A Lecture

ON

THREE CASES OF SPONTANEOUS SUBARACHNOID HAEMORRHAGE:

WITH ESPECIAL REFERENCE TO THE OCCURRENCE OF MASSIVE ALBUMINURIA AND KORSAKOW'S SYNDROME.*

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(With Special Plate.)

DURING recent years the subject of spontaneous subarachnoid haemorrhage has received increasing attention. Lumbar puncture has shown that this may occur without being necessarily fatal, and that its occurrence is indicated by a symptom-complex which in itself may be diagnostic. With this knowledge fresh cases are being more frequently recorded.

In each of the three following cases the diagnosis was made, not only by the characteristic mode of onset, but also by finding blood diffused through the cerebro-spinal fluid. Two were fatal. In one of these the diagnosis was confirmed at post-mortem examination, in the other a necropsy could not be obtained.

CASE I.

On the evening of November 10th, 1928, a young man, aged 26, was admitted to the Montagu Hospital, Mexborough, under the care of Dr. J. A. R. Lee, with the following history. He was returning home from his work as a bank clerk, and whilst walking along the street is said to have stepped off the footpath to allow someone to pass. In so doing he slipped and fell. It is also said that he had his hands in his pockets at the time and that he fell heavily head forwards. He did not attempt to get up. He was found to be unconscious, and was at once removed to hospital.

On admission he was completely unconscious; the limbs were in a condition of extreme rigidity. The arms were flexed across the chest so tightly that his clothes had to be cut off—the right arm being more rigid than the left. The legs were extended. The pupils were contracted. There was thought to be a slight facial paralysis, but this was uncertain. He remained in the same condition during the night.

The next morning his temperature was slightly raised (above 100° F.). There was some vomiting, and occasional twitching of the face. At 9 a.m. he was more conscious, looked about, but did not respond to questions. He is said to have spoken once. A sample of urine drawn off was found to contain a good deal of albumin.

When I saw him in consultation, about 10 p.m. of November 11th, the condition was as follows. He lay quietly on his back. The eyes were open and he looked about with a vacant confused expression. He did not show any response to questions or orders. The pupils were equal; of medium size, and reacted to light. There was occasional fibrillary contraction in the muscles of the left side of the face. The arms were flexed across the front of the chest, and any attempt at extension was strongly resisted. This was more marked on the right than on the left. No reflexes could be obtained in the arms. The abdominal reflexes were absent. The legs were rigidly extended—the right more than the left. The knee-jerks were brisk, also the ankle-jerks.

The plantar reflexes were markedly and briskly flexor on stimulation of the inner sole, and as markedly and briskly extensor when the outer margin was stimulated. In the right foot this persisted on repeated trials; in the left foot toe extension disappeared after a few repetitions. On simultaneous stimulation of the inner and outer parts of the sole the big toe remained motionless; the remaining toes were flexed.

The systolic blood pressure was 120, pulse 104. There was incontinence of urine. A catheter specimen showed a very heavy cloud of albumin. Except for a slight abrasion on the forehead, there was no evidence of any injury to the head. Lumbar puncture showed greatly increased pressure. The fluid was deeply blood-stained throughout. Unfortunately the specimen of cerebro-spinal fluid came to grief in transit to the laboratory, and therefore we have no detailed report on it.

On November 13th the temperature rose to 101° F. and remained there. The rigidity persisted. On November 15th the report was: "He answers 'Yes' and 'No' sometimes when questioned, but I think he does not understand the questions. He is taking a fair amount of milk and water. The urine yesterday contained less albumin and to-day only a faint trace."

* Delivered before the Chesterfield Division of the British Medical Association.

He remained in much the same condition until death on November 18th, just a week after the attack.

A necropsy was made on November 19th by Drs. J. A. R. and R. T. Lee, who report as follows:

"We examined the skull very carefully and could not find any fracture. The whole surface of the brain was congested evenly, not in patches. There was some dark blood at the base of the skull in the region of the pons. It was clotted, and seemed recent. Nothing was found abnormal in any other organ."

