Brainstem haematomas: review of the literature and presentation of five new cases

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SUMMARY Fifty-one cases of brainstem haematoma that have undergone neurosurgical intervention since Finkelnburg's first exploration in 1905, are presented, together with five new cases. The location, incidence, aetiological and pathological factors, as well as clinical syndromes are reviewed. Diagnostic criteria are presented. The distinction between brainstem "haematoma" and "haemorrhage" is stressed. A retrospective comparison of 56 surgically treated cases and 31 conservatively managed cases is made, as is an analysis of the natural history of the disease process. Based on currently available data, the conclusion is drawn that patients with brainstem haematomas fare quite well after surgical therapy.

Nearly half a century after Finkelnburg first explored the brainstem for haematoma¹ Teilmann² reviewed the world literature and provided summaries of 46 cases of documented vascular malformations of the pons. He thus established a new clinicopathological syndrome that (quoting McLean³) "... produces a kaleidoscopic wealth of signs which are ... a neurological delight for diagnosis, and neurosurgical despair for therapeutics."

With the advent of computed tomography and microsurgical techniques, the burdens of both diagnosis and "therapeutics" have become considerably lighter. Since the plea for surgical exploration of suspected lesions in the brainstem was made by Lassiter, *et al.*,⁴ in the hope of finding surgically approachable cystic tumours, a number of neurosurgeons have encountered and successfully drained unsuspected brainstem haematomas.

The purpose of this report is to document our experience with five patients harbouring brainstem haematomas, to present a comprehensive review of cases from the world literature that have undergone neurosurgical therapy, and to discuss views on the pathophysiology and management of these lesions.

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Classification: Distinction of haematoma from haemorrhage

A careful distinction must be made between (1) focal subependymal brainstem haematoma, and (2) diffuse tegmentobasilar hypertensive haemorrhage (fig 1). The subependymal haematoma is most often the result of bleeding from a cryptic brainstem vascular malformation, usually telangiectasia. It is a focal, compressive lesion which displaces rather than destroys brain tissue. It occurs in a younger age group and causes neurological deficits which are often partially reversible.

Hypertensive brainstem haemorrhage usually causes a diffuse lesion occurring in an older age group and most often associated with profound, irreversible neurological deficits which are often fatal.

Arseni and Stanciu⁵ described two similar groups of patients: "Group I" and "Group II". Papo, *et al.*⁶ referred to "intra-axial" and "subependymal" haematomas. Goto, *et al.*⁷ spoke of "tegmentobasilar" and "tegmental" haematomas. "Group II", "tegmental", and "subependymal" all describe the same lesion—the brainstem haematoma. It is usually limited by the ependyma of the fourth ventricular floor, although there may occasionally be leakage into the ventricular system, causing bloody CSF and nuchal rigidity. There may be extension laterally (mimicking a cerebellopontine angle tumour), or toward the brachium pontis.

Fig 1 Distinction between the focal subependymal brainstem haematoma and the diffuse tegmentobasilar hypertensive haemorrhage. The subependymal haematoma is most often the result of bleeding from a cryptic brainstem vascular malformation, usually a telangiectasia. It is a focal, compressive lesion which displaces rather than destroys brain tissue. It causes neurological deficits which are often partially reversible. The hypertensive brainstem haemorrhage results in profound, irreversible neurological deficits which are usually fatal.

Case material

Location

Among a series of 51 cases collected from the literature, one medullary and five midbrain haematomas are recorded; the remainder were pontine.

Incidence

Brainstem haematomas are rare. The incidence of both haematomas and haemorrhages in various necropsy series⁸⁻¹¹ has been noted to be between 6% and 9% of all intracranial haematomas. Dinsdale,⁸ in a review of 19,000 necropsies, found 30 cases of haemorrhagic brainstem lesions, 27 of which had ruptured into the fourth ventricle; three of these were brainstem haematomas.

The range of ages in this series is from 17 months to 65 years, with a mean age of 24 years. Seventeen patients were under 16 years of age.

Aetiological and pathological factors

It appears that the fatal brainstem haemorrhages are hypertensive in origin as has been noted in various necropsy series.^{8,9} Alternatively, non-fatal subependymal brainstem haematomas evolve differently, arising from the vascular malformations described below. Hypertension does not appear to play a role (having been noted in only three of the 56 cases outlined in table 1).

