Four Cases of Intracranial Tumour with Mental Symptoms.

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THIS paper is an attempt to answer certain questions that have been raised by four cases of intracranial tumour which were studied at the bedside and subsequently followed out in the laboratory. In the clinical course of all four the mental symptoms were the most striking facts, and indeed only one (Case 3) presented other evidence of intracranial mischief. In considering cases of mental disease which exhibit gross cerebral disease, we can just as easily minimise as exaggerate the part the lesion plays in the production of the mental symptoms. The only safeguard is to think anatomically. As yet our knowledge of the finer structure of the brain is very imperfect, but the work of Fleschig has given us a general idea of the architecture of the brain; and it is in the light of those anatomical facts that we seek to investigate our cases.

As the cases are in themselves of considerable interest I have thought it advisable to give fairly complete reports.

CASE 1.—A. B—, a clerk, æt. 50, was admitted to Wodilee Asylum on December 7th, 1906.

He was well-built and well-nourished, but looked more than his age. The sclerotics were congested. The tongue was slightly furred. The temperature was normal. A high-tension pulse numbered 72 beats per minute. His gait was good. The pupils were equal and their reflexes normal. The knee-jerks were slightly exaggerated. No ankle-clonus was elicited, and both plantar reflexes were normal. No sensory disturbances were discovered. The other systems were free from gross lesion.

The alienation was characterised by rapid mental failure. Although no definite history of the onset could be obtained it was said to be of only a few weeks' duration. On admission he answered questions quite rationally, but in conversation showed considerable mental confusion, and had fantastical delusions, such as, "that his legs were made of glass." The confusion gradually became more profound and his reaction time very slow. For hours he would sit listlessly with his head on his chest. It was difficult to arouse him and he immediately lapsed back into his stuporose state. The stupor gradually became more profound and he began to pass his motions under him. Pulmonary congestion intervened, and he died April 22nd, 1907.

An examination was conducted fourteen hours after death.

The skull cap had average characteristics. The dura was normal,

but it bound the frontal lobes to the whole extent of the superior aspect of the small wing of the sphenoid. The convolutions were flattened and appeared dry and glazed. There was a fair quantity of fluid in both cisternæ. The pia was not adherent to the cortex. On the under-surface of the brain the longitudinal fissure was obliterated by a tumour which penetrated into the substance of both frontal lobes. As the greater bulk of the tumour was buried in the frontal lobes the visible portion was encircled by a rim of brain tissue which could be easily stripped from it. There were no traces of the olfactory bulbs or tracts, but the optic tracts, the circle of Willis and the knee of the corpus callosum were intact. The cranial nerves were normal. The frontal lobes were flattened, and especially in the right prefrontal region the convolutions were distorted and wasted. Both ascending frontal convolutions and the adjacent portions of the frontal convolutions were, however, fairly plump.

Although the tumour actually penetrated into the substance of the frontal lobes it appeared to do so by displacement and pressure atrophy and not by replacement of the brain tissue. The involvement of the lobes was wide rather than deep, but on the right side it reached as high as the medulla of the prefrontal lobe. The new tissue was sharply demarcated from the brain tissue, had a firm consistence and a structure like glandular tissue.

There was no thinning of the grey matter except in the neighbourhood of the growth. The vascular channels of the white matter were dilated. The fifth ventricle was greatly dilated and the lateral ventricles moderately dilated.

The basal nuclei and ponto-bulb were normal.

The heart was of average size with its left ventricle contracted. It displayed very healthy features.

The right lung was very emphysematous and showed marked engorgement. The left lung was also emphysematous. Its lower lobe appeared bulky. There was general engorgement, and in the lower lobe marked œdema.

The liver and spleen were very adherent to the diaphragm.

The kidneys had numerous cysts. Their capsules were adherent and their cortices thin.

The bowels, pancreas and adrenals were normal.

