I N T R A C R A N I A L T U M O U R.

AN ANALYSIS OF THIRTY-EIGHT CASES IN CHILDREN.

being

A Thesis for the Degree of M.D.
of the University of Edinburgh,

by

GEORGE CRUIKSHANK BURGESS,

M.B., Ch.B.
Forfar.

August, 1913.

CONTENTS.

INTRODUCTION.

In this Thesis I intend to give the Analysis of Thirty-eight Cases of Intracranial Tumour in Children which came under my observation, either directly or indirectly, during four years at the Infirmary for Children, Liverpool.

I would emphasise the fact that I have no intention of writing a treatise on the subject, but simply of giving the cases as I found them. In some matters, indeed, the statistics and my observations differ from those recorded in the standard text-books.

The cases, a list and the general particulars of which are in the frontispiece, were all taken without selection as they came to the Infirmary. The fact that I was able to obtain post-mortem examinations of thirty-six of these adds to the value and interest of the analysis as such.

DEFINITION.

Under the term intracranial tumour it is convenient to include not only tumours, in the strict sense, but also all forms of cysts, etc., contained in the cavity of the cranium, since their clinical phenomena correspond closely with those of tumours.

NATURE.

The cases (see frontispiece) I have collected were of the following nature:

Table No.1.

Intracranial Tumours in Children.

Tubercular	28 cases
Sarcoma	4 cases
Glioma	4 cases
Gumma	2 cases
Total	38 cases.

Thus by far the most common form of Intracranial Tumour in Children is tubercular in nature (73.4%): next comes the sarcomata (10.5%) and gliomata (10.5%):

the gummata were only 5.2%. These percentages are rather higher for the tubercular tumours and rather lower for the sarcomata and gliomata than in Starr's collection of 269 cases.

I had no cases of cysts or carcinomata of the brain, so I have necessarily come to the conclusion that these conditions are rarely met with in children.

FREQUENCY.

The following table gives a comparison with the other diseases commonly met with in childhood.

Table No.2.

Comparative Table of Diseases of Children.

Total Admission = 2172.

Diseases of Nervous System	276.
Intracranial Tumours	38.
Tumours of Other Parts	24.

It will be seen that this condition represents a very small percentage (1.8) of the whole, and that, contrasted with other organic diseases of the central nervous system, intracranial tumours are relatively

rare, but that the brain is one of the most common sites of tumour formation in the body.

AGE.

The common ages for tubercular tumour of the brain in children is, I find, from 3 to 5 years. The sarcomata and gliomata appear at rather later ages. In my series of cases the ages were as follows:-

Table No.3.

Tubercular:	Under 2 years of age	.....	1 case.
	2-3 " " "	.....	1 case.
	3-4 " " "	.....	6 cases.
	4-5 " " "	.....	5 cases.
	5-6 " " "	.....	3 cases.
	6-7 " " "	.....	3 cases.
	7-8 " " "	.....	3 cases.
	8-9 " " "	.....	1 case.
	9-10 " " "	.....	2 cases.
	10-11 " " "	.....	1 case.
	11-12 " " "	.....	2 cases.
	Total	.....	28 cases.
Sarcoma:	5 years old	.....	1 case.
	6 " "	.....	1 case.
	11 " "	.....	1 case.
	12 " "	.....	1 case.
	Total	.....	4 cases.

Glioma:	5 years old	.....	1 case.
	6 " "	.....	1 case.
	8 " "	.....	1 case.
	10 " "	.....	1 case.
	Total	.....	4 cases.

Syphilis:	8 years old	.....	1 case.
	10 " "	.....	1 case.
	Total	.....	2 cases.

SEX.

Tumours of the brain, in my experience, effect boys more commonly than girls. The reason of this I do not know. In the adult, of course, the male sex are more liable to head injuries, syphilis, alcoholic excess, and mental overstrain: but this is not the case in childhood, although all five of my cases with a history of injury were boys. (See Table No.10). In my records there are 21 boys and 17 girls, as follows:-

Table No.4.

	Boys.	Girls.	Total.
Tubercle	16	12	28
Sarcoma	2	2	4
Glioma	2	2	4
Syphilis	1	1	2
Totals	21	17	38

Hilton-Fagge (System of Medicine, p.768) gives the proportion of 27 to 15, and Oberneier as 10 to 6, males to females at all ages.

FAMILY HISTORY.

(1). Of the twenty-eight cases of tubercular tumours, there were -

Table No.5.

A. Family History of Tuberculosis - 7 cases.

Case No.3	Father, Mother and Brother died from Pulmonary Tuberculosis.
Case No.7	Father died from Pulmonary Tuberculosis.
Case No.8	Brother died of Tubercular Meningitis.
Case No.9	Mother was Tubercular.
Case No.13 ...	Mother strongly Tuberculous.
Case No.23 ...	Father died of "chest trouble," age 34.
Case No.27 ...	Sister had Pulmonary Tuberculosis.

B. No Family History of Tuberculosis - 15 cases.

C. No mention of tubercular F.H. ... - 6 cases.

Thus in only seven cases out of twenty-eight (or 25%) was there a definite history of tuberculosis.

(2). The cases of sarcomatous and gliomatous tumours presented no family history of any import.

(3). In the two clinical cases of gummata of the brain, no history could be obtained either of syphilis or of tuberculosis.

PREVIOUS ILLNESSES.

Table No.6.

Illness.	Nature of Tumours.	No. of Cases.
Measles	Tubercular	5
Scarlet Fever	Tubercular	1
Purpura Haem:	Tubercular	1
Tuber: Ostitis	Tubercular	1
Tuber: Arthritis ..	Tubercular	2
	Total	10

As can be observed by this table, a definite history of previous illness was obtained in ten out of twenty-eight cases of tubercular tumours; in five of these (i.e. 50%), the previous illness was measles. This bears out the accepted opinion that measles is a frequent precursor of tuberculosis in its various forms. If, however, the next table be

studied it will be seen that a tubercular intracranial tumour is frequently associated with a tubercular lesion elsewhere in the body. This suggests that the cerebral condition was not necessarily the primary tubercular focus in the body, but may have been secondary to a focus elsewhere. The long interval of time occurring between the attack of measles and the onset of the typical symptoms of the tumour, further supported this conclusion.

Table No.7.

Previous Illness	Interval	Other Conditions found
Measles:		
Case No.7	?	Tub: Mesenteric Glands.
Case No.9	1 year	Pulmonary Tuberculosis.
Case No.12	2 years	Tuberculosis of Lungs and Bronchial Glands.
Case No.33	9 months	Pulmonary Tuberculosis.
Scarlet Fever:		
Case No.23	4 years	General Tuberculosis.
Purpura Haemorrhagica:		
Case No.13	2 months	Pulmonary Tuberculosis.
Tubercular Ostitis:		
Case No.29	2 years	Pulmonary Tuberculosis.
Tubercular Arthritis:		
Case No.14	2 years	Pulmonary Tuberculosis.
Case No.30	1 year	Pulmonary Tuberculosis.

In three of the cases the previous illness was actually a tubercular condition, viz: in one case, tubercular ostitis, and in two cases, tubercular arthritis. We may naturally conclude that the tumour formation in the cranial cavity was secondary either directly or indirectly to these foci.

I see no reason to assume that either Scarlet Fever (in Case No.23), or the Purpura (in Case No.13) had any importance as predisposing factors.

PRIMARY AND SECONDARY TUMOURS.

In this connection I have found that the tubercular tumours on the one hand, the sarcomata and gliomata on the other, form a distinct contrast to one another, since (as Table No.8 shows) the tubercular tumours are usually secondary, whilst the sarcomata and gliomata are usually primary.

Table No.8.

	Tubercle.	Sarcoma.	Glioma.
Primary	3	4	4
Secondary	25	0	0
Totals ...	28	4	4

The next Table (No.9) gives an analysis of the probable sites of the primary foci of the tubercular tumours as found at the autopsy.

Table No.9.

Probable Primary Tubercular Foci.

(a)	Lungs only	12 cases.
(b)	Lungs and Bronchial Glands	4 cases.
(c)	Bronchial Glands only	3 cases.
(d)	General Tuberculosis	1 case.
(e)	Tubercular Mesenteric Glands ..	2 cases.
(f)	Tubercular Bones or Joints	3 cases.

It is to be noted that where the condition was a secondary one, the primary focus in 85% of the cases was situated in or about the lungs.

In only three cases was the tubercular condition primary in the brain.

INFLUENCE OF INJURY ON THE CAUSATION.