Virchow¹¹ first reported a haemangioma of the pons as an incidental finding at necropsy in 1851. Cushing and Bailey¹² elucidated the microscopic description of capillary telangiectasia, and noted that the floor of the fourth ventricle is a favoured site for them, most commonly in the upper pons. Russell¹³ coined the phrase "cryptic vascular malformation" using it to describe angiomatous malformations which measure less than 2-3 cm in maximum dimension. Of the 85 brainstem cryptic vascular malformations reviewed by McKormick and Nofzinger,¹⁴ more than 10 haematomas due to cryptic telangiectasia were noted. Gerlach¹⁵ spoke of

"microangiomas", defining them as malformations under 2 cm in diameter. With recent technology, the size of a "cryptic" lesion has been reduced considerably.

Courville¹⁶ commented on the tendency of these lesions to separate rather than destroy pontine fibres, thus accounting for their quiescent clinical status prior to rupture.

Teilmann² retrospectively reviewed 45 case of necropsy-documented pontine haemangiomas, nearly half of which were telangiectasias, one third were cavernous haemangiomas, and the remainder were venous or arteriovenous malformations.

On the other hand, Cole and Yates¹⁷ postulated that the most likely cause of the primary pontomedullary haemorrhages would be a microaneurysm arising from the vertebrobasilar arterial system. However, no such lesion has been noted in this review.

Margolis *et al*¹⁸ proposed that haematomas arising from cryptic malformation often result in the destruction of the anomalies when they bleed, thereby accounting for the high incidence of negative histological findings among such specimens. This has been true even when the wall of the haematoma has been biopsied. Of the 56 haematomas summarised in table 1, only 13 cases had positive pathological reports given. Eleven cryptic malformations and one venous angioma were noted. In one case,¹⁹ a recurrent pontine haemangioblastoma, complete with mural nodule, was found at necropsy in the site of a previously removed haematoma.

Antecedent trauma, usually minor, was noted in four of the surgically treated brainstem haematomas. Currier *et al*²⁰ noted a similar incidence (3/39) in patients with medullary infarction.

Clinical syndromes

Pathophysiologically brainstem cryptic malformations are congenital lesions that probably remain asymptomatic until they bleed. Periodic bleeding of small volume may explain the relapsing clinical course often noted. In some cases,

