THE SO-CALLED GENERAL SYMPTOMS OF INCREASED INTRACRANIAL PRESSURE

Clinico-pathological Report Based on a Study of 100 Cases

FATHY W. TADROS, PH.D., M.R.C.P.(Edin.), D.P.M.

O. H. SEROUR, M.CH.

S. A. ZAKI, M.D., B.Sc.

RASHAD SAKR, M.D., D.CH.

From the Faculty of Medicine, Cairo University

THERE has been much controversy regarding the pathogenesis of the so-called general symptoms of brain tumours. They are often ascribed to one or more factors which produce an increase in the intracranial pressure. It is generally accepted that these factors are: firstly, the increase in the contents of the intracranial cavity produced by the size of the tumour and the surrounding oedema; secondly, the effect on the vascular system, producing rise in the venous pressure; and, thirdly, the effect on the flow of the cerebrospinal fluid.

The present work is devoted to the study of the general hydrocephalic symptoms in 100 consecutive cases of increased intracranial pressure. An attempt at clarification of the controversies with regard to the pathogenesis of these symptoms, based on the pathological changes encountered in these cases, is made.

Material and Methods

The material presented comprises 100 consecutive cases of increased intracranial pressure, 56 with infratentorial and 44 with supratentorial lesions. Of the 56 infratentorial cases, 32 were cerebellar tumours, one subarachnoid cyst between the tentorium and superior surface of the cerebellum, five pineal region tumours, five brain-stem tumours, four cerebellopontine angle tumours, five post-meningitic hydrocephalus, two choroid plexus tumours in the fourth ventricle and two cases of gliosis of the aqueduct of Sylvius. Supratentorial lesions comprised nine parasagittal meningiomas, six thalamic tumours, five temporal, one occipital, four parietal-occipital, five frontal lobe tumours, four extensive tumours in one hemisphere, two lateral ventricle tumours, two suprasellar tumours, one case of corpus callosum tumour, four cases of secondary metastatic nodules irregularly dispersed in the cerebral hemispheres and one case of chronic subdural haematoma.

Fifty-one cases were gliomas, of which 29 were

in the cerebellum, brain stem and around the aqueduct and 22 in the cerebrum; 12 cases were meningiomas, nine parasagittal, one in the lateral sphenoid ridge, one suprasellar and one infratentorial in the pineal region. The rest comprised 10 tuberculomas, eight cerebellar and two hemispherical; four pinealomas; two neurinomas; two choroid carcinomas; one cranio-pharyngioma; one cholesteatoma; four haemangioblastoma cerebelli and two chronic abscesses, one cerebellar and one temporal.

In each case serial coronal sections of the brain were made and the extent of the lesion verified. The ventricles were examined with particular attention to their size and position. Paraffin sections stained with H. and E. were prepared from the lesion, parts of the affected and contralateral hemispheres, including the basal ganglia and diencephalon, and the brain stem. The optic nerves and chiasma from six cases showing papilloedema and six cases of post-papilloedemic optic atrophy were examined histologically, using H. and E. and Smith Queigley stains.

The clinico-pathological data relevant to our issue and discussions of the pathogenesis of the general symptoms of increased intracranial pressure will be presented in two separate parts, the first dealing with the factors responsible for the production of hydrocephalus in space-occupying lesions, while the second deals with the pathogenesis of the individual manifestations of the so-called general symptoms of increased intracranial pressure.

Factors Responsible for Production of Hydrocephalus in Space-occupying Lesions

Pathology

At autopsy all the cases showed the presence of tonsillar herniation, indicating a downward shift of the whole brain. Tumours in the supratentorial space showed, in addition, displacement of the cerebral hemispheres to the contralateral side and

uncal herniation that was more marked on the side of the lesion.

All cases, whether the tumour was supra- or infratentorial, central or lateral, were associated with internal hydrocephalus which was either communicating or partially so. Tumours arising in or near ventricles, as those of the third ventricle and suprasellar, pineal, cerebellar and cerebello-pontine angle tumours, produced obstruction to the flow of the CSF. In these cases the ventricular system above the level of the obstruction was dilated and central in position. Supratentorial tumours away from the cavity of the third ventricle showed dilatation of the third and of the two lateral ventricles only. The ventricular system was shifted to the opposite side of the lesion. There was a kink at the upper end of the cerebral aqueduct near its opening in the third ventricle (Figs. 1 and 2). This part of the aqueduct was flattened from side to side with obliteration of the flow of CSF. This finding was noted irrespective of the site of the tumour in the hemisphere, whether frontal, temporal or occipital, peripheral or central (Figs. 3, 4, 5 and 6).