Microscopic examination: Tumour.—Although sharply defined from the brain tissue, which was condensed and rich in blood-vessels, the tumour had no capsule. It was rich in cells, which were arranged in whorls. At the periphery of a whorl the cells were roughly spindle-shaped and their elongated swollen nuclei stained faintly. At the centre of the whorl the cells, which were epithelioid in character, were massed together. As a rule the cell protoplasm was scanty and ill-defined, while the nucleus was round, swollen and faintly stained; but all variations from this to the spindle-cells were encountered. At the centre of some whorls there were hyaline bodies which were of a roughly oval shape, and contained several distorted nuclei concentrically arranged. The new tissue was well supplied with imperfectly formed blood-vessels that had undergone hyaline degeneration at an early period.

Cerebrum.—There was a poor sub-pial felting. The nuclei of the molecular layer were judged to be numerically increased, but it contained few fibres. At its periphery some spider-cells with thick processes were met with. The peri-vascular spaces were dilated. Some arterioles showed hyaline degeneration. The nerve-cells, which were situated in dilated peri-cellular spaces, were of good shape, had well differentiated processes and showed moderate chromatolysis. Their nuclei were centrally placed and stained well; a few showed vacuolation.

Cerebellum.—A very slight peri-vascular gliosis of the white matter was the only abnormal feature met with in cerebellar sections.

CASE 2.—M. P—, a hawker, æt. 34, first came under observation on November 13th, 1901. Neither a family nor a personal history was obtained, but the patient's circumstances were known to be poor and her habits irregular.

She suffered from a left-sided hemiplegia and showed the characteristic gait. The left facial muscles, especially those of the lower segments, were paresed. The tongue was protruded in the middle line. The mental condition of the patient made an investigation of a questionable internal strabismus of the left eye impossible. The left upper limb, which showed a typical contracture of the forearm and hand, was profoundly paresed; its muscle showed very marked myotatic irritability. The left lower limb was slightly paresed. There was no paresis of the right limbs.

The special senses, so far as they could be investigated, appeared normal.

The pupils were unequal; the left, which was the larger, was not circular. The right pupillary reflexes were active. The left pupil was immobile. The fundi had healthy appearances.

A general impairment of sensation, which was fairly profound in the case of the upper limb, was noted on the left side.

The muscles of the left hand were wasted, but although those of the arm and forearm were flabby, no wasting was noted in them.

Both knee-jerks were exaggerated.

An imperfect ankle clonus was obtained in the left lower limb.

Babinski's sign was sometimes elicited from the left foot.

The heart was not enlarged to percussion. There was a soft, limited murmur at the apex. The second sound was reduplicated.

The chest, owing to a lateral curvature of the spine, was of poor shape. The lungs were, however, healthy.

The digestive and renal systems appeared healthy.

She was discharged on July 21st, 1902. During her residence she showed marked mental confusion and was quite incoherent. She could not give a satisfactory account of herself and appeared to have vague delusions of impending danger. Her memory for recent events was very defective.

The patient was re-admitted on August 14th, 1904. She was stuporose, and when aroused appeared incapable of any mental receptivity. Occasionally she became restless, tried to get out of bed, picked at the bed-clothes and kept muttering to herself. When questioned she appeared to hear the voice, but, to judge from her behaviour, failed to grasp its meaning.

Her vital processes were all depressed. The temperature was subnormal. The extremities were cold and at times cyanosed. The pulse, which averaged ninety-two beats per minute, was irregular in force and rhythm, and of very poor quality. Sordes covered the teeth. The tongue was dry, cracked, and covered by a brown fur. There was a corneal ulcer on the left eyeball. A trace of albumen, some pus and a few equivocal tube-casts were noted in the urine.

On August 24th, 1904, she had a rigor, and on August 29th, 1904, died from broncho-pneumonia.

A *post mortem* was conducted twenty-six hours after death.

The skull-cap and base had normal characteristics. The dura was slightly congested. The pia was thickened in the region of the right Rolandic area, but was readily stripped from the cortex cerebri. The left cerebral hemisphere was larger than the right. The right ascending frontal and parietal convolutions and the adjacent convolutions were wasted. The other cerebral convolutions had a plump appearance.