In many of my cases there was a history of a blow or other injury to the head. Often, however, I believe that the parents, anxious to find a cause, attributed all the trouble to some fall or injury

which as nearly as possible coincided with the first appearance of the symptoms. In other cases the falls seemed to me to be symptomatic of the vertigo, associated with the condition which had already commenced. As seen from the following table (No.10), there were only five cases where the condition seemed definitely to have commenced after an injury to the head.

Table No.10.

Case	Sex	Injury to	Tumour	Position
No.1	M.	Forehead	Tubercle	Cerebellum, &c.
No.6	M.	Nose	Tubercle	Pons
No.11	M.	Forehead	Tubercle	Cerebrum
No.24	M.	Face	Tubercle	Cerebellum
No.32	M.	Occiput	Glioma	Cerebellum

This table presents three points of interest, viz:-

1. The cases were all boys.
2. The situation of the tumour in the brain in at least three of the cases was remote from the site of injury to the head.
3. Four of the cases were tubercular tumours, the remaining, fifth, a glioma.

In the case of the tubercular tumours, the injury probably lowered the vitality of the particular part of the brain, predisposing it to tumour formation. In the case of the glioma, another explanation may be the effects of a haemorrhage into the tumour, the symptoms of which were previously absent or ill-defined.

SIGNS AND SYMPTOMS.

The following are all the signs and symptoms which were observed in my cases.

A. General Signs and Symptoms.

1. Headache.
2. Vertigo.
3. Vomiting.
4. Optic Neuritis.
5. Optic Atrophy with loss of vision.
6. General Epileptic Convulsions.
7. Disturbance of the Mental and Intellectual Functions.
8. Wasting.
9. Fainting Fits.
10. Slow Pulse.

B. Other Signs and Symptoms.

1. Motor Derangements. Paralysis of movement, paresis, spasms, convulsions, tremors, contractures.
2. Sensory Derangements. Loss of sense to touch and pain,

hyperaesthesia. Derangements of the senses of sight and hearing.

3. Reflex Derangements. Superficial reflexes diminished. Deep reflexes diminished or exaggerated. Disorders of organic reflexes (micturition, defaection, respiration, cardiac action, speech, swallowing.).
4. Trophic Derangements. Acute bedsore, atrophied joints and muscles, sloughing of the cornea.
5. Enlargement of the Head.

GROUPING OF SYMPTOMS.

The manner in which these symptoms were grouped together varied considerably in different cases (see Table No.11). In the majority both sets of symptoms were present, the general symptoms preceding the localising. There were five cases, however, in which the general symptoms only were present, and one case (No.18) with localising symptoms only.

Table No.11.

General followed by Localising Symptoms	23 cases.
Localising followed by General Symptoms	9 cases.
General Symptoms only present	5 cases.
Localising Symptoms only present	1 case.

I have found it difficult sometimes to draw a definite line between general and localising symptoms. Thus headache, an important general symptom, may be-

come a localising symptom where the pain is confined to one part (see Table No.13). Severe and persistent vomiting, another important general symptom, seems to me, might indicate the position of the tumour in the pons varolii and medulla oblongata.

MODE OF ONSET.

In thirty-four cases (i.e. 89%) the disease was slow in onset. In the remaining four cases (11%) the onset was acute with convulsions.

Table No.12 shows the first symptom noted in all the series.

Table No.12.

Headache	14 cases.
Vomiting	8 cases.
Convulsions	4 cases.
Paresis	5 cases.
Strabismus	4 cases.
Lethargy	1 case.
Giddiness	1 case.
Loss of Vision	1 case.
Total	38 cases.

Thus the first symptom noted was headache in 37% of the cases, vomiting in 21%, and convulsions in 10.5%.

The onset of the symptoms may be acute, and the condition settle down for a considerable time thereafter. Thus in Case No.2, a girl aged 4½ years had suffered a year previous to admission from fits followed by vomiting. These symptoms lasted for about a week. From that time for about a year she had suffered from constant, but not severe, headache and her parents noticed her head was getting larger. Acute symptoms again arose a week before admission to the Infirmary, a year after the onset, and she died seventeen days later. At the post-mortem examination tumours were found growing in the right frontal lobe and in the cerebellum. These had been growing slowly in the white matter, causing atrophy rather than destruction, of the brain. There was also an extensive dropsical effusion into the ventricles which, by vastly increasing the intracranial pressure, caused the enlargement of the head and, I think, may have been the cause of the general symptoms, at the onset well marked, diminishing in intensity.

ANALYSIS OF INDIVIDUAL SYMPTOMS.

Headache.

This was a common general symptom. In all my 38 Cases it was:-

First and most marked symptom in .... 14 cases.
A secondary symptom in ..... 8 cases.
Unnoted in ..... 16 cases.

It was thus present in at least 58% of the cases, the earliest and most prominent symptom in 37%, and unnoted in 42% of my cases. In all probability it had been present at some stage of most of the latter but had been so slight as to be overlooked.

Its severity was very variable, from slight in early stages to intense in the later stages. In one case (No.36) the pain was very intense, the girl burying her aching head in the pillow and shuddering at any loud sound. The pain was localised to the occipital region (see Table No.13), and this part was tender on percussion.

In Case No.13 (Tumours of the right optic thalmus and the right occipital lobe) the headache underwent unusual exacerbations, the patient, during the intervals, being quite free of pain. The late Dr Hilton Fagge (System of Medicine, p.771, Vol.1, 4th

Edition) quotes a somewhat similar case of Abercrombie's - a boy age 6, who began to suffer from fits of severe headache, recurring at first about once a fortnight, and leaving him in good health in the intervals. After five or six months the attacks became persistent and when 2 months later he died, a tubercular tumour was found in the cerebellum. He quotes a similar case recorded by Lebert.

Situation of pain. In the majority of cases the headache was general. In only four cases was the pain located to any particular part as is seen in the following table.

Table No.13.

Case	Situation of Pain	Position of Tumour.
No.3	Frontal	Right cerebrum, Pons, Cerebellum.
No.7	Occipital	Cerebellum
No.32	Occipital	Cerebellum
No.36	Occipital	Pons

It would seem therefore as if occipital headache was present in tumours situated in the posterior portions of the brain, but I have no evidence to conclude that frontal headache can be relied upon as an

indication of the site of the tumour.

Vertigo.

Vertigo or giddiness was another frequent general symptom. It was present in thirty-two (i.e. 84%) of my cases, the degree of severity varying as shown in the following table.

Table No.14.

1.	Very prominent	3 cases	= 9.4%
2.	Marked	12 cases	= 37.6%
3.	Minor	17 cases	= 53%
	Total	32 cases.	

It was a constant and early symptom where the cerebellum was the seat of the tumour, and it would seem to me that if the giddiness is early and severe, the tumour is likely to be cerebellar in situation.

Vomiting.

Vomiting was another frequent symptom. It was present in thirty (77%) of my cases. Its severity varied as shown in the following table.

Table No.15.

Marked ..... 24 cases = 80%

Slight ..... 6 cases = 20%

Total ..... 30 cases.

The vomiting occurred independent of meals, and often when the stomach was empty, e.g. on waking in the morning. It occurred early and proved an obstinate and a troublesome symptom of the disease in the six cases shown in the following table.

Table No.16.

Case	Tumour	Situations
No.1	Tubercular	Cerebrum, Cerebellum.
No.3	Tubercular	Cerebrum, Pons, Cerebellum.
No.6	Tubercular	Pons Varolii.
No.21	Glioma	Pons Varolii.
No.25	Tubercular	Pons, Cerebrum.
No.36	Glioma	Pons, Medulla Oblongata.

It will be seen that tumours situated in the Pons Varolii or its neighbourhood seem to be associated with early and persistent vomiting. In the fourteen cases where the headache was severe, the vomiting also proved a troublesome symptom.

Optic Neuritis.

Optic neuritis was present in thirty-four (i.e. 89.5%) of my cases. I regard it as the most important, from a diagnostic point of view, of all the general symptoms, and for that reason I have gone very fully into the literature of the subject.

It was the earliest general symptom observed in two (Nos.25 and 31). In one case (No.36) it did not appear until the day before the child died. In ten of the cases, it was more marked on one side than on the other.

The particulars of my four cases, in which there was no optic neuritis, are noted in the following table.

Table No.17.

Case	Tumour	Situation	Duration
No.6	Tubercular	Pons	3 months
No.11	Tubercular	Cerebrum	3½ months
No.18	Glioma	Pons	2 months
No.29	Tubercular	Cerebellum	4 months

In only eight cases was the acuity of vision disturbed in the early stages, and even where the neuritis was extensive (as in Case No.13), there was

no interference with vision observed until a few days before the child died. In this connection Dr Stephen Mackenzie states that he "would go so far as "to say that in the practice of physicians who "examined all their cases with the ophthalmoscope, "whether the case was a cerebral one or not, at least "in one half, if not more, of the cases in which "optic neuritis was discovered it would be found un- "associated with any marked and often without any "apparent appreciable defect of sight." (Transactions of the Ophthalmological Society of the United Kingdom, Vol.1, p.97).