A careful examination of the brain by Professor Douglas failed to show any aneurysm. His report is as follows:

"Generalized congestion of the pia mater, chiefly on the vertex. On the whole, the congestion is more marked over the surface of the left hemisphere than the right. It is more intense in the region of the left motor area, comparatively slight over the right. On the mesial surfaces the vessels are engorged rather more on the right side than on the left. The appearance is suggestive of acute changes in the meninges over the left frontal lobe. Similar changes are seen in the interpeduncular space and on the anterior part of the pons. Considerable discoloration at the basal surface of the frontal lobes on both sides, more marked on the right, where there is definite haemorrhage under the surface of the pia mater. No recent bleeding was seen in any of these parts. On cutting through the cerebellum a recent clot of blood about the size of a pea, and sharply defined, was found in the region of the inferior vermis, apparently just above the fourth ventricle in the middle line. It was semiliquid, and did not appear to be intimately mixed with tissue substance."

Portions of the brain from various parts were examined microscopically by Miss E. C. Eaves, M.D., who reports as follows:

Pathological Changes.

The vessels in the meninges were congested, and haemorrhages had occurred in different regions, most marked in the frontal lobe. In some areas the clot had become partly organized, but elsewhere the haemorrhage was apparently very recent, and extended a little way into the substance of the brain. Multiple small haemorrhages were present in the brain substance or around small or medium-sized vessels. The possibility has to be considered that the blood had tracked down the adventitial sheath of the vessel from the meninges. There were, however, situations in which a leakage of blood from the vessel could be observed. Haemorrhages were present in the corpus striatum and optic thalamus, as well as in the frontal lobe.

The changes in the tissue may be described as: (a) secondary to the haemorrhage; (b) primary changes.

(a) *Secondary*.—Some parts of the cortex appeared softer than normal. Microscopically there were numerous cells of the mesoglia type, and in some places there were groups of Körnchenzellen. The function of the latter is probably to aid in the removal of the debris of the softened nervous system. Friedmann,¹ in his experimental studies on the nervous system, has shown that these cells may appear in two or three days. The nerve cells exhibited a considerable degree of chromatolysis.

(b) *Primary Changes*.—The vessels of the meninges were thickened and stained in an abnormal manner with methylene blue (Fig. 3), as did some of the vessels in the nervous system. It was considered worth while to make an analysis of the cerebral cortex to ascertain if an unusual amount of calcification had occurred. The calcium amounted to 19.8 mg. per 100 grams of cerebral cortex, an amount about five times in excess of that found normally at the age of this patient,² and four times that found in old age accompanied by arterio-sclerosis. In some parts of the frontal cortex the vessels, and particularly the capillaries, seemed unusually numerous, even allowing for the fact that the congested and dilated condition would make them appear so. (Fig. 1.) This increased number of vessels was, however, limited to certain areas, and here the vessel walls were markedly different from the normal. There was proliferation both of the tunica intima and adventitia, and plasma cells were present. (Fig. 2.) There were many cells of mesoglia type in apposition to or near the vessel wall. Some of the blood vessels, and especially the very dilated ones, exhibited a thinning of the wall at intervals, although the vessel wall was elsewhere thickened. There was a slight increase of neuroglial cells.

The pathological findings are similar to those of a syphilitic arteritis. In this disease intracranial aneurysms may occur even in a young adult, and cause death from haemorrhage (Aldren Turner³). The limited area of the increased number of vessels is, however, difficult to explain. Nissi⁴ has shown also that plasma cells are not pathognomonic of general paralysis, nor of syphilitic disease of the brain, but that they may be produced by any slight irritation, and are present in many diseases of the brain.

It is necessary to refer also to the experimental work of Friedmann.¹ He found that a destruction of nervous tissue may act as an irritant to the mesodermal tissues. In as little time as five to ten days the vessels of the irritated area increase in number, and are thickened by proliferation of the endothelial wall. The softening of the nervous tissue brought about by the haemorrhage could therefore act as an irritant and bring about a new formation of vessels in a limited area.

The possibility of an angiomatous condition may be considered. Calcification may occur to an unusual degree in this condition, as in two cases described.² However, haemorrhage was not a feature in these cases, although the patients, being epileptics, had numerous falls.

The histological report shows that, although this man was thought to be in sound health prior to his fall, his intracranial arteries were definitely and considerably abnormal. The changes found in them are, in Miss Eaves's opinion, not inconsistent with a syphilitic origin. Unfortunately, owing to the loss of the specimen of cerebrospinal fluid in transit to the laboratory, the means of confirming this or otherwise is wanting. At any rate, whether syphilitic or not, no aneurysm was found after a careful search of the hardened specimen, and the distribution of the blood did not point to one single focal origin. The presence of so large an amount of calcium in the brain points to changes of rather wide extent and long development.