Naked-eye examination of the brain stem showed the presence of multiple hæmorrhage irregularly dispersed in 23 cases (Figs. 7, 8 and 9). In two of these cases there was hæmorrhages in the diencephalon as well, on the same side of the lesion (Fig. 6). Histological examination revealed the presence of ischæmic infarcts in 61 cases. The infarcts were of recent origin. Some showed early necrosis, others presented complete breakdown of tissue, while others still showed the presence of compound granular corpuscles (Fig. 10).

Hæmorrhages only were present in 12 cases, infarcts in 50 and both together in 11. Thus vascular changes in the brain stem were present in 73 cases. Out of these 73 cases, 32 were supratentorial, i.e. 72.7% of supratentorial cases and 44 infratentorial, i.e. 73.2% of infratentorial cases. It is evident from these percentages that vascular changes in the brain stem occur with equal frequency in supra- and infratentorial cases.

Œdema in the tumour and surrounding brain tissue was present in 26 cases: nine supratentorial, i.e. 20.5% of supratentorial cases, and 16 infratentorial, i.e. 28.5% of infratentorial cases. Cerebral œdema associated with brain tumours was therefore absent in a large number of cases in our series. When present it was seen in the tumour as well as in the rest of the affected hemisphere. It is noteworthy that œdema was present only in cases that were surgically treated. The cases that were not interfered with surgically or subjected to lumbar puncture or ventriculography did not show œdema.

In 23 cases lumbar puncture was performed and CSF pressure and chemistry were recorded. The pressure was increased in 10 out of these 23 cases (41.7%). At autopsy it was confirmed that those cases with normal pressure of CSF had complete obstruction to its flow with a non-communicating hydrocephalus. On the other hand, the lesion in the cases showing rise of pressure was located in such situations as to cause only partial obstruction to the flow of CSF producing communicating hydrocephalus (Table 1). Somewhat conflicting results are reported from examination of spinal fluid pressure, e.g. Ayer (1929) and Puusepp (1928) reported normal fluids in only a few cases of their series, while Greenfield and Carmichael (1925) and Mandelboim (1930) found normal or even sub-normal pressures in the majority of their cases.

Discussion

Considering the pathological findings in the 100 cases of brain tumours presented in this report, we are of the following opinion: firstly, obstruction to the flow of the CSF is the main factor in the pathogenesis of increased intracranial pressure. This is produced either by the presence of the tumour along the route of flow of the CSF, as in the majority of infratentorial and intraventricular tumours, or by displacement of the brain, as in supratentorial tumours. If we recall that the cranial cavity is divided by the tough falx cerebri and tentorium cerebelli into three compartments, two supratentorial and one infratentorial, the contents of either of the supratentorial compartments are increased when a space-occupying lesion reaches a certain size. Faced with the bony calvarium and tough dural membranes, the swollen brain can only escape laterally under the falx to the contralateral side and downwards through the tentorial opening. The lateral and downward shift of the brain would kink the junction of the third ventricle with the aqueduct. Obstruction at this site with consequent internal hydrocephalus results. It is worthy of mention that hydrocephalus arising by the mechanism just described depends on the size rather than the site of tumour in the various lobes, or its being deeply seated or superficial in position. The attempt at correlation between the site of the tumour and time of appearance of hydrocephalic manifestations is thus rendered futile. The only factor determining the early or late appearance of hydrocephalic symptoms in relation to focal manifestations, in our opinion, is the presence of the tumour in a dominant lobe or a silent area.

Secondly, we believe that the role of the increase in contents of the intracranial cavity caused by the size of the tumour and the surrounding œdema in the production of increased intracranial