Causation of Double Optic Neuritis associated with Intracranial Tumour. There is great controversy about these causes. There are, at present, three views:-

(1) Von Graofo's Increased Pressure Theory supposed that the condition was due to an obstructed venous return from the eye, from compression of the cavernous sinus by the increased intracranial pressure. This theory had to be abandoned when it was shown that the communication between the superior ophthalmic and anterior facial veins is so free that even complete obstruction of the cavernous sinus does not produce venous stasis and oedema in the optic papilla.

(2) Mechanical Theory is practically a modification of Von Graefe's Theory. This theory was advanced when it was found that the subvaginal space surrounding the optic nerve is continuous with the subarachnoid space in the cranial cavity. Hence the increased intracranial pressure would interfere with the lymph return from this space swelling the sheath and increasing the pressure to such an extent (on the nerve) as to obstruct the venous return from the papilla. No doubt a mechanical stasis in the veins would so be followed by exudation, and then the appearance of inflammation would be produced. An argument in favour of this theory is that optic neuritis may be greatly benefited or will even disappear after trephining and the cerebro-spinal fluid allowed to run off.

On the one hand, in Cases Nos.6 and 29, in which there was marked post-mortem appearance of increased intracranial pressure, there was no neuritis. On the other hand, in Cases Nos.22 and 25, where there had been little increased intracranial pressure, optic neuritis was an early and prominent sign. Again, optic neuritis occurred quite irrespective of the size of the tumour.

In some cases no distension of the vaginal sheath has been observed while neuritis has been

present. Increased intracranial pressure occurs in chronic hydrocephalus and cerebral haemorrhage, but neuritis is rarely seen in these conditions.

Deutschmann (Ophthalmic Review, April 1887) conducted a series of elaborate experiments in animals to discover what degree of hydrops of the optic nerves in animals is required to produce changes in the disc similar to those of the choked disc in man, and to find if there were any conditions under which a moderate and even transient dropsy of the nerve, such as is found post-mortem in man, is associated with the occurrence of optic neuritis.

With antiseptic precautions he injected warm sterilized agar-agar solution into the optic nerve of an animal; he found, on examination, that there was swelling and oedema of the papilla but no neuritis. He next made injections into the cranial cavity so as to fill the optic nerve sheath by increased intracranial pressure; the injections were renewed from time to time, and kept up for several weeks. Dissection showed that the sheath was forcibly distended, but that the papilla, the nerve trunks and sheaths were absolutely free from any trace of inflammation.

(3) <u>The Descending Neuritis Theory</u> holds that the inflammatory process makes its way downwards

from the cranial cavity to the optic nerve and the optic papilla - the inflammation being produced either by a toxic substance from the tumour, or by a direct downward extension of a meningitis.

The irritant or toxic substance, supposed to be produced in or around the tumour, co-mingles with the cerebro-spinal fluid and is conveyed by it into the sub-vaginal space which surrounds the orbital portion of the optic nerve, producing irritation and inflammation of the nerve.

Leber was the first to advance this theory of irritation, but before we can adopt it, the presence of the irritant, and its nature, requires to be demonstrated.

The only conclusion as to the causation of optic neuritis at present possible is that the increased intracranial pressure is essential. Dr Gower (Diseases of the Nervous System, Vol.II., p.770) thinks that the neuritis is due to more than one mechanism which varies in relative degree in different cases. One of these is that extension of a process of tissue irritation to the optic tract and nerves which reaching the papilla lights up a more considerable inflammation. Another is the distension of the sheath of the optic nerve by fluid from the sub-arachnoid space containing, it may be, irritating products. A

third is the meningitis which, as we have seen, often occurs in cases of intracranial tumour, may extend directly to the optic nerves.

Diagnostic Value. - In all cases in children with head symptoms, I think the fundus oculi ought to be examined, without regard to the visual acuity. If optic neuritis is present it almost clinches the diagnosis of intracranial tumour: at the same time if it is absent, it does not necessarily exclude this diagnosis.

Optic neuritis may be present in children in other conditions - meningitis, cerebral abscess, acute otitis and chorea (Ashby and Wright, Diseases of Children, pages 461 and 488). Dr Hughlings Jackson (Transactions of the Ophthalmological Society of the United Kingdom, Vol.I., p.61) states that"optic neuritis can be found in some cases clearly, from the symptoms, cases of intracranial disease - where post-mortem examination discloses no local disease within the cranium or in any other part of the body."

Localising Value of optic neuritis is very small. It was early in onset and intense in character in tumours of the cerebellar and quadrageminal regions, except in Case No.29 where it was absent during the whole course of the disease.

Pathological Value. In the case of syphilitic tumours arrested by treatment, the neuritis will subside and may disappear altogether. This is well shown in the following two cases of my series.

Case No.38. A girl, age 8. The mother informed Dr Peter Davidson (under whose care the child was) that for about 8 weeks she had suffered from constant headache, chiefly in the occipital region, giddiness and vomiting occasionally and irregularly without any relation to food, also weakness in the left arm and leg. On examining her eyes there was no interference with vision either subjectively or objectively, but there was marked optic neuritis. There was no albuminuria. Potassium iodide was administered in large doses with the result that the optic neuritis disappeared, while the headache, vertigo and vomiting also disappeared. There was no family history of syphilis.

Case No.37. A boy, aged 10, was brought to the Infirmary chiefly because of weakness of vision, and staggering. There was no albuminuria. His eyes, examined by Dr Stevenson of the Liverpool Eye Infirmary, showed marked optic neuritis. His vision was almost completely gone. Large doses of potassium iodide and mercury were given. Rapid improvement to complete recovery ensued, and all traces of the

optic neuritis disappeared. There was no family history of syphilis.

Optic Atrophy with Loss of Vision.

Primary optic atrophy was not present in any of my cases. Post-neuritic atrophy was noted in three of my cases. The following tables (Nos.18 and 19) give the general particulars of the condition in those cases.

Table No.18.

Case	O.N.	O.A.	Visual Acuity
No.4	Early	Early	Blindness early
No.7	Early	Late	Diminished from first
No.24	Early	Late	Blind last month only

In Case No.4 the impaired vision was the first symptom noted by the child's parents that anything was wrong.

Table No.19.

Case	Duration of Illness	Tumour	Position of Tumour
No.4	1 month	Sarcoma	Cerebellum
No.7	15 months	Tubercular	Cerebellum
No.24	11 months	Tubercular	Cerebellum

From the latter table it would seem as if the optic atrophy can occur as readily with quick-growing sarcoma as with the slowly-developing tubercular tumours. In all three cases, be it noted, the tumour was situated in the cerebellum, and the optic neuritis was early and intense in character, as I have already observed, in these cases.

General Epileptic Convulsions.

In four of my cases the first symptom of the condition was general convulsions. The following tables give the particulars of these cases.

Table No.20.

Case	Age	Nature	Position
No.2	$4\frac{1}{2}$	Tubercular	Frontal Lobe and Cerebellum
No.10	3	Tubercular	Cerebrum and Pons
No.23	7	Tubercular	Corpus Striatum
No.27	$3\frac{1}{2}$	Tubercular	Corpus Callosum

In addition, convulsions was a prominent feature in seven other cases, making eleven (i.e. 28%) in all.

Disturbance of the Mental and Intellectual Functions.

Mental or intellectual disturbances do not appear, in my experience, to be a marked symptom in children. The only alterations I have observed are a mental apathy or an irritable condition.

The following table (No.21) shows the incidence and, in a general way, the degree of the disturbance.

Table No.21.

	Marked	Slight	Absent	Total
Tubercle	4	8	16	28
Sarcoma	0	1	3	4
Glioma	0	2	2	4
Gumma	0	0	2	2
Totals	4	11	23	38

Wasting.