Relation of Trauma to Haemorrhage.

This raises a point of much practical importance. Had this fall occurred whilst the man was at his work the question of compensation would almost certainly have arisen.

The history is that "he was walking along the road and, the footpath being narrow, he stepped off it to allow a lady to pass; that in so doing he slipped, and fell forward. As he had his hands in his pockets he could not save himself, and fell on to his forehead." There was only a slight abrasion on the forehead, and a careful examination of the skull showed that there had been no fracture or obvious injury to the skull. It is obvious that immediately before he stepped aside there had been no intracranial haemorrhage, for this does not begin without symptoms. On the other hand, immediately after he had fallen he was found to be unconscious and made no attempt to pick himself up, so that within that short time the haemorrhage must have been considerable.

Probably the immediate cause of the haemorrhage was the sudden muscular effort in trying to save himself when he slipped. This would be quite in keeping with the history often recorded in cases of spontaneous subarachnoid haemorrhage, in which the seizure occurs with surprising suddenness whilst lifting something comparatively slight, such as a coal-pan, or, as in a case under my care some years ago, hitting a four at cricket. If that is so, the actual fall would be not merely due to the slip but to the onset of haemorrhage, and with this rapidly extending he quickly became unconscious and rigid.

The second point of interest is that within a few hours of the attack this patient showed transient massive albuminuria. This sign was first recorded by Widal in 1903,³ and since then has been observed from time to time. It is by no means always present. Symonds⁶ points out that, although there is good physiological evidence that this may be due to disturbance of nervous centres concerned with renal secretion, hitherto in such cases the absence of renal disease has never been clearly established on clinical or post-mortem evidence. In this case the clinical evidence is that the albumin practically disappeared in a few days, and at necropsy the kidneys were found to be healthy.

In the second and third cases the point to which I would call attention was the presence of typical Korsakow's syndrome.

CASE II.—Spontaneous Subarachnoid Haemorrhage in a Healthy Man of 36: Prolonged Pyrexia: Korsakow's Syndrome: Recovery.

A married man, aged 36, always very strong and active, on January 6th, 1928, in the evening, whilst visiting at a house, felt a sudden intense pain in the back of the neck, much more severe than anything he had ever felt before. With great difficulty he managed to walk home. He vomited several times, but the pain persisted. Next day there was a temperature of 100°, headache, pain in the eyeballs, and stiffness of the neck.

With rest in bed he improved during the next day or two. On January 9th he came downstairs, but still felt pain and stiffness in the neck. The next day he stayed in bed and took a purgative as he had been somewhat constipated. After the bowels had acted he fell asleep and was sleeping quietly at 11.30. About 2 p.m., when food was taken to him, it was found that he could not be roused, and, in fact, was comatose.

I saw him about 3.30 p.m. He was deeply comatose, the respirations were noisy and irregular, of Cheyne-Stokes type. He was pale, slightly cyanosed, the eyes half closed, pupils

small, divergent squint. The pulse was soft, small, and showed increased frequency. The legs were flaccid; no knee-jerks could be obtained, and no Kernig's sign. There was slight toe extension on the right foot, no response on the left.

The diagnosis of subarachnoid haemorrhage, due to a leaking aneurysm, was made. Lumbar puncture showed a free flow at rather increased pressure, deeply blood-stained throughout. The Wassermann reaction was negative.

On January 11th he was rather better; he had had a restful night, sleeping most of the time; he would wake up and respond vaguely when spoken to, but was very dull and heavy. The pupils were normal in size, equal, and reacted normally; there was no cranial palsy, and no head retraction. He passed urine voluntarily. Knee-jerks were still absent. Toe flexion on both right and left. The next day his condition was much the same—he was rather more restless. There was slight toe extension on both sides. Lumbar puncture again gave blood-stained fluid at slightly increased pressure.

During the next ten days his condition did not change very much; there was pyrexia every day, with a pulse always about 130. At one period catheterization was necessary, and this was followed by some cystitis, which soon passed off. During all this time the knee-jerks were constantly absent, whilst the plantar response varied. He was chiefly in a lethargic stuporose state, but could be roused, and he recognized those around him in a confused way.