In making transverse slices of the brain, the tumour was first discovered in one passing through the junction of the middle and lower thirds of fissure of Rolando. At this level the tumour was mainly confined to the white matter and was not sharply defined from the normal tissue; as it showed a tendency to spread into the white rays of the convolutions, the distribution of the new growth was most erratic. The anterior third of the corpus callosum was invaded, and at a point one inch behind the genu the whole breadth of the structure was affected. The neighbouring grey matter, with the exception of the two lips of the fissure of Rolando, was unaffected. The tumour tissue was of a dull grey colour, and had a worm-eaten appearance. There were numerous cysts. In a section passing through the middle of the insula the thickened semi-gelatinous pia-arachnoid was seen filling the sulci and fissures of the brain. The grey matter was relatively more affected. The tumour was very vascular; some of the vessels were of astonishing calibre. There was no general atrophy of the grey matter.

No macroscopic lesions were discovered in the basal ganglia, ponto-bulb or cord.

The heart was large: the cavities on both sides were distended with *ante-mortem* clot. The aortic valve was slightly incompetent. The mitral cusps were thickened and covered with warty vegetations; the orifice was judged to be contracted.

The aorta was atheromatous. At one place there was a dimpled appearance, as if of a commencing aneurysm.

There were numerous areas of consolidation in the right lung; these areas had a dull grey colour, and from their bronchioles pus was extruded.

The apex of the left lung was adherent. There was nothing to note in its parenchyma beyond marked congestion.

The liver was enlarged, and showed marked nutmeg colouration. The gall-bladder was distended with fluid bile.

The capsules of both kidneys were easily stripped off. Their cortices were somewhat atrophied.

Microscopic examination: The pia arachnoid.—The cellular elements

were greatly increased. The fibrous matrix was notably hypertrophied, and here and there some coarser fibres were noted. Many of the vessels, which were present in great numbers, had very imperfect walls. In the thickened areas these changes were exaggerated.

The tumour.—At the edge of the tumour long processes were seen meandering into the normal brain tissue. In the immediate neighbourhood of the processes there was a marked proliferation of the glial nuclei. The processes consisted of an intimate neuroglial felting rich in nuclei; numerous large spider-cells were encountered in them. When established, the tumour tissue showed a very dense neuroglial network with pseudo-cysts of various size. For about three-fourths of their circumference, the walls of the pseudo-cysts were well-defined and exhibited a very condensed network; the remaining fourth was ill-defined, not differing in texture from the tumour tissue. There was an abundant vascular supply; the walls of the vessels were in good condition.

The cerebrum.—The grey matter in the neighbourhood of the tumour was the seat of marked changes. There was a well-marked sub-pial felting. The molecular layer was moderately sclerosed. The vessels were tortuous, and showed hyaline change. At various points small hæmorrhages were noted; in one instance the process was quite recent; in the others a reactive proliferation of the neuroglia had occurred. The small pyramidal cells were represented by nuclei surrounded by the faintest trace of protoplasm; in some it tapered into an apical process. Some of the nuclei had a granular appearance. The large pyramidal cells were of similar appearance.

The rest of the grey matter showed no increase of the glia-cells. The vessels were in every way healthy, but some pigment granules were seen in the perivascular spaces. The nerve-cells were of good shape; the large pyramids showed a moderate degree of chromatolysis.

CASE 3.—J. H—, a carter, æt. 33, was admitted to Woodilee Asylum on August 16th, 1900.

There was nothing noteworthy in the family history.

Twelve years ago, and again six years ago, he had rheumatic fever. He was unmarried, and intemperate in his habits.

About the end of July, 1900, he attempted to drown himself in the Clyde, declaring he was an unpardonable sinner.