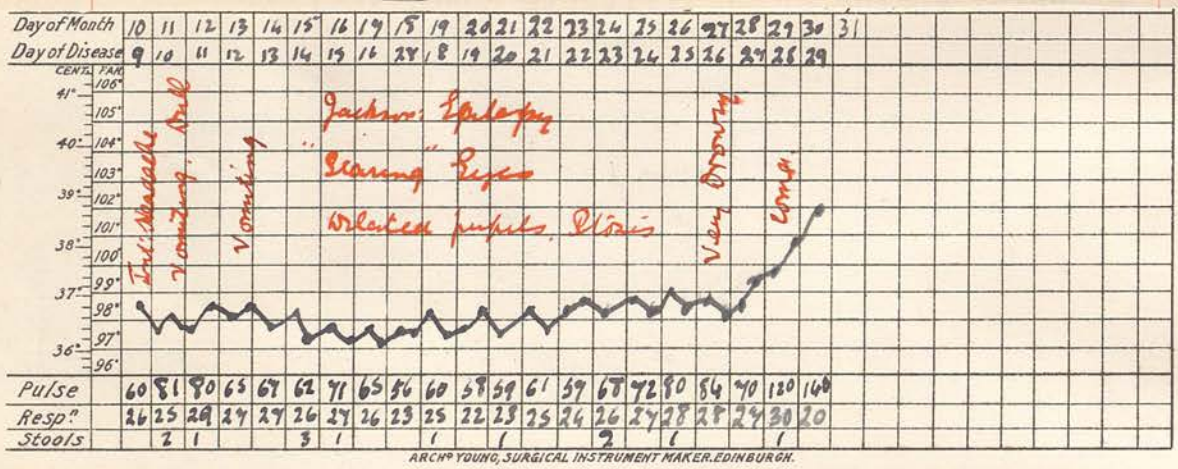
In all my cases where emaciation was noted the tumours were tubercular; of these there was early and complete wasting in five, and in sixteen the wasting was in the final stages of the disease.

Records of Temperature, Pulse and
Respiration in

Case No.5.

Sarcoma of Corpora Quadragemina.

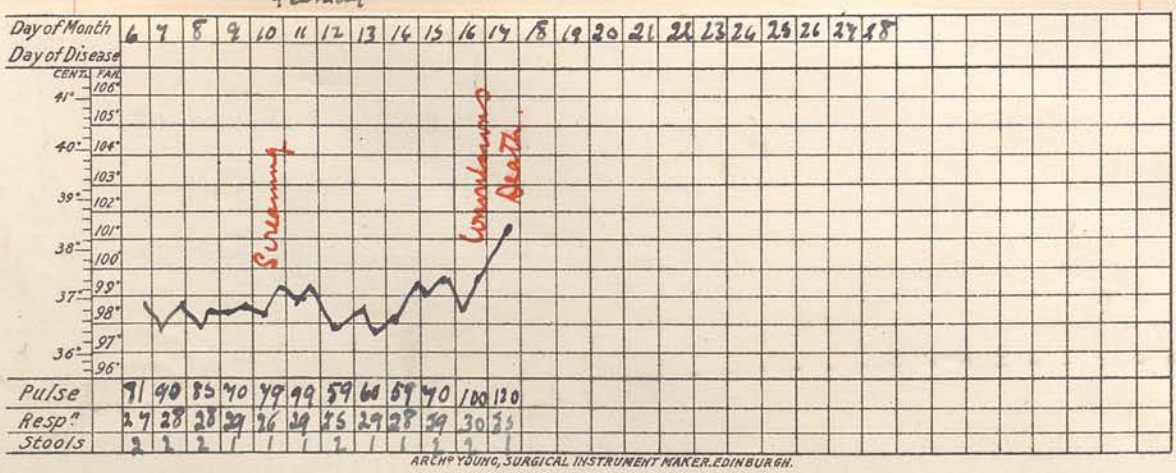
William Zero. / Age. 11. years. / Schoolboy /
October.



Case No.30.

Tubercular Tumour of Right Rolandic Area.

Arthur Jamieson / Age 5 years.
Fireman



Fainting Fits.

In only one case (No.7) was there any note of fainting fits. The boy (age 7) had three, according to the mother's statement, on the day previous to admission.

Pulse.

In my uncomplicated cases of tumour of the brain the pulse was distinctly slow - the lowest being 56, in Case No.5 - and the temperature normal. But where there was any febrile complication, such as meningitis or pulmonary tuberculosis, the pulse and temperature rose accordingly.

I append charts showing the pulse and temperature in two of the uncomplicated cases. It will be observed that in each case the pulse and temperature rose just before death.

Motor Symptoms.

The motor symptoms varied from a paralysis of small muscles, such as the muscles of the eye, to a paralysis of a large part of the body, e.g. a hemiplegia. They presented themselves in various forms such as complete paralysis, paresis, spasms, localised convulsions, tremor and tetanus. Contracture was observed in two cases.

Sensory Symptoms.

The sensory symptoms are not so well noted probably from the difficulty of ascertaining them in children. But there was observed loss of sense to touch and pain, hyperaesthesia, loss of vision and deafness.

Reflex Symptoms.

The condition of the superficial reflexes has been noted in three cases only, and in these they were diminished.

Deep reflexes were in some cases diminished, in others exaggerated.

In the organic reflexes, retention and incontinence of urine, incontinence of faeces, Cheynes-Stoke breathing, aphasia (in one case), and dysphagia were observed in different cases.

Trophic Symptoms.

An acute bedsore was present in one case (No.26). Atrophied joints and muscles occurred in several cases. Inflammation and sloughing of the cornea took place in one case (No.36).

Enlargement of Head.

Enlargement of the head was noted in two of my cases (Nos.2 and 24). These cases proved to me so

interesting that I investigated the literature of the subject.

The skull to all intents and purposes forms a completely closed box, entirely filled by the delicate and vitally important organ, the brain with its coverings. If these contents are added to, as by a tumour, there is an increased intracranial pressure. The brain does not transmit this pressure equally in all directions, the strong falciform ligament and the tentorii cerebelli dividing the cranial cavity into three chambers more or less separated from each other. Evidences of the increased pressure was shown post-mortem, by the flattening of the convolutions, obliteration of the sulci, pallor of the surface of the brain, the dry sticky condition of its membranes and the scanty amount of sub-arachnoid fluid.

It is the increased intracranial pressure which causes most of the general symptoms, such as headache, vomiting, vertigo, etc., of intracranial tumour. Thus where the pressure is great, these symptoms will be most marked, but where it is slight, they will also be slight or even absent altogether.

The degree of the intracranial pressure will depend on the size of the tumour, and the presence or absence of dropsical effusion into the lateral

ventricles. The rate of increase will depend on the rapidity of growths of the tumour.

Tumours situated in the neighbourhood of the third ventricle and quadrageminal bodies (Cases Nos. 2 and 24) cause a dropsical effusion into the lateral ventricles. This is due to pressure on the straight sinus, producing mechanical congestion of the great veins of Galen, as well as of their radicles, on the walls of the lateral ventricles. Dr Stephen Mackenzie (Lancet, Vol.I., 1880, p.559) does "not think "that this is the sole or essential cause". He considers that "subtentorial tumours, especially when "involving the middle lobe, will be extremely likely "to cause constriction or obliteration of the cere-"bro-spinal foramen, or some point above this - the "fourth ventricle or aqueduct of Sylvius ..... When "the communication between the general ventricular "cavity and the sub-arachnoid space is obliterated, "the fluid poured out by the choroid plexuses, and "possibly the obstructed venae Galeni, is dammed up, "distends the ventricles and causes the extreme "hydrocephalus sometimes found."

Tumours in any part of the brain may, I believe, be associated with hydrocephalus, yet they may be placed, e.g. in the frontal and sphenoidal lobes, that they cannot directly cause any obstruction to

the outlet of the ventricles. The probable explanation, I think, is that when the increased pressure drives down the cerebellum and pons into the foramen magnum the passage of fluid from the fourth ventricle into the sub-arachnoid space is choked.

Pressure on the iter by cerebellar or pontine tumours will cause hydrocephalus. Blockage of the exit of the fourth ventricle by tumours of the cerebellum or medulla will also cause it.

Owing to the increased intracranial pressure and the dropsical effusion into the ventricles, the tumour to get room to grow may have to

(1) Eat away the bony walls of the skull. Hale White (Conditions of the Bones of the Skull and Dura Mater in Cases of Tumours of the Brain, Guy's Hosp. Rec., Vol.43, 1886) examined a large number of skulls of patients who had died from cerebral tumours and found thinning of the bones.

This usually corresponds with the position of the tumour, but it also extends over the roof and sides. Hale White reports a case where actual perforation occurred into the tympanum.

(2) Separate the sutures. Bonetus in 1691 said "The head can scarcely grow on account of the solid-"ity of the bones unless the sutures become separat-"ed, which happens on account of the greatness of the

"tumour." (Theasurus Medico Practicus, Vol.I., p. 630, Geneva, 1691.) Gowers (Diseases of the Nervous System) says: "Even the sutures of the skull may be "separated by the powerful distensile force." Hale White does not mention any increase in the cranial dimensions except in the case of a boy aged $7\frac{1}{2}$ years whose cranial bones, "seemed not to have come to-"gether."