TABLE I

Name	Sex	Diagnosis	CSF Pressure	CSF Protein
S.S.	F	Pineal-region meningioma	N	50
M.M.	M	4th vent. choroid carcinoma	N	100
A.Az.	M	Pinealoma	N	80
W.M.	F	Gliosis of Sylvian aqueduct	N	80
Kh.I.	F	4th vent. choroid carcinoma	N	70
H.S.	M	Post. fossa meningioma	N	65
A.Sh.	M	Cerebellar hæmangioblastoma	N	70
M.D.	M	Pineal-region glioma	N	75
Y.M.	M	Cerebellar medulloblastoma	N	40
M.Sh.	M	Rt. cerebellar tuberculoma	N	40
A.T.	M	Upper cerebellar glioma	N	40
H.S.	F	Cerebellar glioma	N	20
I.Ch.	F	Cerebellar medulloblastoma	N	90
T.F.	M	Lt. frontal glioma	Raised	120
S.A.	F	Meningioma, parasag.	"	90
A.Sh.	M	Cerebello-pontine angle neuroma	"	100
An.B.	M	Lt. cerebellar tuberculoma	"	150
A.A.	M	Occipital glioma	"	40
R.M.	F	Parasag. meningioma	"	40
F.A.	F	Parieto-occip. glioma	"	20
M.H.	M	Corp. callosum glioma	"	120
A.Az.	F	Rt. cerebell. abscess	"	100
H.R.	M	Rt. lower cerebellar glioma	"	60

FIG. 1.—Glioma in basal ganglia region. Ventricular system is dilated and shifted.
Aqueduct in upper midbrain is compressed and markedly displaced laterally.

FIG. 2.—Section of hemisphere showing ventricular ependymoma, dilatation and shifting of ventricular system. Aqueduct appears in upper midbrain compressed laterally and shifted.

were compressed and actually flattened without corresponding appreciable signs of neural deficiency. As to œdema surrounding the brain tumour (Reichardt, 1905; Cassirer and Lewy, 1920; Fortig, 1921), it was noticed that it occurred in 26 cases only and that these cases were interfered with surgically or by puncture.

Thirdly, venous obstruction or rise of venous pressure as a cause of decreased absorption or increased formation of CSF is doubtful. The absorption of CSF in the superior sagittal sinus is a passive process and depends only on the gradient of pressure between it and the sub-arachnoid space. The sinus is well protected in

pressure is over-emphasized. It must be noted that the growth of the tumour is associated with simultaneous destruction of brain tissue. It is also known that the brain is a collapsible organ. This fact is well demonstrated in seven cases of huge meningiomas encountered among the present series. In these cases the cerebral hemispheres

FIG. 3.—Occipital lobe glioma. Ventricular system above aqueduct is dilated and shifted to opposite side.

FIG. 4.—Temporal lobe compression by middle fossa meningioma. Ventricular system dilated and shifted.

FIG. 5.—Deep frontal glioma. Ventricular system dilated and shifted to opposite side.

FIG. 6.—Section from case of parieto-occipital glioma showing dilatation and shifting of ventricular system. Hæmorrhages in basal ganglia region on same side of tumour.

FIG. 7.—A frontal meningioma. Both lateral ventricle and third ventricle dilated. Section of pons shows hæmorrhages.

FIG. 8.—Section of cerebral hemisphere showing glioma in left thalamus. Brain stem shows hæmorrhages in the midbrain.

FIG. 9.—Chronic abscess in right temporal lobe. Ventricular system above aqueduct is dilated and shifted to left side. Section of midbrain shows secondary hæmorrhages.

FIG. 10.—Photomicrograph of brain stem in a case of frontal glioma showing ischemic infarct with compound granular corpuscles. ($\times 180$)

the tough membranous dura and the pressure of the intracranial contents is not directly transmitted to the sinus, which is neither compressible

nor collapsible. This fact, coupled with our findings of normal CSF pressure in a large number of cases which showed high degrees of hydrocephalus, both pathologically and symptomatically, renders the decreased absorption of CSF as a cause of hydrocephalus improbable. As to the increased formation of CSF, it is known that it is an active process of secretion by the choroid plexus which depends on the arterial blood flow in that plexus.

We believe that the level of pressure in the subarachnoid space depends on the site of the tumour. Those occurring in or near the ventricles producing complete obstruction of the CSF with non-communicating hydrocephalus are accompanied by normal or low subarachnoid pressure. On the other hand, those causing partial obstruction show the reverse.

Pathogenesis of the so-called General Symptoms of Increased Intracranial Pressure

The incidence of symptoms of increased intracranial pressure in the 100 cases presented in this work is shown in Table 2.

Headache

Headache was the commonest symptom in all our cases. In 95% it was the first symptom and was preceded by vomiting in only 5%. These five cases were all children suffering from sub-

tentorial tumours. It was noted that headache disappeared when hydrocephalus was advanced. In fact, when hydrocephalus reached the stage in which it was accompanied by post-papilloedemic optic atrophy headache was never complained of.