On the following day he was admitted in a state of profound depression. He refused to give any information about himself, and was so resistive that it took four men to undress him. He was fairly well nourished. His tongue was coated and his breath was very foul. The pupils were equal and responded to light and accommodation. There were no motor or sensory disturbances. The superficial and deep reflexes were normal. The pulse numbered 78 beats per minute, and the temperature was normal. No evidence of organic lesion was discovered in the cardiac, respiratory, or renal systems.

During the next month he remained in a state of profound stupor; he had to be forcibly fed; he passed his urine and fæces in bed. He occasionally became restless and had to be watched; once he flung himself into the fireplace, bruising his face.

On October 1st, 1900, he was noted to be taking his food and to be more cheerful.

For the next year there was little change in the patient's condition. He was usually found with his head bowed on his chest. He seldom spoke. On several occasions he was sent out to work, but was said to be of little use.

On September 16th, 1905, he was reported to have had a shock—no paresis detected—and thereafter became suddenly and wildly excited. He remained in this state for two or three days and then relapsed into his stupor. Since then he has had several seizures with no resulting paresis. The pupil reflexes were normal. The fundi oculi showed no change. No localising symptoms were ever noted. During the last months of life he lay, as a rule, absolutely still in bed in a stupid state, and lost flesh rapidly. After another seizure—March 20th, 1906—he became still more stuporose, and died on March 28th, 1906.

A *post-mortem* examination was conducted thirteen hours after death. The body was emaciated. There was a punched-out ulcer below the left external malleolus. No enlarged glands or scars were detected.

The skull-cap was of average size; the density was increased in parts and the vascular channels were of average depth. The dura was of average thickness and rather anæmic; it was slightly adherent to the cap, but markedly so to the posterior part of the left cerebellar fossa. At this situation it was also adherent to the left cerebellar hemisphere. The subdural space was dry. The cerebral convolutions had a fairly plump appearance. At the angle formed by the posterior border of the left cerebellar hemisphere with the incisura posterior cerebelli, a nodule about the size of a pigeon's egg was buried in the substance of the hemisphere. On the superior aspect of the hemisphere it occupied an area about the size of a shilling, and to this surface the tentorium was firmly adherent. It was of stony consistence, and on section was found to consist of a mass of calcareous material surrounded by a tough fibrous capsule. A fibrous cord was all that remained of the left vein of Galen. The basal vessels were healthy. There was considerable dilatation of the lateral ventricles, and their choroid plexus showed cystic degeneration. Several punctiform hæmorrhages were noted in the right internal capsule. The basal ganglia and ponto-bulb presented healthy appearances.

Beyond a moderate degree of fatty change in the cardiac muscle, there was nothing to note about the condition of the heart.

The right lung was markedly adherent to the chest wall. It weighed twenty-three and a quarter ounces and was greatly engorged. An irregularly wedge-shaped area of consolidation was situated at the anterior margin of the lower lobe; the cut surface showed patches of caseous necrosis. The bronchial glands were enlarged and were the seat of caseous necrosis.

The left lung was œdematous and congested.

The liver was large and very adherent to the diaphragm; it showed a moderate degree of fatty change.

The spleen was large and its pulp soft.

The kidney, supra-renals, pancreas and bowels showed no gross changes.

Microscopic examination: the tumour.—There was a well-defined capsule of coarse fibrous tissue with few cellular elements. The

contents of the capsule had undergone calcareous change. In the neighbourhood of the new growth there was a well-marked gliosis of the cerebellar tissue. At no point was a cellular infiltration encountered.

The cerebrum.—There was a slight subpial felting. No spider-cells were encountered. The molecular layer was free from neuroglial fibres. The vessels occupied slightly dilated spaces, but their walls were normal. The nerve-cells were of good shape; their nuclei were well stained, and no disintegration of the tigroid substance was noted.

CASE 4.—C. L—, female, æt. 33, a hair-mattress maker, was admitted to Woodilee Asylum on January 22nd, 1906.

