiliated to the Hebrew University-Hadassah Medical School, Jerusalem.

Electrocortical reactions of pinealectomized and intact rats to lethal doses of pentobarbital.

K. Behroozi, M. Assael, I. Ivriani and I. Nir, Department of Applied Pharmacology, Hebrew University School of Pharmacy and Talbieh psychiatric Hospital, affiliated to the Hebrew University-Hadassah Medical School, Jerusalem.

A combination narcosis suitable for acute neurophysiological experiments.

J. E. Steiner, Laboratory of Oral Physiology, Hebrew University - Hadassah School of Dental Medicine, Jerusalem.

### SESSION III

Chairman: R. Werman, Department of Pharmacology \*.

Organization of the giant axons in the ventral nerve cord of the American cockroach (*Periplaneta americana*).

M. Spira, I. Parnas and F. Bergmann, Department of Zoology, Hebrew University and Department of Pharmacology \*.

Histological and electrophysiological studies of normal and degenerated giant axons of the American cockroach.

F. Bergmann, M. Spira and I. Parnas, Department of Zoology, Hebrew University and Department of Pharmacology \*.

Microelectrode studies on the giant fibers of the American cockroach.

I. Parnas, M. Spira, R. Werman and F. Bergmann, Department of Zoology, Hebrew University and Department of Pharmacology \*.

Electrical and mechanical properties of the abdominal extensor muscles of the prawn *Palaemon elegans*.

D. Dagan and I. Parnas, Department of Zoology, Hebrew University.

## The Pavlovian Society of North America Announces:

### FIRST INTERNATIONAL MEETING IN MEMORY OF Dr. RAUL HERNANDEZ-PEON

The new Auditorium The Johns Hopkins Hospital, Baltimore, Maryland.  
September, 6-7, 1968.

**Executive Committee:** W. Horsley Gantt; William G. Pare; William G. Reese; David McRiech; B. F. Skinner; Stewart Wolf.

**Program Committee:** J. Perez - Cruet, Chairman; Joseph Stephens; James Bloch.

**Arrangements Committee:** Harry A. Teitelbaum.

## AMERICAN ELECTROENCEPHALOGRAPHIC SOCIETY

Two symposia will be held at the annual meeting of the American Electroencephalographic Society at the San Francisco Hilton, San Francisco, Sept 12-14, 1968.

1. "The Role of Electroencephalography in the Management of Coma" will be held Friday, Sept 13, 1968 at 9 AM. Participating will be: Robert Gumnit, Moderator; Jean Reiher, Michael Saunders, Daniel Silverman, Janice Stevens, and Jolyn Tucker.

2. "Current Problems in Soma-Dendritic Relations" will meet Saturday, Sept 14, 1968, at 9 AM. Participating will be Dominick Purpura, Moderator; Marc Colonnier; George Gerstein and Philip Nelson.

## SYMPOSIUM ON BASIC MECHANISMS OF THE EPILEPSIES.

November 18 - 21, 1968

The Broadmoor Colorado, Springs

**Organizing Committee:** Herbert H. Jasper, Chairman; Reginald G. Bickford; William F. Caveness; F. Kiffin Penry, Secretary; Alfred Pope; Arthur A. Ward, Jr.; Dixon M. Woodbury.

**Opening Address:** H. Houston Merritt, New York.

**Introduction: The Definition and Challenge of Epilepsy as a Clinical and Experimental Phenomenon:** Herbert H. Jasper, Montreal; Alfred Pope, Boston; Arthur A. Ward, Jr., Seattle.

**Structural Organization of the Cerebral Cortex:** J. Szentagotai, Budapest; Disc.: Marc Colonnier, Montreal.

**Ultrastructural and Neurochemical Studies of the CNS in Normal and Convulsive States:** Eduardo De Robertis, Buenos Aires.