In Case (No.2) of my series, a boy of $4\frac{1}{2}$, the first symptom noticed by his parents was the enlarging head, and the sutures were found to be separated. In Case (No.24), a boy, age $6\frac{1}{2}$, the longitudinal and frontal sutures were open about three quarters of an inch.

In both these cases the tumours were tubercular, were situated in the cerebellum and were accompanied by extensive dropsical effusion into the ventricles.

COMPLICATIONS.

Meningitis was a complication which accompanied six of my twenty-eight tubercular cases, as a general tubercular meningitis, the symptoms of which obscured the clinical picture of the tumour. Below are two tables (Nos.22 and 23) which I have put for comparison.

No.22 shows the number of cases in which the symptoms of meningitis were noted during life. No.23 shows the cases in which meningitis was found post-mortem.

Table No.22.

Tubercle	6 cases.
Sarcoma	0 cases.
Glioma	0 cases.
Total	6 cases.

Table No.23.

	General.	Local.	Absent.
Tubercle	6	16	6
Sarcoma	0	1	3
Glioma	0	1	3
Totals	6	18	12

Pulmonary Tuberculosis was noted in sixteen of the tubercular cases during the life time of the patient (see frontispiece).

Tubercular Bones and Joints. 3 Cases (Nos.14, 29 and 30).

General Tuberculosis. 1 Case (No.13).

The following table shows the complications which were found post-mortem. I append it in order to compare with the conditions stated above.

Table No.24.

Pulmonary Tuberculosis	12 cases.
Pulmonary Tuberculosis and Tubercular Bronchial Glands ...	4 cases.
Tuberculosis of Bronchial Glands ..	3 cases.
General Tuberculosis	1 case.
Tubercular Bones and Joints	3 cases.
Tubercular Mesenteric Glands	2 cases.
Total	25 cases.

Thus there were only three cases of tubercular tumour which were without complications of some sort.

DIFFERENTIAL DIAGNOSIS.

Is there an intracranial tumour present?

The existence of a tumour in the cranial cavity is probable when the general symptoms such as headache, optic neuritis, characteristic vomiting, and a slow pulse are present.

The following table, No.25, shows the diagnosis with which twelve of my cases were sent into the Infirmary.

Table No.25.

1.	Tubercular Meningitis	6 cases.
2.	Hydrocephalus	2 cases.
3.	Cerebral Abscess	1 case.
4.	Hemiplegia	1 case.
5.	Pulmonary Tuberculosis	1 case.
6.	General Tuberculosis	1 case.

In the case of the Tubercular Meningitis - it was in the six cases a complication of the tubercular tumour-formation which was present within the cranial cavity. Optic neuritis, as is well known, may be present in tubercular meningitis - but not always and is not nearly so distinct as that present with intracranial tumour.

I have carefully examined the fundi oculi of five cases of uncomplicated tubercular meningitis, the diagnosis being subsequently confirmed by post-mortem examination. In three cases there was a blurring of the disc; in one there was some swelling of the veins, but no appearance of neuritis, and in the remaining one there was no alteration during the whole course of the disease.

Cases Nos.2 and 24 were admitted with a diagnosis of Hydrocephalus, but both had intense optic neuritis (also optic atrophy in No.24), and the other symptoms were so typical of, that there was no difficulty in arriving at a diagnosis of, tumour in the cerebellum with dropsical effusion into the ventricles in each case. This was subsequently confirmed at the autopsy.

Case No.10 was admitted as a case of Cerebral Abscess, but on careful examination I could find no suppurative condition in the throat, ear or nose. The onset of the illness was sudden, but in the absence of any local cause and the slow regular course, I arrived at the diagnosis of tumour or tumours in the cranial cavity. Optic neuritis was present.

Case No.18 was sent in as a Hemiplegia, but the slow onset and gradually advancing paralysis led one to the proper diagnosis. There was no optic neuritis present in this case.

Case No.9 was admitted as a Pulmonary Tuberculosis and No.20 as a General Tuberculosis. These conditions overshadowed the head symptoms which, however, were typically present. Optic neuritis was well marked in both cases.

I always examined the urine to exclude Chronic Bright's Disease as the causation of head symptoms,

but never had any case of that condition with symptoms resembling those of intracranial tumour.

The more I have studied intracranial tumour and its diagnosis, the more I find I rely on the presence or absence of optic neuritis. The only cases where any difficulty arises, however, is I think where optic neuritis is absent, or developed very late in the disease.

In Case No.36, a glioma, the optic neuritis did not appear until the day before the child died but here there was little difficulty in arriving at a diagnosis before that, because the other general symptoms were typically present.

There were only four of my cases in which papillitis was absent, as is shown in the following table.

Table No.26.

(Optic Neuritis Absent).

Case	Duration	Tumour	Situation
No.6	3 months	Tubercular	Pons Varolii
No.11	$3\frac{1}{2}$ months	Tubercular	Cerebral
No.18	2 months	Glioma	Pons Varolii
No.29	4 months	Tubercular	Cerebellum

In Case No.6 the duration of the disease, the slow advancing paralysis and the involvement of cranial nerves made the diagnosis.

In Case No.11 there were the other typical general symptoms indicating the presence of a tumour within the cranial cavity.

In Case No.18 there were no general symptoms present, but the localising symptoms of paresis advancing to paralysis led me easily to the diagnosis.

In Case No.29 there was the least difficulty because the other general and the localising symptoms were typical of a tumour situated in or about the cerebellum.

REGIONAL DIAGNOSIS.

In Case No.1 there were no less than seven tubercular tumours, five through the cerebral hemispheres, and two (one about the size of a small hen's egg) in the cerebellum; and yet the only localising symptom was internal squint of the left eye, the general symptoms, however, being well marked.

In Case No.23 there was a tumour about the size of a boy's marble in the right corpus striatum, but there were no localising symptoms present.

It is therefore evident that tumours may be of considerable size or may even be multiple without creating any focal symptoms, or they may be multiple and only one give rise to localising symptoms.

In the following tables I will attempt to classify the localising symptoms peculiar to each region or regions affected in the cases of my series. I will not emphasise the general symptoms which were to a great or less degree present in all cases but one.

Tumours of the Cerebellum.

The table No.27 shows the cases where the cerebellum only was affected, and the important localising symptoms noted briefly in each case.

Table No.27.

Case	Tumour	Localising Symptoms
No.4	Sarcoma	Blindness. Retraction of Head.
No.7	Tubercular	Ataxia. Tremor.
No.9	Tubercular	Tetanus. Trismus. Ataxia.
No.12	Tubercular	Paresis of right arm. Ataxia.
No.14	Tubercular	Right Strabismus.
No.17	Sarcoma	Ataxia. Rigidity of neck.
No.24	Tubercular	Ataxia. Enlargement of Head.
No.29	Tubercular	Difficulty of speaking. Facial Paralysis.
No.32	Glioma	Strabismus (Rt. Internal) Deafness. Ataxia. Right Facial Paralysis, reflexes exaggerated.
No.35	Tubercular	Ataxia.

The next table, No.28, gives a list of cases where the cerebellum was largely affected and adjacent regions with the important localising symptoms briefly noted in each.

Table No.28.

Case	Parts affected	Localising Symptoms
No.2	Cerebellum, Cerebrum.	Enlargement of head, Internal squint.
No.3	Cerebellum, Pons, Cerebrum.	Ptosis and Hemiplegia.
No.22	Cerebellum, Pons, Crura Cerebri.	Internal squint. Ataxia.
No.28	Cerebellum, Lenticular Nucleus.	None.

The tumours in these four cases were all tubercular. Judging from the above two tables, one is justified, I think, in giving the following Table (No.29) as the important localising symptoms of cerebellar tumour.

Table No.29.

Localising Symptoms of Cerebellar Tumour.

1. Internal Strabismus.
2. Ataxia.
3. Enlargement of Head.
4. Facial Paralysis.

5. Ptosis.
6. Tremor.
7. Difficulty in speaking.
8. Deafness.
9. Rigidity of neck.
10. Exaggerated reflexes.
11. Monoplegia.
12. May be entirely absent.

It will be observed that unilateral paralysis of the cranial nerves (the third, fifth, sixth, seventh and eighth) may occur, and form important localising symptoms.

Tumours of the Pons Varolii.

Table No.30 gives the cases with the pons Varolii only affected, and Table No.31 the cases where there are other parts affected as well.

Table No.30.

Case	Tumour	Localising Symptoms
No.6	Tubercular	Internal squint. Facial paralysis. Spastic Gait. Exaggerated reflexes.
No.18	Glioma	Hemiplegia of left side, facial paralysis of right.
No.21	Glioma	Staggering.

Table No.31.