Table 1 (a) Patient characteristics

Case	Author	Year	Age (yr)	Sex	Clinical presentation	Physical examination
1	Finkelnburg ¹	1905	14	M	2.5 yr headache, episodic vertigo	?
2	Friedrich ³²	1922	41	M	2 yr intermittent dizziness and vomiting. 3 wks visual loss and (R) hemiparesis	Papilloedema, horiz nystagmus. (L) V, VI, VII. (R) hemiparesis
3	Marcus ³³	1925	32	F	1 yr relapsing and finally progressive diplopia, (L) hemimotor, -sensory, cerebellar dysfunction	Nystagmus, (L) V, VII, IX-XII deficits, (L) hemimotor, -sensory cerebellar deficits
4	Bailey ³⁴	1919	21	F	8 yr h/o episodic headache and transient unconsciousness	?
5	Berger ³⁵	1927	25	M	5 wk (L) hemiparesis (sudden onset): after remission: progressive (L) gaze paresis, (L) hemimotor, -sensory, cerebellar deficits	Horiz nystagmus, (L) hemiparesis (L) V, VII, VIII, IX-XII. Comatose after admission
6	Dandy ³⁶	1932	?	?	4 yr intermittent brainstem symptoms	?
7	Gros ⁴⁴	1948	32	M	1 m relapsing headache, tinnitus and vertigo. 2 wk (R) facial paresis, hearing loss, and diplopia	Nuchal rigidity, horizontal nystagmus. (R) V, VII, VIII, hemimotor, -sensory, cerebellar deficits
8	Scoville <i>et al</i> ⁴⁵	1949	44	F	2 yr intermittent headache, (R) hemisensory, cerebellar. 1 m bilateral III. 1 wk increasing headache, vomiting, stupor	Bilateral MLF, VII, (L) Horner's (R) hemiplegia. Comatose
9	Tenuto <i>et al</i> ⁴⁶	1958	33	F	3 yr intermittent headache and vomiting; (R) facial paraesthesias	Papilloedema. (R) V, VII, VIII. (R) hemisensory loss
10	Scott <i>et al</i> ⁴⁷	1960	41	M	3 wk headache, diplopia, (L) leg weakness 1 m later 2 m later	Horizontal nystagmus. (L) III, VI. (L) cerebellar dysfunction. Papilloedema, improved after discharge Comatose
11	Courville ¹⁶	1962	53	F	"Idiopathic" hydrocephalus, 9 yr after removal of (R) frontal convexity meningioma	Parinaud's quadriparesis
12	Paillas <i>et al</i> ²²	1963	53	M	20 d diplopia (sudden onset). (R) facial paraesthesias, (L) facial numbness and paresis, diplopia and loss of hearing on (L). 6 wk later, after initial improvement: sudden onset of headache and (R) hemiplegia	Nuchal rigidity. (L) V, VI, VII, VIII. (R) hemimotor, -sensory, cerebellar deficits (R) hemiplegia
13	Kempe ⁴⁸	1964	25	M	1 w (L) hearing loss and tinnitus. 2 d vomiting, vertigo and hiccoughing. 1 d lethargy	(L) Horner's syndrome (L) V, VIII, IX
14	Pouyanne <i>et al</i> ²⁴	1967	44	M	Several m paraparesis, later ataxia. 15 d diplopia. 2 d headache, difficulty swallowing	(R) VI, VII, IX, X. (R) hemisensory, cerebellar deficits
15	Pouyanne <i>et al</i> ²⁴	1967	25	F	1 m vertigo, (L) hearing loss and facial numbness. 3 d headache, (L) hemiparesis, diplopia, lethargy	Horizontal nystagmus. (L) V, VI, VII. (L) hemiparesis
16	Matson ⁴⁹	1968	?	?	Child. No history given	?
17	Koos <i>et al</i> ⁵⁰	1969	11	F	2 d headache, vertigo and vomiting. 1 d ataxia and diplopia 7 d later 1 d later	Horizontal nystagmus, bilateral MLF Headache, bilateral VI, quadriparesis. (L) III, (R) VII, inc. quadriparesis
18	Obrador <i>et al</i> ⁵¹	1970	10	M	5 d headache and vertigo (sudden onset)	Horizontal nystagmus, (L) III, VII. (L) cerebellar dysfunction
19	Abroms <i>et al</i> ²⁵	1971	15	F	3.5 wk episodic headache and vertigo 2 wk (R) hemisensory loss and decreased coordination. 1 wk diplopia	(L) VII, IX, X. (R) hemimotor, -sensory, deficits
20	Martin ⁵²	1972	32	M	3 m headache, diplopia, loss of taste, (L) arm numbness	Horizontal nystagmus, (R) III, VI, VII, VIII. Bilateral V. (L) hemimotor, -sensory, cerebellar
21	Murphy ³⁷	1973	39	M	Several d headache 1 d pain and numbness (R) face	Horizontal nystagmus. (L) VII, X. Eyes tonically deviated to (R). (R) hemisensory loss, (R) arm paresis. Late comatose
22	Scott <i>et al</i> ⁵³	1973	1.5	M	3 d progressive clunissiness. 1 d vomiting, lethargy, diplopia, paraparesis	(L) VI, VII. Bilateral IX, X
23	Arseni <i>et al</i> ⁵	1973	43	F	6 m episodic dizziness. 2 m headache, circumoral paraesthesias, episode of loss of consciousness. 2 wk recurrence of headache, paraesthesias, (+) diplopia. 1 d recurrent headache, diplopia and ataxia	Nuchal rigidity. Horizontal and vertical nystagmus. (R) cerebellar deficits. 7 d later (p improvement): (L) VII, IX, X, (L) hemimotor. (R) hemisensory, depressed consciousness