Beyond the fact that her father died of consumption at the age of thirty-five there was nothing of importance in the family history. This was the first attack of alienation. She had always been in good health, in poor circumstances, and given to alcoholic excess. A week before admission she gave up work for no apparent reason. During the next three days she was quiet and absorbed; at night she kept talking to herself, as if she were praying. After this she became steadily excited; she saw and talked with imaginary people.

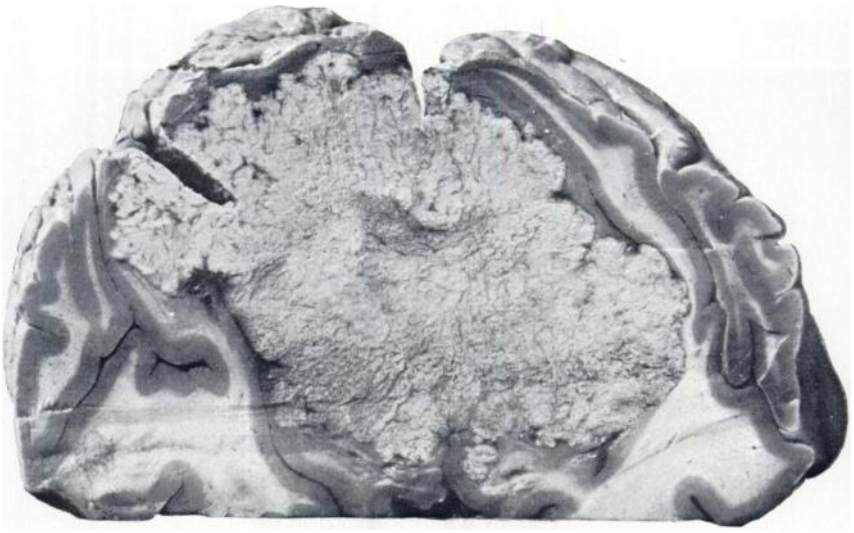
During her residence the patient was in a condition of great psychomotor excitement. The striking feature of the clinical picture was the extent to which her hallucinations appeared to predominate the mental symptom complex. She kept storming and raging at persons, who, to judge from her inconsequent utterances, were threatening her with violence. Occasionally the hallucinations were of a more pleasant nature and she developed a rollicking good humour. At first she mistook the identity of those about her, but soon became able to recognise them. She was very alert in noticing ward incidents and built them into her harangues. She answered questions irrationally. At first she complained of headache, later on it became more persistent and more severe. Owing to her excited condition the physical examination was imperfect. No evidence of gross lesion was discovered.

On April 30th, 1906, she suddenly complained of gastric uneasiness and of severe pain in her head; she sat down on a chair, and before the doctor arrived expired.

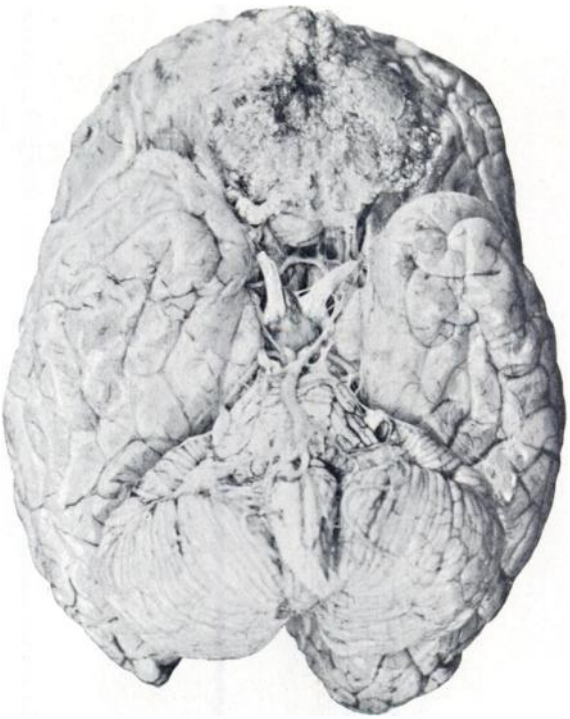
A *post-mortem* examination was conducted twelve hours after death. The body was well-nourished. On the inner aspect of the upper third of the leg there was a cicatrix about the size of a five-shilling piece. There were no enlarged glands or nodes.