Case	Other Parts affected	Localising Symptoms
No.3	Rt. Cerebrum, Cerebellum.	Hemiplegia. Ptosis. Exaggerated Reflexes.
No.8	Rt. Cerebrum, Optic Thalmus.	Hemiplegia. Facial paralysis. Hyperaesthesia.
No.10	Cerebral Hemispheres	Hyperaesthesia. Facial paralysis. Strabismus. Nystagmus. Aphasia. Tremors.
No.22	Crura Cerebri, Cerebellum	Strabismus. Ataxia.
No.25	Cerebrum	Paralysis of right side with left facial paralysis. Squint.
No.36	Medulla Oblongata. Crus Cerebri.	Internal squint. Left hemiplegia. Tremor.

From these tables it would appear to me that the combination of symptoms in disease of the pons Varolii depends on the juxtaposition of the motor tracts and the cranial nerve centres.

Alternate paralysis (paralysis of upper and lower limbs on opposite side with paralysis of the face of the peripheral type on the same side) was present in three of the cases.

Tumours of the Medulla Oblongata.

Case No.36 is the only case where the medulla oblongata was affected in my series. It was secondarily affected along with the pons Varolii and crura cerebri.

The symptoms were, as already stated, right facial paralysis (peripheral type), paralysis of the left upper and lower limbs, internal squint of the right eye, with inflammation and sloughing of the cornea, and dysphagia.

Double optic neuritis was present, but it commenced only the day before the girl died.

Tumours of the Crura Cerebri.

The crura cerebri were affected in three of my series but adjacent and other parts of the brain were so involved that it was impossible to differentiate the symptoms peculiar to this region.

Tumours of the Quadrageminal Region.

In Case No.5, a boy aged 11 years, a sarcoma was found at the autopsy situated in the corpora quadragemina.

The general symptoms were but slightly marked except the pulse which was very slow - 56 on admission. He reeled about like a drunk person, and

there were clonic convulsions affecting the muscles of his arms and legs. His eyes were staring, pupils dilated, conjunctival reflexes gone. There was a paralysis of convergence, ptosis, and a loss of the upward movement of the eyes.

Tumours of the Corpus Striatum.

Case No.23, a girl age 7, was admitted with all the typical symptoms of an intracranial tumour, viz: headache, sickness, vomiting and optic neuritis. The clinical picture was, however, obscured by the symptoms of tubercular meningitis - from which she died. There were no definite localising symptoms. At the autopsy a tubercular tumour was found situated in the right corpus striatum.

Tumours of the Optic Thalmus.

A boy aged 4 years, Case No.13, was sent to the Infirmary as a case of general tuberculosis, and on examination he was found to be suffering from a hemiplegia and hemianaesthesia of the left side, and in addition he had headache, vomiting, vertigo and optic neuritis. At the post-mortem examination subsequently held a tumour was found in the right optic thalmus compressing the internal capsule, and another tumour (both were tubercular) in the right occipital

lobe.

Case No.19, a girl age four years, was admitted with severe headache and giddiness: and optic neuritis was found present in both eyes - more marked on the left eye - but no localising symptoms of a tumour could be obtained. At the post-mortem examination subsequently held there were two tumours of a tubercular nature found, one over the right optic thalmus and another on the left. The bronchial glands were also tubercular.

Tumours of the Corpus Callosum.

In Case No.27, at the post-mortem examination, the brain weighed 30½ oz. and two tumours of a tubercular nature were found; one, about an inch in diameter, being situated in the corpus callosum, and the other, a much smaller mass, in the right olivary nucleus.

The case was a girl three and a half years old sent to the Infirmary as a case of tubercular meningitis. She had, however, been suffering for four and a half months from headache, severe vomiting and giddiness, and latterly from convulsions with clonic spasms of the head and leg. Examination of the fundi oculi revealed marked optic neuritis. There was some pulmonary tuberculosis. The localising

symptoms were not definite and in the copious notes of the case I failed to find the train of symptoms stated by Dr Bristowe (Brain, Vol.XII., p.318) to be characteristic of a tumour situated in this region.

Tumours of the Internal Capsule.

The only case of this is in Case No.13 where the tumour in the right optic thalmus caused compression of the internal capsule and a hemiplegia and hemianaesthesia followed.

Tumours of the Occipital Lobe.

In the same case, No.13, a small tumour was found situated in the right occipital lobe but there were no localising symptoms during life, other than from involvement of the internal capsule.

Tumours of the Temporal Lobe.

I have no case in my series where the temporal lobe only was affected; but Cases Nos.10 and 26 this lobe was affected along with other parts of the brain. In neither case was there any indication of paraphasia.

In Case No.10 there was inability to speak from paralysis.

Tumours of the Parietal Lobe.

In Cases Nos.10 and 26* the parietal lobule was also affected, but the symptoms, if any, were overshadowed by the involvement of the adjacent Rolandic area.

Tumours of the Rolandic Area.

The following table shows the cases in which the Rolandic area was involved and the probable localising symptoms.

Table No.32.

Case.	Localising Symptoms.
No.11	Local convulsions of left side. Hyperaesthesia. Paresis of left side.
No.30	No localising symptoms.
No.31	Left brachial monoplegia. Left facial paralysis.
No.33	Complete left hemiplegia and hemianaesthesia.

Thus the localising symptoms of a tumour in this region I would classify as follows:-

Table No.33.

Localising Symptoms of Tumours of Rolandic Area.

1. Irritative.

   Hyperaesthesia.
   Local Convulsions.

2. Destructive.

   Monoplegia.
   Complete Hemiplegia.
   Hemianaesthesia.

3. May be no localising symptoms.

Tumours of the Frontal Region.

Case No.20, Annie Danuel, a girl age 9, was admitted to the Infirmary on November 12th and died on November 17th, 1910. At the post-mortem examination the brain was found to be unusually soft and weighed thirty-six ounces. The dura mater was not adherent to the skull and there was no evidence of a general meningitis. At the lower part of the frontal lobes and situated in the longitudinal fissure in such a way as to unite the two lobes, there was a tumour about the size of a duck's egg. It was hard, almost cartilaginous, in consistency and the brain around the tumour was soft and pulpy. Examination of the tumour showed it to be of a tubercular nature.

The history was that the patient for some time

had had a peculiar walk. Only a week before did she begin to complain of headache and the day after she took to bed. She became stupid and difficult to wake up; this became gradually worse until she became quite unconscious. On admission she was quite unconscious but was able to swallow fluids. Her pulse was 120, respirations were 32. There were no physical signs present in the chest. Optic neuritis was well marked in both eyes. Localising symptoms were absent, the only symptom that might be accepted as typical of the region was the drowsy stuporose condition. It is to be observed that the tumour was situated well to the front portion of the lobes, away from the Rolandic area.

This case was sent in and admitted as one of tubercular meningitis.

The following table (No.34) gives the cases where the frontal and other regions were affected with the important localising symptoms briefly noted.

Table No.34.

Case	Position	Localising Symptoms
No.1	Cerebrum and Cerebellum	Internal squint
No.10	Left Frontal Lobe, Pons	Aphasia
No.20	Frontal Hemispheres	?
No.26	Frontal and Rolandic areas.	Left Hemiplegia

In these four cases the tumours were all of a tubercular nature.

The only interest in this table is the aphasia produced in Case No.10 from the involvement of Broca's convolution.

PATHOLOGICAL DIAGNOSIS.

Tubercular Tumours.

Intracranial tumour in children is so frequently tubercular that I conclude it to be so unless there are signs to make me suspect the contrary. The points I always ascertain to confirm the diagnosis, or otherwise, are as follows:-

1. Associated tubercular conditions, or
2. Previous tubercular trouble.
3. Family history of tuberculosis.
4. Definite history of injury to head causing the onset of the symptoms.
5. Early and marked emaciation.
6. Calmette's conjunctival reaction.
7. Von Pirquet's Test.
8. Absence of very marked improvement on treatment with potassium iodide and mercury.

The following table, No.35, illustrates the importance of these points, in diagnosis, as applied to my cases.

Table No.35.

Associated tubercular condition	17 cases.
Previous tubercular history	3 cases.
Family History of Tuberculosis	7 cases.
History of injury to head	4 cases.
Early emaciation	5 cases.
Calmette's Conjunctival Reaction ..	3 cases.
Von Pirquet's Test	
No reaction to potassium iodide ...	

The last three tests were applied in the three cases only because there was some doubt as to whether the condition was tubercular or not.

Syphilitic Tumours.

These are readily diagnosed by treatment with potassium iodide. I have already referred in full to two cases (Nos.37 and 38) where the diagnosis and treatment of gummata of the brain were successfully carried out by the administration of potassium iodide.