Table 1 (a) Continued

Case	Author	Year	Age (yr)	Sex	Clinical presentation	Physical examination
24	Arseni <i>et al</i> ⁵	1973	40	F	1 m headache, nausea and vomiting . . . improved. 2 w progressive recurrence and ataxia, dysphagia and somnolence	Nuchal rigidity, horizontal and rotatory nystagmus. (L) IX, X. (L) hemimotor, -sensory, cerebellar
25	Zeller <i>et al</i> ²³	1975	4	M	4 w (L) ptosis, progressive (R) facial paresis.	Vertical nystagmus. Bilateral MLF. (L) V, (R) VII. Bilateral IX, X
26	Papo <i>et al</i> ⁶	1976	18	F	3 m headache, ataxia. 4 d increased headache, vomiting	Nuchal rigidity, papilloedema. Horizontal nystagmus. (R) hemicerebellar deficits
27	Papo <i>et al</i> ⁶	1976	23	M	1 m diplopia (sudden onset)	(R) VI
28	Becker <i>et al</i> ⁵⁴	1978	55	F	1 d headache, vomiting, slurred speech. (R) hemiparesis	Vertical nystagmus. Bilateral V, VI. (L) V, IX, X. (R) hemiparesis.
29	Humphreys ⁵⁵	1978	10	M	6 d headache, later fever and vomiting	(R) VII, hemiparesis. Became comatose and decerebrate on the (R) hours after admission
30	La Torre <i>et al</i> ⁵⁶	1978	38	F	2 yr episodic headaches. 1 m increasing frequency of headaches, and vomiting	Drowsy. Nuchal rigidity, papilloedema. Bilateral VI. Upward gaze, palsy
31	Doczi <i>et al</i> ⁵⁷	1979	14	F	3 m headache. 2 m (R) hemiparaesthesia. 2 m (L) ptosis and diplopia	Drowsy. (L) Horner's syndrome, (L) VI. (L) hyperreflexia
32	Pau <i>et al</i> ⁵⁸	1979	10	F	2 yr before: episode of headache and vomiting. 1 m progressive headache, vomiting and drowsiness	Drowsy. Eyes deviated to the (R) (R) V, VII. (L) hemiparesis
33	Sundarsen <i>et al</i> ⁴³	1979	3	M	2 m progressive (R) hemiparesis and headache	(R) VII, XII. Hemiparesis.
34	Vaquero <i>et al</i> ⁵⁹	1980	32	M	1 yr progressive (R) hearing loss. 2 m (R) facial numbness and episodic headaches	Horizontal nystagmus. (R) V, VII, VIII. Ataxia
35	Vaquero <i>et al</i> ⁵⁹	1980	19	M	2 m (R) V, sensory VII. 10 d progressive ataxia, vertigo and vomiting	Nystagmus. (R) V, VII
36	Cioffi <i>et al</i> ⁶⁰	1981	53	M	10 d vomiting, headache, (R) facial pain, ataxia	(R) V, VII, VIII, IX, X. (R) hemimotor, -cerebellar deficits. Later became progressively more drowsy
37	Naito <i>et al</i> ⁴²	1980	36	F	?	?
38	Mattos Pimenta <i>et al</i> ⁶¹	1981	25	F	10 d neck pain (sudden onset). Later: diplopia and progressive (L) hemiparesis	Nuchal rigidity. (R) VI, (L) VII, IX, X. (L) hemiparesis, somnolent 2 d later
39	Mattos Pimenta <i>et al</i> ⁶¹	1981	25	F	3 d vomiting and dizziness	1 d later: dysphagia. 1 d later: (R) ptosis, (R) ophthalmoparesis, (L) hemisensory deficit
40	Pak <i>et al</i> ⁶²	1981	44	F	2 m (L) VI (sudden onset). 1 m (R) VI. 2 wk circumoral numbness	Bilateral MLF, VI, taste, IX, X. Truncal ataxia
41	Wong ³⁹	1982	13	M	5 d ataxia, diplopia, clumsiness of 1 d headache and vomiting	Horizontal and lateral nystagmus. (L) III, VII, VIII, IX, X. (R) VI. Severe ataxia. (L) hemimotor, -sensory deficits
42	O'Laoire ³⁸	1982	28	M	2 m progressive perioral tingling, diplopia, slurred speech, (R) sided numbness	Horizontal nystagmus. (L) VII, bilateral VI, dysarthria, (R) hemisensory deficit
43	O'Laoire ³⁸	1982	14	F	2 m progressive (R) facial tingling, ataxia, finally drowsiness and diplopia	(L) ptosis, VI, (R) V, nystagmus, drowsy. (L) hemicerebellar deficits
44	O'Laoire ³⁸	1982	15	F	36 h acute onset of headache and vomiting, slurred speech and drowsiness	Drowsy, bilateral VI, VII, ptosis. Ataxic
45	O'Laoire ³⁸	1982	26	F	1 d acute onset of (L) arm tingling; Later headache, vomiting and drowsiness	Drowsy, nuchal rigidity. Nystagmus, dysconjugate gaze, paresis, (L) hemisensory deficit, (R) cerebellar deficit
46	O'Laoire ³⁸	1982	26	M	2-5 wk stepwise course of mouth tingling; (L) sided tingling, slurred speech and diplopia	Vertical gaze paresis, nystagmus, (L) VII, ataxia, spastic gait
47	O'Laoire ³⁸	1982	53	M	18 m episode of nystagmus and diplopia. 2 m relapsing course of diplopia and ataxia, dysarthria, bilateral ptosis later drowsiness	Drowsy. Bilateral MLF, dysarthric, ataxic, spastic limbs
48	Beatty <i>et al</i> ⁶³	1983	23	M	1 yr incoordination. wks diplopia, dysarthria decreased penmanship	Nystagmus, diplopia, internuclear ophthalmoplegia, dysarthria, ataxia
49	Coffey <i>et al</i> ⁶⁴	1985	41	M	(R) pontine syndrome	(R) pontine syndrome
50	Bosch <i>et al</i> ⁶⁵	1985	25	F	2 d headache, vertigo, (R) sensorimotor dysfunction. 1 d bilateral facial palsy. (R) hemiplegia and horizontal gaze paralysis. Comatose during angiography	Dysarthria, bilateral VII, (R) hemiparesis
51	Tsutsui <i>et al</i> ⁶⁶	1986	27	F	4 wk diplopia. 2 wk (R) facial paresis, (L) sided numbness. 1 d decreased hearing O.D., ataxia, vertigo	(R) V, VI, VII, VIII, nystagmus. (L) hemisensory deficit