It appears, as is generally accepted, that headache is the result of stretch of the arachnoid covering with its blood vessels by the expanding brain (Penfield, Evans, Brickner and German, 1934). The cessation of the complaint of headache is usually ascribed either to the clouding of consciousness accompanying the hydrocephalus in its late stages or to the fact that the arachnoid has reached its maximum limit of stretch.

Vomiting

Vomiting was encountered in 57 cases: 47 subtentorial (84% of subtentorial lesions) and 10 supratentorial (22.7% of supratentorial lesions).

TABLE 2 INCIDENCE OF MAJOR SYMPTOMS OF INCREASED INTRACRANIAL PRESSURE AMONG 100 CASES OF BRAIN TUMOURS						
Headache	Vomiting	Papilloedema	P.P. Optic Atrophy	Mental Symptoms	False Localizing Signs	Fits
100	57	86	53	72	39	8

It was more common in children (39 cases, 68.4%) than in adults (18 cases, 31.6%). It was noticed that most brain tumours in children were located in the cerebellum, fourth ventricle and pineal region and that vomiting in adults was associated more commonly with cerebellar tumours. These facts conform with the observations of Puusepp (1928). In four out of the five cases of brain-stem tumours in our series vomiting was absent. In the fifth it occurred late when other symptoms of hydrocephalus developed in the terminal stage. This was also noted by Wilson (1931).

Vomiting was an early symptom in the evolution of hydrocephalus. It often accompanied headache, especially when the latter was at its peak. In young children (five cases) it appeared even earlier than headache, i.e. it was noted before the child was able to complain of the latter. Vomiting like headache showed amelioration with the advance of hydrocephalus.

On account of the close relationship of vomiting to headache we believe that vomiting is a reflex mechanism in response to the pain afferents which produce headache. Its frequent occurrence with cerebellar and fourth ventricle tumours is due to the early and severe headache accompanying these tumours rather than to their intimate and close relation to the vomiting centre in the medulla. This view is supported by the following facts: firstly, in tumours of the brain stem the lesion is nearer to the vomiting centre than in cerebellar and fourth ventricle tumours, yet vomiting was rarely noticed in such cases. Secondly, medullary centres other than the vomiting centre are the last to be affected in cases of increased intracranial pressure. Had vomiting been due to the direct effect of the neoplasm on the cerebellum or fourth ventricle, one could hardly imagine how the vomiting centre alone could be affected while the rest remain intact and, in particular, the vasomotor centre, which is closely related to the vomiting centre. One would argue that vomiting might be due to ischaemic irritation of the vomiting centre produced by the stretch on brain-stem vessels consequent to shift of the brain. But ischaemia affects all the medullary centres equally and in absence of a proof of a selective sensitivity of vomiting centre this explanation seems improbable.

Papilloedema

Papilloedema was present in 86 cases. This agrees with its incidence in Paton's (1909) and Van Wengen's (1929) series.

In the 14 cases in which fundus changes were absent were four brain-stem tumours, four parasagittal meningiomas, four secondary metastases, one cerebello-pontine angle tumour and one

chronic subdural haematoma. It is noted that nearly half of the meningiomas in our series (four cases out of nine) showed no fundus changes. This conforms with the findings of Bollack and Hartmann (1928), who described the presence of papilloedema in 22 out of 59 meningiomas. It is also observed that papilloedema was absent in cases with minimal increase in the intracranial pressure as well as in those with very slowly growing tumours.

Out of the 86 cases showing papilloedema, 55 were supratentorial (79% of the supratentorial cases) and 51 infratentorial (91% of the infratentorial lesions). From this it is evident that fundus changes are somewhat commoner in infratentorial lesions. It was interesting to observe that papilloedema was advanced (4 D) in the majority of supratentorial compared with the subtentorial tumours (27 out of 35 in the first and only 16 out of 51 in the second). Far-reaching in its significance is the finding that 14 cases that presented complete obstruction to the flow of CSF with non-communicating hydrocephalus and normal or subnormal pressure of CSF in the subarachnoid space showed very minimal papilloedema (2 D). In some of these cases, particularly those that proceeded rapidly to optic atrophy, it was really difficult to judge if the atrophy was post-papilloedemic.