On January 20th there was some ptosis and nystagmus, also slight Kernig's sign. The temperature returned to normal on January 25th; pulse 84, still very lethargic but clearer in between. A faint knee-jerk was obtained in the right leg for the first time on this day.

At the beginning of February he was having restless nights and sleeping all day. When aroused he was in a state of complete euphoria. He would smile, and say he was quite well; he had no idea of time or place; would begin an answer sensibly and at once wander off into confused nonsense. No nystagmus was present, and the fundi were normal. Knee-jerks were present, though very feeble.

He improved gradually, and by February 23rd he was coming downstairs each day. Mentally he presented the features of Korsakow's syndrome. There was smiling euphoria, together with unblushing accounts of what he had done, which were quite imaginary. Thus, in the presence of his wife and doctor, when asked what he did yesterday, he said at once that he had been out walking in the garden, although he had not been out of the house for six weeks. When corrected by his wife, he said, "Oh, didn't I?" and did not seem at all bothered. This mental condition continued for several weeks.

On June 20th, 1928, he was going about as usual, and driving his motor car for long distances (100-mile runs). He was sleeping and eating well. Slight headache and vertigo were present at times. His speech was slow and deliberate, with definite hesitation for words at times. Memory for recent events was not very good. His expression was rather fixed; there was slight tremor of the lids, but no nystagmus; knee-jerks were still only obtained on reinforcement.

At the present time, more than a year after his attack, he has been able to resume his full work, which involves a good deal of public speaking. He finds that for this he is obliged to rely on notes such as he did not require previously.

As regards the possible cause which set the bleeding going in this case it is said that just before going to make the call where it happened the man had been playing very vigorously with his little child—throwing it up in the air and lifting it on to his shoulders, etc. The second haemorrhage was no doubt set going by the effort connected with the act of free purgation. It emphasizes the importance of complete and prolonged rest after even the slightest subarachnoid haemorrhage.

This case appears to have been one of leaking aneurysm in which there was a primary small leak and a secondary larger but not fatal one.

The last of the three cases I have to record proved to be primarily a case of chronic granular nephritis, of which the subarachnoid haemorrhage was only an item. The presence of Korsakow's syndrome showed itself in rather a striking way.

CASE III.

A woman, aged 44, was brought into my ward on a stretcher on August 7th whilst I was going round, and before leaving I looked at her and asked some questions. She was rather confused, but said she had suffered from fits for several years, and that she was quite accustomed to them. I assumed that one had occurred in the street, and that she had been brought in because she was in a state of post-epileptic confusion. The next day when I saw her my house-physician said that lumbar puncture

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FIG. 1.—Cerebral cortex (frontal lobe). All the blood vessels dilated and more numerous apparently than normal. The spaces round the vessels may be due to faulty fixation. Magnification 60.

FIG. 2.—Proliferation of endothelial layer of vessel wall. Blood outside the vessel. There are two or three large microglia cells, only the nuclei of these being distinctly stained. Magnification 510.

FIG. 4.—Cerebral cortex (frontal lobe). Magnification 280.

FIG. 3.—Apparently calcified vessels, near corpus striatum; similar vessels in meninges. Magnification 60.

From microscopic sections prepared by Miss E. C. Eaves, M.D., Lecturer on Physiology, University of Sheffield.

showed a blood-stained fluid at increased pressure, and that he had obtained the following history from her husband.

Three weeks previously she had sudden excruciating pains in the nape of the neck, with severe headache and vomiting for three days. After that she had pain behind the eyes, and headache. The evening before admission she had another sudden pain in the head, fell down unconscious, and remained so until shortly before coming in. She had never been subject to fits, and her statement to me the day before was entirely a confabulation.

During the next few days this condition of Korsakow's syndrome persisted; she would at any time, when asked what she had for dinner, give a detailed account of an ordinary plain dinner, with particulars elaborately filled in, although taking nothing but liquid food.

Further haemorrhages occurred, and she died on August 18th. She had albuminuria, retinal haemorrhages, and a blood pressure of 200/125. No necropsy was allowed.

The occurrence of Korsakow's syndrome in subarachnoid haemorrhage has been emphasized by more than one observer. Herman⁷ of Warsaw, in a paper based on eighteen cases of subarachnoid haemorrhage seen in Flatau's Clinic, writes: "Chez ces malades apparait fréquemment le syndrome net de Korsakow." He states that it was first noted by Goldflam, and later by Mackiewicz.