The skull-cap was of average size, thickness and density; it was anæmic. The dura was unduly adherent. The skull base was symmetrical.

Over the right temporal lobe the membranes were bound to the cortex cerebri by a neoplasm. The area of attachment was limited in front by the posterior lip of the Sylvian fissure, and it extended backwards almost to the longitudinal fissure; its upper margin formed an angle of 110° with the posterior limb of the fissure of Sylvius, and its lower margin corresponded with the sulcus temporalis inferior. In the immediate neighbourhood of this area the dura was thickened and hyperæmic. Thrombosis had occurred in the superior longitudinal and right sigmoid sinuses. The basal vessels appeared



CASE 1.



CASE 1.

To illustrate Dr. R. M. Marshall's paper.

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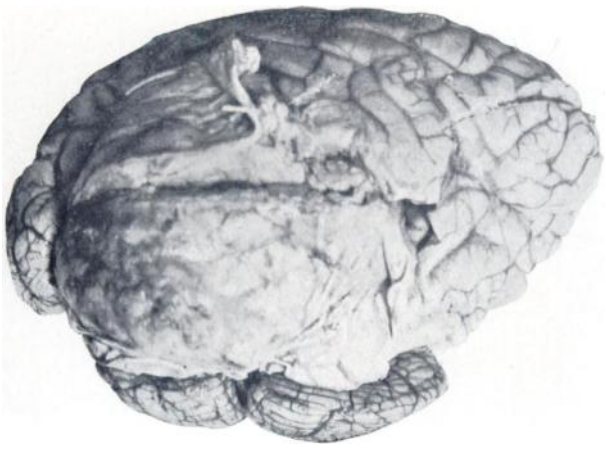
CASE 2.

CASE 4.



CASE 4.
To illustrate Dr. R. M. Marshall's paper.

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healthy. The two cerebral hemispheres were equal, and had a dry, glazed appearance. The convolutions were flattened, the sulci were shallow. When examined in transverse section the neoplasm was found to have extended mostly along the cortical surface, to which it had bound the pia arachnoid, and the dura. At its centre the tumour had penetrated to a slight extent into the white matter. Here the periphery was not clearly defined; areas of normal brain tissue were interspersed with areas of infiltration. The tumour tissue, as a whole, was firm and of healthy appearance, but here and there minute areas of softening were present. There was no dilatation of the lateral ventricles. The third ventricle contained a mass about the size of a pea; it lay between the layers of the choroid plexus, had a hyaline appearance and a cartilaginous consistence. No macroscopic changes were discovered in the basal ganglia or ponto-bulb.

The left ventricle of the heart was contracted. The cavities of the right side were distended with clot. No organic lesion was found.

The lungs were healthy.

The liver was of good size and consistence.

No morbid changes were detected in the kidneys, adrenals, pancreas or bowels.

Microscopic examination: the tumour.—The tumour had obviously originated in the membranes. It was impossible to differentiate the pia from the dura: they were replaced by a broad band of fibrous tissue rich in cellular elements. The involvement of the cortex cerebri was very erratic. The first change was a round-cell infiltration; the cells were massed together, and little more than their nuclei could be seen. Around these areas the brain tissue was disintegrating, but the neuroglial stain showed the presence of spider-cells in the *débris*. Two changes affected the cellular elements of the new growth: a proliferation of fibrous tissue, or a fatty infiltration of their scanty protoplasmic bodies. Vessels of large calibre and imperfect walls were encountered; the capillary system was very imperfect. No tubercle bacilli were noted.

The cerebrum.—The capillaries were moderately injected. The arterioles exhibited slight hyaline change. The peri-vascular spaces were dilated. A very imperfect sub-pial felting was present. The molecular layer was thinned, but free from fibres. The small pyramids were fairly normal in appearance; the large pyramids show marked disintegration of the tigroid substance.