Summing up, we can safely deduce that the incidence and degree of papilloedema depends on the amount of CSF in the basal subarachnoid space. This is supported by the fact that in cases of idiopathic (benign) intracranial hypertension which are associated with a fully communicating hydrocephalus papilloedema was often very marked and chronic. Although fundus changes are commoner in subtentorial tumours, yet the changes are severe and more advanced in the supratentorial lesions.

In discussing the pathogenesis of papilloedema in cases of brain tumours we have to differentiate between the mechanisms underlying each of its two main components: swelling of the optic disc and congested retinal veins. We believe that swelling of the optic disc is due to stagnation of CSF in the base of the brain and hence in the sheaths of the optic nerves. This is due to impairment of the movement of CSF over the hemisphere to reach the superior sagittal sinus. Normally the circulation of the CSF is maintained by the propulsion and suction effect of the brain pulsations on the subarachnoid space (Dott, 1954). In case of increased intracranial pressure such pulsations are diminished or absent. The view of Paton and Holmes (1911) that the increased subarachnoid CSF pressure is transmitted to the

FIG. 11.—Section of optic nerve in a case of advanced papilloedema. No interstitial oedema is detected. The subarachnoid space is widened. (× 82)

optic sheath with consequent pressure on optic veins and development of papilloedema cannot explain the many cases of brain tumour showing papilloedema in which normal or even subnormal pressure of CSF was present. Reichardt's (1905) and Spatz's (1930) suggestions that interstitial oedema in optic nerves, tract and chiasma is responsible for papilloedema were not substantiated in our cases. We studied the optic nerves, tracts and chiasma in 12 cases manifesting papilloedema or post-papilloedemic optic atrophy microscopically for the presence of interstitial oedema. In none of these cases was such a finding encountered (Fig. 11). As to the pathogenesis of congested retinal veins, we agree with the current opinion that the rise of venous pressure associated with brain tumours is the responsible factor.

The pathogenesis of optic atrophy following papilloedema requires careful consideration. It was a complication in 43 out of 51 subtentorial tumours manifesting papilloedema (84%), while it was only noted in 10 out of 35 supratentorial cases (28.6%).

It was more common and earlier in cerebellar and fourth ventricle tumours than those of the brain stem and supratentorial lesions.

At autopsy it was noted that post-papilloedemic optic atrophy was directly related to the degree of dilatation of the third ventricle. It was observed that in cases of hydrocephalus secondary to brain tumours the third ventricle is enlarged at the anterior part of its floor and to a lesser extent in its transverse diameter. Other dimensions were hardly affected. Table 3A and B, compares the dimensions of the third ventricle in 13 tumour cases and in 12 normal cases.

The anterior part of the floor of the third ventricle is the most delicate part. It constitutes both the optic and the infundibular recesses. The dilatation of the optic recess occurs at the expense of compression, flattening and, later, atrophy of the optic chiasma and roots of the optic nerves. In some cases these were reduced to flattened thin filaments stretched over the dilated floor of the third ventricle, which appeared as a cyst. In confirmation of this hypothesis: firstly, three cases (two subtentorial and one supratentorial tumours) had their fields of vision examined before the onset of atrophy and complete blindness. In these cases various field defects were shown indicating compression of various fibres of the optic chiasm and nerves with patchy limitation of conduction before final retrograde atrophy had set in. Secondly, in hemispherical tumours optic atrophy

TABLE 3A
MEASUREMENTS OF THIRD VENTRICLE IN 13 CASES OF SPACE OCCUPYING LESIONS

No.	Age Year	Pathology	Duration	A-P Diam.	Vertical Diameter				Maxim. Width
					Optic Recess Level	Infund. Recess Level	Mamill. Body Level	Posterior Level	
1	14	Cereb. tub.	1 yr.	32	38	36	17	8	14
2	17½	Ependym. 4th vent. . . .	1 m.	35	25	28	16	8	10
3	25	Hæmangio. cereb. . . .	1 yr.	32	30	40	18	9	8
4	19	Subten. arach. cyst. . . .	3 yr.	35	30	40	16	7	15
5	15	Glioma cereb.	7 m.	32	32	38	14	9	9
6	17	Cereb. tub.	9 m.	35	32	35	20	9	7
7	17	Medull. cerebel	4 m.	35	35	37	18	8	10
8	12	Medull. cerebel	2½ m.	33	28	28	15	7	8
9	17	Ependym. 4th vent. . . .	3 m.	35	35	40	18	8	9
10	18	Astrocyt. Lt. front-parietal.	6 m.	35	28	32	20	9	5
11	55	Diff. gliom. Rt. hemisph.	3 m.	35	30	37	22	8	5
12	39	Glioma Lt. frontal	4 m.	35	35	37	18	9	7
13	35	Deep frontal glioma . . .	3 m.	35	35	40	20	8	4
Mean values				34.5	31.8	36.2	17.9	8.2	8.6