Goldflam,⁸ writing in 1923, says:

"In nearly all cases, at the height of the illness, certain psychic disturbances are observed, such as disorientation as regards time, places, and persons. These are generally neither severe nor constant. In some cases these disturbances come to the fore from the beginning, and consist of delirium, motor unrest, and confabulation, corresponding with Korsakow's syndrome; a good description is given by Flatau. This psychosis may outlast the fever and meningeal symptoms by weeks. Gradually the patient recovers from his delirium, corrects his confabulation himself, and the psychic symptoms disappear completely in, say, six weeks, as occurred in one case. In their place there remains for a time forgetfulness of actions just performed—for instance, the patients tell the same story many times over without realizing it. On the whole, it may be said that memory is well retained in such cases for events previous to the illness, but there is more or less complete amnesia, either for the whole duration of the illness, or for the first few days.

"It is known that Korsakow first described his syndrome in cases of polyneuritis, hence the name 'polyneuritic psychosis.' Since then the syndrome has been observed in a number of different conditions, from toxic and infectious diseases to cerebral contusion and incurable diseases, such as progressive paralysis and senile dementia. So far as I know, it has been described in subarachnoid haemorrhage only by Flatau, and yet it is present in all cases, more or less distinctly, not infrequently in a quite typical form. (It may be mentioned in passing that it is not found in cerebral apoplexy.) Whereas the prognosis in many of the conditions mentioned is absolutely bad, or grave, it is good in subarachnoid haemorrhage, for the psychic disturbances disappear completely. It may be assumed that the anatomical changes in these cases are of a temporary nature, and that the alterations in the cortex brought about by the extravasations of blood are capable of complete regression."

Mackiewicz's paper appeared in a Polish publication which I have not been able to obtain, nor has it been possible to trace the exact reference to Flatau's publication said to have been made in 1917.

The question of prognosis was of considerable importance in the second case. The somewhat prolonged persistence of mental symptoms caused considerable anxiety, as the patient held a highly responsible position involving much speaking in public, and his whole future depended on being able to continue this. In his case Flatau's views have been fully confirmed.

Symonds⁹ has divided the cases into four clinical groups, on the following lines.

Group 1.—Sudden large haemorrhage leading to coma and death within a few hours.

Group 2.—Initial haemorrhage sudden and severe, but it is checked; initial coma gives way to mental confusion with severe headache; ends in recovery or further attack and death.

Group 3.—Initial haemorrhage small; headache and vomiting may lead to confusion, coma, and death; or the patient may recover.

Group 4.—Slow leaking; gradual headache and vomiting. [This group is somewhat hypothetical.]

Of the cases here recorded No. 1 would come under Group 2, whilst Nos. 2 and 3 would come under Group 3.

If there is extensive and advanced general arterial disease, syphilitic or other, there is no *a priori* reason why a vascular rupture should not occur in a meningeal vessel just as it does in one that is intracerebral. So also if the case is one of some general disease in which a tendency to haemorrhages is a recognized feature, such as purpura, haemophilia, scurvy, leukaemia, etc. Also in cases of embolic aneurysms due to septic infection the possibility of a meningeal bleeding must be always present. In all the above circumstances subarachnoid haemorrhage may occur, and the reason why it does so less commonly than intracerebrally is due to various factors which need not concern us here.

In some of the above groups of cases the immediate source of the bleeding has been a pre-existing aneurysm demonstrable at necropsy. In others, although no aneurysm is found, the site of bleeding is strongly indicated by the appearance of the meninges at and around some particular area, and even the ruptured vessel may be found—usually a very difficult matter. Apart from these groups, which together form the bulk of the cases of subarachnoid haemorrhage met with at necropsy, cases occur from time to time which cannot be included under any of these headings. Such cases have certain features in common. Thus they often occur in comparatively young people—early adult life; they usually come as bolts from the blue in persons thought to be in excellent health; they are accompanied by pyrexia for the first week or so; there may be subretinal haemorrhages. In a considerable number of the fatal cases of spontaneous subarachnoid haemorrhage of this class an aneurysm at the base of the brain has been found, and it has been suggested that such would be found more often were the examination made with sufficient care. These have been termed "congenital aneurysms," and it is assumed that they have arisen from some localized defect of development at this particular point, usually in the circle of Willis.