Wasserman reaction is now a valuable ally in diagnosing lesions of a suspected syphilitic nature.

Sarcoma and Glioma.

When we can definitely exclude tuberculosis and syphilis, the tumour may be either sarcoma or glioma. If these are primary, as in my eight cases, the diagnosis between the two formations is practically impossible; as a matter of fact these tumours are often more or less combined as a glio-sarcoma.

PROGNOSIS.

The only recoveries in my series of cases were the two cases of gumma. The remaining thirty-six all died sooner or later either as the direct result of the tumour or from some associated morbid condition (see Table No.36).

The disease is therefore so fatal a one that when we are satisfied of the existence of a tumour of the brain we can have little expectation - tumours due to syphilis excepted - of the child recovering.

Course.

In the majority of the cases the progress is markedly slow - lasting it may be years (see Table No.37), and the course is steadily downwards.

In Case No.3, tubercular, the illness commenced with a fit, and for six years thereafter there was only a constant frontal headache. When acute symptoms supervened, she died within two weeks.

In Case No.13, the symptoms of brain tumour came and went in exacerbations.

In all the other cases, gummata excepted, the course was steadily downwards.

Duration.

The following tables show the duration of my cases.

Table No.36.

	Shortest	Longest	Average
Tubercular	3 weeks	6 years	9 months
Sarcoma	1 month	3 months	2 months
Glioma	1 month	10 months	5 months

Tubercular tumour in the brain may thus last for one or two years.

In Case No.3 the condition remained more or less stationary for six years.

The sarcomata and gliomata are evidently much more rapid, the former lasting from one to three

months, and the latter from one to ten months.

Terminations.

Table No.37 shows the immediate cause of death in the thirty-six cases.

Table No.37.

Causes of Death.

From Exhaustion	11 cases.
Tubercular Meningitis	6 cases.
Pulmonary Tuberculosis	8 cases.
After Operation	5 cases.
From a Fit	3 cases.
Pulmonary Congestion	2 cases.
Heart Failure	1 case.
Total	36 cases.

TREATMENT OF THE CASES.

General Measures.

The usual attention was always paid to the stomach, bowels, and the avoidance of bedsores.

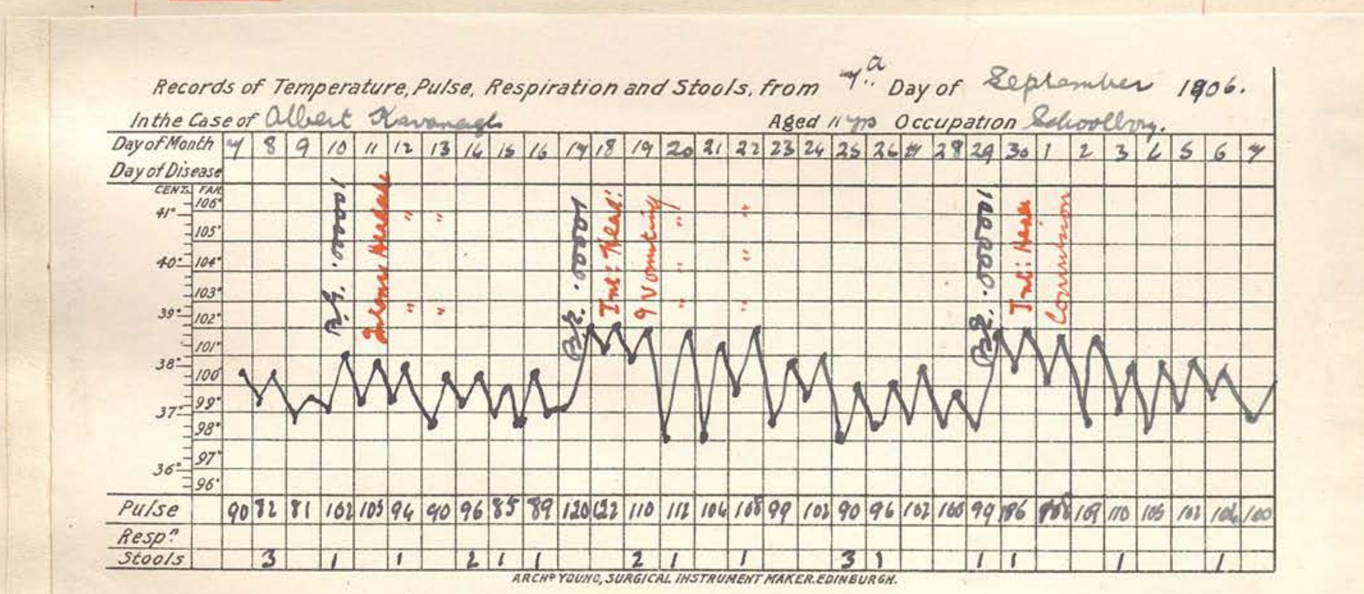
Medicinal Treatment.

In five cases of intracranial tumour, although

there was no evidence of syphilis, potassium iodide was administered. Case No.34, tubercular, seemed for a time to improve on it, whilst the two gummatous cases (Nos.37 and 38) recovered. Cases Nos.10 and 20 showed no improvement.

Tuberculin Treatment

for tubercular intracranial tumour may be a thing of the future, but at present, in my experience at any rate, tuberculin aggravates the condition, as can be seen in the following chart of Case No.29.



Symptomatic Treatment.

Headache. - If severe, an icebag was applied to the head, and hot bottles to the feet, whilst calomel was given internally.

Acetanilid, phenacetin, and other drugs usually

given for ordinary headache are, I find, of little use.

Vertigo. - Complete rest was always enforced when the vertigo was severe.

In Case No.8 counter irritation in the form of a small blister was applied to the mastoid region, and it seemed to give some relief.

Vomiting. - Complete rest in the recumbent position often relieved this condition. Iced milk or iced water was frequently given where the vomiting was severe, whilst in three very obstinate cases we had recourse to nutrient enemata.

General Epileptic Convulsions. - If convulsion rapidly succeeded convulsion, we usually administered a little chloroform.

In Case No.3 an inhalation of nitrite of amyl proved very useful: this is recommended by Dr John Thomson (Clinical Examination and Treatment of Sick Children, p.136).

Treatment of Paralysis.

The paralysed parts were kept warm night and day, and massaged for about a quarter of an hour twice a day.

Records of Pulse, Temperature, Respirations

and Stools in

Case No.32.

Glioma of Cerebellum-Operation

Robert Ulbeck. Age. 4.

admitted 16th Dec. 1905 Operation. - Jan^y 4th 1906.

Died 6 Jan. 1906.

Dec. Jan.

Date	24	25	26	27	28	29	30	31	1	2	3	4	5	6
Time														
BOWELS	0	0 0	1 0	0 0	0 0	1 1	1 1	1 1	0 0	0 1	0 0	1	1 1	1
PULSE	96/108	104/88	120/104	106/102	99/102	54/98	108/104	120/120	125/124	110/118	120/128	120/140	100/160	160/160
RESPIRATION	35	40	38	42	40	40	42	34	41	40	41	40	40 52	40 72

Fahrenheit: 106° 105° 104° 103° 102° 101° 100° 99° Normal 98°

Operation

Exam 10.45 pm

Operative Treatment.

Operation for the removal of an intracranial tumour was attempted in five of the cases.

1. We thought we had localised the precise position of the tumour, and that was within reach.
2. The damage to the brain did not seem extensive.
3. The tumour appeared to be single.
4. There seemed to be no associated morbid condition in these cases.

The operations were all more or less completely unsuccessful, as can be seen in the following Table No.38.

Table No.38.

Results of Operation.

Case	Age	Tumour	Result
No.4	12	Sarcoma	Lived 3 days
No.5	11	Sarcoma	Lived 8 days
No.11	3	Tubercular	Lived 4 days
No.17	9	Sarcoma	Lived 5 days
No.32	7	Glioma	Lived 2 days

Operation for the removal of an intracranial tumour cannot at the present time be recommended.

MORBID ANATOMY.

Tubercular Tumours.

In my twenty-eight cases there were altogether fifty-seven tumours distributed as follows:-

Table No.39.

1. Single ..... 14 cases.

2. Multiple ..... 14 cases.

Two Tumours	7 cases
Three Tumours	3 cases
Four Tumours	2 cases
Five Tumours	1 case
Seven Tumours	1 case

Sizes. The largest tumour was, in Case No.20, about one inch in diameter. The smallest were present in Case No.1, where three of the seven tumours were about twice the size of a green pea. The common size was that of a marble.

Situations. The situations of the fifty-seven tumours are shewn in the following table.

Table No.40.