TABLE 3B
MEASUREMENTS OF THIRD VENTRICLE IN NORMAL BRAINS

No. of Cases	A-P Diameter	Vertical Diameter				Width mm.
		Optic Recess Level	Infund. Recess Level	Mamill. Body Level	Posterior Level	
1	30	19	24	15	7	2
2	30	20	23	16	8	3
3	30	18	23	17	8	2
4	30	20	24	17	7	4
5	30	20	25	17	8	3
6	27	22	24	16	8	2
7	29	25	30	19	7	4
8	31	19	23	16	7	2
9	30	18	23	15	7	3
10	30	17	24	16	7	2
11	30	18	23	17	8	4
12	32	20	26	18	8	2
Mean Values	29.9	19.7	24	16.6	7.5	2.75

occurred late in 10 cases and not at all until death in 25 cases. In these cases the third ventricle was not as markedly dilated as in cases of subtentorial tumour. Thirdly, in cases of otitic hydrocephalus and idiopathic hypertensive hydrocephalus where the hydrocephalus is communicating and the ventricles are not dilated to any considerable extent the papillœdema is more marked, and however chronic it may become even for a year or more it rarely, if ever, leads to post-papillœdemic optic atrophy.

The mechanism of optic atrophy in tumours directly related to the optic nerves and chiasma is due to direct pressure. This was seen in suprasellar tumours.

Mental Symptoms and Disturbances of Consciousness

We prefer to discuss both these symptoms under one heading because we believe that mental symptoms of raised intracranial pressure have a special general character which is related to a certain degree of disturbance of consciousness. This character is a form of an organic reaction type, in which the patient shows slow reaction, inattention, indifference, poor response, poor memory and sometimes faulty insight into the gravity of his condition. In the intellectual sphere the finer qualities of mind are not clouded. On the affective side more often than not the patients are apathetic, show lowering of spirits and loss of instinctive reaction much more than the labile unstable emotions. With the progress of the case usually lethargy, drowsiness and stupor supervene and later complete loss of consciousness when the patient leads a vegetative existence.

Most of our cases (70 out of 100) showed this progressive deterioration. They included frontal, temporal, diencephalic and ventricular as well as

subtentorial tumours. One of the series was a case of glioma of the corpus callosum. His mental picture was that of lack of insight into his physical ailment and an appreciable degree of disorientation. He had heaviness and ataxia of both lower limbs and did not mention the headaches unless asked about them. However, he did not show any specific type of mental picture.

Specific types of mental picture have been described with brain tumours. These are often so to speak, positive symptoms unlike the negative general character often met with. They were noted by Oppenheim (1889) and Wilson (1931). Wilson, however, did not think of these syndromes as specific to brain tumours, as they were met with in cases of encephalitis (Wilson, 1918).

Sometimes, however, the person concerned is predisposed and the specific psychiatric symptoms are coincidental.

None of our series showed any specific positive mental picture. The analysis of these symptoms shows that they all represent some degree of disturbance consciousness, e.g. loss of memory is unconsciousness of the past, disorientation is poor consciousness of the surroundings, while apathy and poor response are a manifestation of lack of attention, i.e. unconsciousness of the present.

Though the mental symptoms accompanying brain tumours at various sites are of the same reaction type, yet they may differ in degree and in the extent to which they dominate the clinical picture. Thus they might still be of localizing value.

It is thought that pathologically these mental symptoms were associated with tumours which interfere with large association fibre systems, viz. corpus callosum, occipito-temporal fasciculus and occipito-frontal fasciculus (Wilson, 1931; Burns,

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1908; Pfeifer, 1928). This conception, adequate enough in explaining the occurrence of mental symptoms in tumours of the corpus callosum and frontal lobe, does not explain the association of mental symptoms with tumours of thalamus, suprasellar region, intraventricular tumours and subtentorial tumours.