Parkes Weber⁹ has pointed out that the term "congenital aneurysms" is inaccurate, and does not express what is meant, and that the assumption that there is an aneurysm in all these cases, whether it is found or not, overlooks the fact that a rupture may occur without necessarily being preceded by an aneurysmal dilatation, such as might occur in undue fragility of vessels or in a naevoid angiomatous condition, either of which might give rise to a spontaneous subarachnoid haemorrhage under some slight extra strain. Such alternatives seem to fit in rather better with some of those cases in which there are recurrent attacks of "leaking aneurysm" without a fatal result occurring. It is difficult to think that an actual aneurysm at the base of the brain which has once leaked can be in such a physical condition as to leak repeatedly at considerable intervals without a fatal bleeding ere long. On the other hand, it is quite conceivable that slight ruptures of vessels might arise in different parts of the meninges from fragile vessels or from a naevoid condition, and each in turn be sealed up and permanently closed.

The point at issue seems to be, are these cases of leaking "congenital" aneurysm occurring in younger persons merely a defect at one point of otherwise normal vessels, or are they a localized manifestation of a wider defect of intracranial vessels, possibly of developmental origin? If the latter, then whether the haemorrhage is preceded by an aneurysmal dilatation or not would appear to be a matter of more or less secondary importance, the primary cause being an abnormal state of the vascular development. So far as we have been able to review the literature of these cases we have found very few records as to the microscopical condition of the intracranial vessels other than some of the basal arteries. As a rule the pathological information available is limited to the naked-eye changes found at necropsy or the microscopic examination of the aneurysm itself. Case i, which clinically conformed in every point with the classical description of spontaneous subarachnoid haemorrhage, is therefore recorded because a detailed examination of the whole brain has been made and definite evidence of rather widespread vascular changes

has been found. It suggests that we are dealing with such a widespread abnormal vascularization of the intracranial area. My thanks are due to Professor Douglas and Miss Eaves for their valuable help in the pathological investigation of this case.

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POST-ENCEPHALITIC DELINQUENCY.*

BY

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In a recent book¹ I recalled that during the war, from September, 1915, to February, 1916, first at Commercy and later at Verdun, as medical officer in charge of the neuropsychiatric services, I was struck by several cases of encephalomyelitis presenting peculiar mental symptoms. It was these mental forms of the disease that first put me on the road to discovering epidemic encephalitis, which, along with Moutier and Calmettes, I then called subacute encephalomyelitis. In a series of 208 patients studied at Commercy and Verdun, three had attracted my attention by their symptoms of fever, delirium, and mental confusion, which, without investigation, had been attributed to typhoid fever. One of these patients, suffering from amnesia, affective indifference, transient excitation, followed by asthenia and indolence, suggestibility, faulty judgement, stereotyped gestures, and negativism, resembled a case of dementia praecox. A fourth patient, a few weeks after influenza, had become almost demented and suffered from somnolence and intellectual collapse. A fifth had difficulty in speaking, an uncertain gait, headache, intention tremor, hemiparesis, and hemianaesthesia of the right side. A sixth, whose illness at the inception resembled, but was proved not to be, typhoid fever, suffered from diminished vision, clonic spasms of the eyes, unaccompanied by lesions of the fundus, and opisthotonos. Finally, a seventh had been seized with epileptic crises, with progressive mental deterioration, speech defects, and other symptoms too discordant to be attributed to cerebral tremor.

Eight further cases observed afterwards at Bar-le-Duc showed ill-defined mental symptoms—for example, impaired memory, intellectual deterioration, hebétude, and somnolence, but unassociated with modification of the cerebrospinal fluid. In most of these cases an infectious state was noted, accompanied by febrile reaction, delirium, fatigue, inertia, and rigidity. Although the cause of the condition, in spite of researches then being carried out, was unknown, the patients improved under treatment, but several, after the acute phase had passed, had a fixed expression, slackening down of their actions and thought processes, and in some cases a tremor—symptoms which are singularly allied to those of what is now called epidemic encephalitis. As well as these purely mental forms I described convulsive choreic, meningitic, hemiplegic, ponto-cerebellar, ponto-medullary, acute ataxic, and anterior poliomyelitis forms. Of this polymorphism Professor Achard wrote:

"It might at first be believed, and was as a matter of fact believed for some years, that the disease described in France and in Austria in April and May, 1927, respectively, was not the same, so different were the two pictures drawn of it. Our fellow-countrymen had described a disease of very diverse forms; the Viennese author a disease very simple in its symptoms. . . . It was not until later that it was noticed that the disease could assume varied, changing, and unexpected aspects, and that this very diversity appeared to be one of its essential attributes. . . . Von Economo's description represents only one type of the disease.