Remarks.

In Cases 1, 2 and 3 the mental symptoms are identical—mental confusion with transient delusions passing more or less rapidly into profound stupor. These are given in our neurological text-books as the classical mental symptoms of brain tumour, and are attributed to increased intracranial pressure. But the association in cases of cerebral tumour of these mental symptoms with *post-mortem* evidence of increased intracranial

pressure is far from constant, and the present tendency is rather to emphasise their frequent association with tumours of the frontal lobes. Great diversity of opinion exists, however, regarding the facts of this association. Gianelli holds that the more the mental symptoms—torpor, intellectual arrest, weakness of memory—are manifested at the beginning of the illness, the more likelihood there is that the neoplasm is situated in the prefrontal lobes. Mills thinks that the only region of the brain in which focal lesions have produced persistent psychic symptoms has been the frontal lobe, and that mental symptoms are more frequently produced by lesions of the right prefrontal lobe; but Phelps asserts that the more positive and distinct the symptoms of mental default are, the more absolutely is the tumour limited to the left prefrontal region. Raymond emphasises the fact of penetration of the cerebral substance, and affirms that a neoplasm of the frontal lobe seldom remains latent when it penetrates, even very slightly, into the cerebral substance. Beevor, as far as we know the only author who has separated extra-medullary from intra-medullary tumours, finds that mental symptoms are more common in the latter class and are most marked when both lobes are affected.

In only one of our three cases (Case 1) did the tumour involve the frontal lobe, and this corresponds with the finding of other observers. Beevor correlates these mental symptoms with tumour of the centrum ovale. Case 2 is an example of this. Putnam and Williams, who have investigated all recorded cases of tumour of the corpus callosum, consider that the only symptoms attributable to this class of tumour are mental—mental failure, stupor, and sometimes hallucinatory delirium. Mott and Barratt record three cases of tumour of the third ventricle, which, by blocking the foramina of Monro or the aqueduct, prevented the escape of fluid from the lateral ventricles and produced a condition of internal hydrocephalus. They comment on the somnolent condition, the intellectual failure, the semi-comatose and even comatose condition of their patients. Case 3 offers a striking parallel to these cases.

Any attempt, therefore, to correlate such a clinical picture with tumours of the prefrontal or frontal lobes ends in confusion; an attempt which undoubtedly arises from the current anxiety to invest the prefrontal lobe with attributes which exist only in the terminology of the psychologist. The prefrontal lobes,

considered ontogenetically, are terminal cerebral zones; they are the anterior nodal points of long association fibres which join the frontal association centres with the posterior association centres. These fibres run backward in the tapetum, which is usually described as a part of the corpus callosum, although many observers hold that it is quite independent of that body. Now, any lesion which affects the integrity of this association scheme, isolates the frontal association centres from the posterior association centres. This isolation will be more or less complete in prefrontal tumours, bilateral intra-medullary tumours, tumours of the centrum ovale and tumours of the corpus callosum. But, as we have seen, the mental symptom-complexes which Mills, Phelps and Gianelli correlate with tumours of the prefrontal lobes, Beevor with bilateral intra-medullary frontal tumours and tumours of the centrum ovale, and Putnam and Williams with tumours of the corpus callosum, are identical —slow or rapid mental failure ending in dementia, torpor or stupor. It then appears that tumours which isolate the frontal from the posterior association centres present a constant clinical picture.

In Cases 1 and 2 this association scheme has been obviously affected, but in Case 3 we cannot show an anatomical destruction of its integrity. A moment's reflection, however, on the mechanism of dilatation of the lateral ventricles will convince us that the structures which form their walls—the basal ganglia, the fornix, the corpus callosum, and the tapetum—must bear the brunt of the distending force. The long association fibres will therefore be early subjected to pressure. Now, the effect of pressure on nerve-fibres varies, according to the degree of pressure, from impairment to complete loss of their power of transmitting nerve impulses. But any interference with the transmission of nerve impulses along those association fibres amounts to an isolation of the frontal from the posterior association centres. As we have seen, this finds a clinical expression in slow or rapid mental failure ending in coma or stupor.