	Single	Multiple	Totals
Cerebellum	6 Tumours	11 Tumours	17 Tumours
Pons Varolii	3 Tumours	9 Tumours	12 Tumours
Cortex Cerebri	4 Tumours	7 Tumours	11 Tumours
Corpora Quadr.	0 Tumours	9 Tumours	9 Tumours
Basal Ganglia	0 Tumours	8 Tumours	8 Tumours
Totals	13 Tumours	44 Tumours	57 Tumours

General meningitis was present in six cases, local meningitis in sixteen cases (see Table No.23).

Sarcomata

were single and primary in the four cases.

Sizes:

Case No.4 ..... Hen's Egg.

Case No.5 ..... Filbert.

Case No.15 ..... Hen's Egg.

Case No.17 ..... Large Walnut.

Situations:

Case No.4 ..... Cerebellum.

Case No.5 ..... Corpora Quadr.

Case No.15 ..... Optic Thalamus.

Case No.17 ..... Cerebellum.

Microscopically round cells were present in all four cases.

Gliomata

were single in my four cases and all were a greyish white red colour. They had the appearance of a local hypertrophy of the normal brain matter, but could be differentiated by the difference in colour and consistence.

Sizes:

Case No.18	.....	Walnut.
Case No.21	.....	Filbert.
Case No.32	.....	Hen's Egg.
Case No.36	.....	Child's clenched fist.

Situations:

Case No.18	.....	Pons Varolii.
Case No.21	.....	Pons Varolii.
Case No.32	.....	Cerebellum.
Case No.36	.....	Pons Varolii.

In September 1909, I was able to study from the beginning what afterwards proved to be a glioma of a typical nature, Case No.36. A girl aged 10 was admitted with the general symptoms of intracranial tumour and symptoms locating it in the pons Varolii.

At the post-mortem examination the tumour was found at the base of the brain as a diffuse overgrowth imperfectly demarcated from the surrounding nerve tissue, and looking as if it were a local hypertrophy of the brain. It was single, as large as a child's clenched fist, soft and almost fluctuating to touch. It took origin from the vicinity of the pons, chiefly on the right side, and extended over the right crus cerebri, and the right medulla oblongata for about an inch. The left pons and the left medulla oblongata were similarly but much less affected. The basilar artery with its branches were embedded in the growth, so as to appear as mere sulci in it.

According to Bland Sutton (Tumours, Innocent and Malignant, p.64), this form, with the gliomatous tissue so abundant as to produce an enlargement of the pons and cerebral peduncles, is rare, and in nearly all cases the child is under twelve. He quotes the following cases, Percy Kidd's case, girl age $6\frac{1}{2}$ (St. Bart's Hosp. Reports, Vol.13, p.257). Gee's case, boy age 9 (St. Bart's Hosp. Reports, Vol.17, p.285).

Microscopically the growth in case No.36 presented very numerous cells with single nuclei, and a scanty amount of protoplasm. The cells had numerous branched processes which interlaced to form a

delicate network. There were numerous dilated thin-walled blood vessels, and there were numerous red blood corpuscles scattered through the section.

SUMMARY OF CLINICAL CONCLUSIONS.

(See Frontispiece.)

1. Thirty-eight cases of Intracranial Tumour in Children admitted during four years to the Infirmary for Children, Liverpool, were as follows:-

Tubercular	28 Cases	= 73.4%
Sarcoma	4 Cases	= 10.5%
Glioma	4 Cases	= 10.5%
Gumma	2 Cases	= 5.2%
Total	38 Cases	

I had no cases of cysts or carcinomata of the brain, so I have necessarily come to the conclusion that these conditions are rarely met with in children.

2. Contrasted with other organic diseases of the central nervous system, intracranial tumours are

relatively rare, but the brain is one of the most common sites of tumour formation in the body. (Table No.2)

3. The common age for tubercular tumour in the brain of children seems to be between three and five years old. Sarcomata and gliomata appear at rather later ages. (Table No.3)

4. Intracranial tumour in children affects boys more commonly than girls (Table No.4), possibly because the former are more liable to injury than the latter. (See Table No.10)

5. In 25% of the tubercular intracranial tumours there was a definite family history of tuberculosis. (Table No.5)

6. Measles was a frequent (17.8%) precursor of the tubercular intracranial tumour. (Tables 6 and 7)

7. Tubercular intracranial tumour is usually secondary to a focus elsewhere in the body: this focus, in 85% of my cases, was situated in or about the lungs (Tables 8 and 9): in only three cases (10.7%) was the condition primary.

The sarcomata and gliomata of the brain were all primary.

8. In 13.2% of my cases the condition seemed to arise definitely after a blow or other injury to the head. In these cases the tumour did not develop just at the site of injury but in some other part of the brain. (Table No.10)

Brain tumour following injury is usually tubercular; occasionally gliomatous. (Table No.10)

9. The manner in which the symptoms are grouped together varies considerably in different cases. (Table No.11). In the majority of the cases (60.5%) general and localising symptoms are present - the former preceding the latter.

10. In 89% of the cases the disease was slow in onset. In the remaining (11%) the onset was acute with convulsions.

11. The first symptom noted was headache in 37% of the cases, vomiting in 21% and convulsions in 10.5%. (Table No.12)

12. After the onset the condition may remain latent for a considerable time. (Case No.2)

13. Headache was present in 58% of the cases. It may undergo exacerbations. (Case No.13).

Occipital headache may be a symptom of tumour situated in the posterior portion of the brain, but there is no evidence to conclude that frontal head-

ache can be relied upon as an indication of the site of the tumour. (Table No.13)

14. Every case of vomiting for which no obvious explanation is forthcoming should have the fundus oculi examined for optic neuritis. (See Case No.6)

15. Tumours situated in the pons Varolii or its neighbourhood seem to be associated with early and persistent vomiting. (Table No.15)

16. Optic neuritis is, in my opinion, the most important general and diagnostic symptom: it may be present without disturbance of the acuity of vision and therefore all cases with head symptoms should have the fundi oculi examined for neuritis whether the vision is interfered with or not.

17. Optic neuritis is usually early in onset and intense in character in tumours of the cerebellum and these cases have often post-neuritic optic atrophy.

18. Enlargement of the head through separation of the sutures may be a symptom of tumours about the pons Varolii, the cerebellum or the medulla oblongata.

The separation of the sutures of the skull in these cases is due to the greatly increased intra-

cranial pressure due not only to the tumour but also to a very considerable dropsical effusion into the ventricles.

19. The existence of a tumour in the cranial cavity is probable when the general symptoms such as headache, optic neuritis, characteristic vomiting and a slow pulse are present, but our stand-by in the differential diagnosis is the presence or absence of optic neuritis. The only cases where real difficulty in the diagnosis arises is where the optic neuritis is absent or develops very late in the disease.

20. The diagnosis of the position of the tumour is always difficult since tumours may be of considerable size or may even be multiple without creating any focal symptoms, or they may be multiple and only one give rise to localising symptoms.

21. The diagnosis of the pathological nature of the tumour is often difficult, sometimes impossible - gummatous cases excepted.

22. The disease is so fatal a one that when we are satisfied of the existence of a tumour of the brain we can have little expectation - tumours due to syphilis excepted - of the child recovering.

23. Treatment is unsatisfactory and mainly

symptomatic - gummatous cases again excepted.

24. Tuberculin treatment for tubercular tumour of the brain cannot, as yet, be recommended.

25. Operation for the removal of an intracranial tumour is also not to be recommended.

26. The most frequent site for intracranial tumour in children is the cerebellum: next in frequency comes the pons Varolii.

BIBLIOGRAPHY.

Drs ASHBY and WRIGHT: Diseases of Children.

Dr BONETUS: Theasurus Medico-practicus, Vol.I.

Dr BRISTOWE: Brain, Vol.XII.

Dr DEUTSCHMANN: Ophthalmic Review, April 1887.

Dr HILTON FAGGE: System of Medicine, Vol.IV., 4th Edition.

Dr VON GRAEFE: Archiv. fur Ophthalmologie, 1860.

Dr GOWER: Diseases of the Nervous System, Vol.II.

Dr HUGHLINGS JACKSON: Transactions of the Ophthalmological Society of the United Kingdom, Vol.I.

Dr STEPHEN MACKENZIE: Transactions of the Ophthalmological Society of the United Kingdom, Vol.I. Lancet, Vol.I., 1880.

Dr BLAND SUTTON: Tumours Innocent and Malignant. St. Bart's Hosp. Reports, Vols. 13 and 17.

Dr JOHN THOMSON: Clinical Examination and Treatment of Sick Children.

Dr HALE WHITE: Conditions of the Bones of the Skull and Dura Mater in Cases of Tumours of the Brain. Guy's Hospital Reports, Vol.43, 1886.