**Biophysics of Nerve Membrane:** J. Walter Woodbury, Seattle; Disc.: Charles F. Stevens, Seattle.

**Cerebral Energy Metabolism and Membrane Phenomena:** Henry McLwain, London; Disc.: Janet Passonneau, St. Louis.

**Immunological Mechanisms of Receptor Sites:** J. del Castillo, San Juan.

**Central Synaptic Transmitters:** David R. Curtis, Canberra; Disc.: Motoy Kuno, Salt Lake City.

**Action of Convulsant Drugs:** Don W. Esplin, Salt Lake City; Disc.: William E. Stone, Madison.

**Pharmacology of Central Synapses:** George B. Koelle, Philadelphia.

**Excitatory and Inhibitory Mechanisms in**

**the Brain:** Sir John C. Eccles, Chicago; Disc.: T. Oshima, Chicago.

**The Epileptic Neuron — Chronic Epileptogenic Foci in Animals and Man:** Arthur A. Ward, Jr., Seattle; Disc.: Eli S. Goldensohn, New York.

**The Epileptic Neuron — Acute Effects of Local Epileptogenic Agents:** Cosimo Ajmone-Marsan, Bethesda; Disc.: David Prince, Palo Alto.

**Properties of Isolated and Deafferented Neurons:** Seth Sharpless, Bronx; Disc.: Lester T. Rutledge, Ann Arbor.

**Mechanisms of Secondarily Induced Epileptic Discharges — the "Mirror Focus":** Frank Morrell, Palo Alto; Disc.: Samuel Barondes, Bronx.

**Role of D.C. Polarization Changes in the Epileptic Discharge:** Heinz Caspers, Munster; Disc.: James L. O'Leary, St. Louis.

**Neuronal Mechanisms Generating the Electroencephalographic Manifestations of the Epileptic Discharge:** Otto D. Creutzfeldt, Munich; Disc.: Daniel A. Pollen, Boston.

**Mechanisms of Spread and Propagation of the Epileptic Discharge:** Herbert H. Jasper, Montreal; Disc.: Dominik P. Purpura, Bronx.

**Sleep Mechanisms in Relation to Epilepsy:** O. Pompeiano, Pisa; Disc.: Carmine Clemente, Los Angeles.

**Factors in Stability and Seizure Susceptibility of Immature Brain:** Dominik P. Purpura, Bronx; Disc.: Stanley M. Crain, Bronx.

**Ontogeny of Focal Seizures:** William F. Caveness, Bethesda; Disc.: Dixon M. Woodbury, Salt Lake City.

**Sensory Precipitation and Reflex Mechanisms in Epilepsy:** Reginald G. Bickford, Rochester; Disc.: Robert Naquet, Marseille.

**Inhibitory Mechanisms in Epilepsy:** Eric Kandel, New York; W. Alden Spencer, New York; Disc.: P. Andersen, Oslo.

**Neurochemical Mechanisms in Epilepsy:** Donald B. Tower, Bethesda; Disc.: Louis Sokoloff, Bethesda.

**Mechanisms of Action of Anticonvulsant Drugs:** Dixon M. Woodbury, Salt Lake City; Disc.: James E. P. Toman, Chicago.

**Genetic Aspects of Seizure Susceptibility:** Guy M. McKhann, Palo Alto; Eric M. Shooter, Palo Alto; Disc.: Julius Metrakos, Montreal.

**Systemic Electrolyte and Some Neuroendocrine Mechanisms in Epilepsy:** J. Gordon Millichap, Chicago; Disc.: Pacla Timiras, Berkeley.

**Neuron-Glia Relations and Control of Electrolytes:** Richard K. Orkand, Salt Lake City; Disc.: Sanford L. Paaly, Boston.

**Model Neurons and Neuronal Systems:** Marvin L. Minsky, Boston; Disc.: Valentino Braitenberg, Tübingen.

**The Pathophysiology of Epilepsy:** Alfred Pope, Boston; Disc.: L. S. Wolfe, Montreal.

**Summary:** E. Earl Walker, Baltimore.  
**Reflections on Epilepsy:** Wilder Penfield, Montreal.