It then appears that the degree of intra-cranial pressure is not of so great consideration in the production of stuporose mental states as the method in which this force implicates the brain. Thus a considerable rise of intracranial pressure, so long as it spends itself on the cortex generally, may be unaccompanied by any degree of stupor, but if it expends itself

directly on the long association fibres there is some degree of stupor. As in our case (Case 3), the usual *post-mortem* signs of increased intracranial pressure may be slight or even wanting.

The following case, reported by Nicoll, illustrates our contention so perfectly that we quote it at some length: "Acute symptoms of intracranial pressure—severe and constant frontal headache, giddiness and vomiting, deepening stupor—developed in a man three months after a head injury. The brain was explored in both frontal regions, but the dura presented no bulging, and pulsated normally. No gross lesion was found. Eleven days later the temporo-sphenoidal lobe was explored. The dura was found bulging and pulseless. A considerable quantity of fluid was evacuated from the lateral ventricles. A subsequent negative exploration of the cerebellum and the occipital lobe for the source of the dilatation was made. The patient recovered, and was well a year after." Here we find dilatation of the ventricles giving rise to symptoms of increased intracranial pressure in the absence of the usual evidences of increased intracranial pressure.

Case 4 stands in marked contrast to the above cases: the clinical picture is one of psycho-motor excitement, with hallucinations of sight and hearing; the *post-mortem* facts are an irritative lesion involving the right audito-sensory and audito-psychic areas, and general compression of the grey matter.

As there is a fundamental affinity between hallucinations and mnemonic images, and as the areas which harbour the neuronic systems underlying the mnemonic images of hearing and sight are obviously affected by the neoplasm, it is only natural to connect the hallucinations with the site of the irritative lesion.

The possibility of a gross lesion originating, as a focal symptom, insane hallucinations or illusions has been questioned by Mills: "I do not know of an instance in which a gross lesion, isolated to a physiological centre or area of the cerebral cortex, or extending only to a limited extent beyond such area or centre in one otherwise possessing a normal brain, has caused an insane hallucination"; further, he holds that in cerebral syphilis when a gumma or multiple gummata are found on necropsy the possibility of hallucinations and illusions being focal is more apparent than real; they find their true explanation in diffuse pathological processes or in the toxæmia

which often plays so important a part in the symptomatology of the disease.

But such distinctions are too nice. Although the fact of their analysis and correction is a clinical touchstone for sane hallucinations and illusions, yet the same morbid process is responsible for the production of the sane and insane varieties as far as their elaboration in the areas of which they are judged to be focal symptoms. The insanity of such phenomena depends on their effect on the personality; in many instances we may find a pathological basis for this effect in diffuse morbid changes or a toxæmia, but we cannot disregard the fact that in themselves they constitute a serious form of "mental stress," and, as the researches of Mondio suggest, may produce apathy or psycho-motor excitement.

To sum up: The situation of a tumour relative to the great association schema, which Fleschig has shown exists in the brain, determines to a great extent the incidence of mental symptoms in its clinical course. Any tumour which isolates the frontal from the posterior association centres produces stupor and varying degrees of dementia. An irritative lesion of these association centres, on the other hand, may produce a perversion of their function. In the case of the posterior association centres this results in hallucinations, and in the case of the frontal association centres, in those perversions of the idea of personality described by Welt, Durante, Jastrowitz and others.

Conjugal General Paralysis.

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THE following three cases of conjugal general paralysis are recorded, not that they present any remarkable features singly, but that collectively they afford further evidence of the close relation of this disease to syphilis.

(1) H. McE—, æt. 42, rivetter, married fourteen years. For six months before admission had been drinking heavily. His father was a drunkard. Patient had been out of work on account of bad trade for two years. Mental symptoms were noticed eighteen months before admission. He was listless, childish and apathetic. On admission,

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