* Abstract of a lecture delivered in English at the Universities of Glasgow and Edinburgh, on the invitation of Professor Currie and Professor Edwin Bramwell, April 22nd and 29th, 1929.

In strict justice it is the most exact description that has the greater merit, and it is Professor Cruchet's publication, which preceded von Economo's by a few days, that takes priority."

I cite these remarks less to return to the question of priority than to emphasize the importance of the polymorphism of encephalitis, a character which I considered essential from the beginning. This polymorphism, which is found in all the clinical forms of encephalitis, is an attribute of the mental forms with which I wish to deal more particularly.

The commonly described clinical syndromes of mental disorder can be recognized only with difficulty in epidemic encephalitis. The maniacal, melancholic, and chronic delirious syndromes are never clearly defined. I have known encephalitic patients with delusions of persecution, but modified, unobtrusive, and unaccompanied by auditory hallucinations. Much the same is true of the cases of megalomania, and of those patients whose psychoses assume the obsessive form. The most common type resembles dementia praecox, but even then with sufficiently marked differences.

I will cite a case representative of the conditions I have mentioned.

THE CASE OF ALIX.

This patient was aged 10 when, on February 10th, 1920, the first symptoms presented themselves; his teachers noticed that his eyelids drooped, and that he had a vacant expression. His vision was blurred and he had diarrhoea. On February 13th, at about 6 o'clock in the evening, he fell asleep during evening preparation. He had a raised temperature and was taken to the school infirmary, where, though he went on sleeping at night, it was observed that his body would every now and then move spasmodically. The next day he went to chapel, but fell asleep there. The fever, spasmodic movements, and blurred vision persisted, and that same evening he was taken to his home in a motor ambulance. On his arrival he was put to bed. He was febrile, delirious, restless, and the spasmodic movements continued. He had no sickness and the diarrhoea stopped.

On February 15th he was very tired and his tongue was parched; movement of the head was accompanied by pains at the back of the neck; his eyes were almost closed and staring upwards; he seemed blind, and could recognize neither the doctor nor his parents. His breath was very fetid, and he could only be roused from sleep with difficulty. Owing to pains in his neck and his loins he was unable to sit up in bed. Kernig's sign was slightly positive. An examination of his blood yielded no relative information. The next day his condition was worse; his teeth were clenched, and he had difficulty in swallowing. For a few moments during the morning vision returned, but he soon fell into a state of torpor.

On February 17th he was able to read a few lines from a newspaper, though it seemed to him to be printed in very small letters. His condition improved; the attacks of sleepiness were shorter, trismus was not so pronounced, his tongue less parched, and he was able to drink some milk without too much difficulty. He had, however, rather restless nights. On February 18th and 19th the fever increased, and restlessness once more became marked, but on the 20th improvement became evident and continued until the 22nd, when he was allowed up for a little while. Delirium was still present, but occurred for shorter periods and less frequently. During the following days he improved so rapidly that he was able to go out and ride a bicycle during the first week in March. He still had attacks of sleepiness, however.

Six Weeks Later.

When I examined him about six weeks later he seemed much better. He would get up about 6 or 7, ride his bicycle, and live a normal life till about 5 or 6 in the evening, when he would go to bed tired and fall asleep. At 7 he would be awakened for dinner, but sometimes he would be so overcome with sleepiness that he would keep mouthfuls of food in his mouth without swallowing them. Occasionally he would fall asleep after each mouthful. On certain days he had to be carried upstairs, undressed, and put to bed; his sleep was restless, and sometimes he was even seized with somnambulism.

The changes in his character were very remarkable; he had become irritable and insolent, and had violent outbursts of anger which were followed immediately by sleepiness. He would repeat the same thing over and over again, and would constantly put the same questions. When a story was read to him he would ask for the explanation of each word, even when he knew it quite well. He was unstable and changed his games frequently. Before his illness he was, if anything, mentally advanced for his age; now he was rather backward. His memory appeared to have deteriorated and his intelligence to have slackened. He complained of fatigue and headache at the slightest mental