The modern conception of the centrencephalic system as the centre of consciousness agrees with pathological observations made in our series. This system consists of the central grey matter of the thalamic, hypothalamus and brain stem. It represents the different levels of consciousness from above downwards.

Pathological specimens examined showed that the lateral and downward shift of the brain produced vascular ischæmia or hæmorrhage in this system due to stretch and tear of the perforating vessels supplying it. This, in our view, is the main cause of the general mental and conscious disturbances which accompany most tumours. It might also contribute to the production of mental symptoms in tumours of frontal lobe or corpus callosum.

False Localizing Signs

TABLE 4

No. of Cases Showing False Localizing Signs	Cerebellar	Decerebrate Rigidity	Cranial Nerves
39	3 (1 parietoccip., 1 frontal, 1 intraventricular (lateral ventricle))	30 (supratentorial)	6 (4 cerebellar, 2 pineal)

The so-called false localizing signs of brain tumours comprise those of long tract or cranial nerve manifestations which are not directly infiltrated or destroyed by the tumour. One of our cases which has a parieto-occipital tumour showed cerebellar signs. Another case of frontal glioma and one of lateral ventricle ependymoma which was infiltrating the cerebral hemisphere manifested intention tremors. Most of our supratentorial tumours manifested decerebrate rigidity in their terminal course. Four cerebellar cases showed symptoms of affection of multiple cranial nerves.

The brain stem which contains all ascending and descending as well as cerebellar tracts is the seat of ischæmia as a result of downward displacement. This, in our opinion, is the main cause of such symptoms. Stretching of the cranial nerves as a result of downward shifting explains their false localizing involvement.

Fits

TABLE 5

No. of Cases Showing Fits	Generalized	Jacksonian
8	4 (1 diffuse frontal, 2 meningioma, 1 subtentorial cyst)	4 (3 meningioma, 1 parietal glioma)

Generalized convulsions occurred throughout the course of one case of deep diffuse frontal glioma. In five cases of meningioma three had Jacksonian fits and two generalized convulsions. A case of parietal glioma presented with Jacksonian fits followed by generalized convulsions only in the early stages of its course; one case of subtentorial subarachnoid cyst which was invaginating the upper surface of the cerebellum and the mid-brain posteriorly had three major fits in its course. It is seen that convulsions occurred in 8% of the cases. Incidences given in the literature are as follows: Parker (1930), 12% of 313 cases; Dana (1916), 25%; and Kroll (1929), 20%. Parker's cases occurred in 83.6% of supratentorial tumours and in only 16.4% of infratentorial tumours.

Jacksonian fits are of highly localizing value (Collier, 1904; Muskens, 1928; Beduschi, 1928), while generalized convulsions are not.

It is generally accepted on both physiological and electro-encephalographic evidence that convulsions are a manifestation of discharge from the diencephalon and the brain-stem reticular formation. Even Jacksonian convulsions are proved to be due to a localized diencephalic discharge in response to afferents from a cortical area. The stimulus here would not be of sufficient strength to evoke the whole diencephalon.

Being of such an origin, convulsions of the non-Jacksonian type would be considered of non-specific localizing value. Hence we find cortical lesions producing generalized convulsions similar to those of central or even subtentorial localization.

Summary

(1) The general symptoms of increased intracranial pressure in 100 cases are described and their pathogenesis discussed.

(2) The report is divided into two parts. The first deals with the factors responsible for production of hydrocephalus in space-occupying lesions. We are of the opinion that obstruction to the flow of the CSF is the main factor in the pathogenesis of increased intracranial pressure.

(3) The second part deals with the pathogenesis and frequency of each individual general symptom

of increased intracranial pressure. The analysis of the pathological findings in our cases has suggested that:

- (a) Headache is due to stretch of the arachnoid covering with its blood vessels by the expanding brain.
- (b) Vomiting is a reflex mechanism in response to the pain afferents which produce headache.
- (c) Papillædema is the result of stagnation of CSF in the basal subarachnoid space and sheaths of optic nerves.
- (d) Post-papillædemic optic atrophy is caused by

dilatation of the anterior part of the floor of third ventricle with consequent compression of optic chiasma and tracts.

- (e) Mental changes are in most cases non-specific and non-localizing and are due to impairment of the centre of consciousness in the centroencephalic system.
- (f) False localizing signs are mainly due to downward displacement of brain and stretching of blood vessels with consequent ischæmia of brain stem.
- (g) A unitary conception of the generalized and Jacksonian fits is discussed.

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