Table 1 (a) Continued

Case	Author	Year	Age (yr)	Sex	Clinical presentation	Physical examination
52	Case A	1985	10	M	5 d headache, (R) facial paresis.	(R) VI, VII, nystagmus.
53	Case B	1983	14	M	1 d recurrent headache, diplopia, ataxia 6 yr petit mal seizures. 5 yr (L) VI N paresis. Treated with R/T and chemo Rx. 1 yr (L) VI again, (+) (L) ptosis, progressive (R) hemiparesis, headache, vertigo, personality change	(R) hemicerebellar syndrome, ataxia (L) VI, VII palsies, bilateral ML. Truncal ataxia, (L) cerebellar deficits
54	Case C	1983	12	F	2 yr acute personality change, transient episodic "fainting spells." 2 m recurrent personality change. 6 wk unsteady gait, slurred speech. Later diplopia, vomiting, uncoordination	Agitated, (R) III, (L) VI, VII, slurred speech, truncal ataxia, (R) cerebellar
55	Case D	1984	42	F	Acute onset of dysarthria, numbness of (R) cheek, dysphagia, mild (R) hemiparesis	(L) facial palsy, (R) arm and leg dysmetria with decreased tone, mild (R) hemiparesis
56	Case E	1973	65	M	13 yr relapsing diplopia. 11 yr severe headache, bilateral ptosis, (L) VI, VIII. 9 yr progressive dysarthria, dysphagia, ataxia. 1 wk recurrent headache and disorientation	Bilateral ptosis, papilloedema. (L) VI, VII, VIII, bilateral IX, X. Impaired memory and orientation

Table 1 (b) Operation and result

Case	Operation	Time to operate	Follow-up	Results
1	Post fossa trephine and cerebellar puncture	?	—	Expired
2	Post fossa trephine and cerebellar puncture	12 hours	1 day	Expired
3	?	1 day	—	Expired
4	Suboccipital craniectomy	?	8 years	Expired
5	Parietal craniotomy	3 weeks	3 days	Expired
6	Suboccipital craniectomy	?	?	Good (?)
7	Suboccipital craniectomy	8 days	3 months	Improved, moderate deficit
8	Transtentorial approach	1 day	7 years	Improved, mild deficit
9	Torkildsen shunt	?	3 months	Improved, normal
10	Suboccipital craniectomy	4 months	4 years	Improved, mild deficit (expired later)
11	Ventriculo-atrial shunt	?	3 years	Expired
12	Suboccipital craniectomy	6 weeks	4 weeks	Improved, moderate deficit
13	Suboccipital craniectomy	6 weeks	6 months	Improved, normal
14	Suboccipital craniectomy	10 days	3-4 months	Improved, severe deficit
15	Suboccipital craniectomy	4 days	2 weeks	Improved, normal
16	Suboccipital craniectomy	?	?	Improved (?)
17	Suboccipital craniectomy	10 days	2 years	Improved, normal
18	Suboccipital craniectomy	1 day	1 year	Improved, mild deficit
19	Suboccipital craniectomy	3 weeks	3 years	Improved, moderate deficit
20	Suboccipital craniectomy	10 days	1 month	Improved, normal
21	Suboccipital craniectomy	14 hours	1 year	Improved, moderate deficit
22	Suboccipital craniectomy	6 days	6 months	Improved, moderate deficit
23	Suboccipital craniectomy	15 days	2 months	Improved, moderate deficit
24	?	3 days	1 month	Improved, mild deficit
25	Ventriculo-peritoneal shunt	15 days	6 weeks	Expired
26	Suboccipital craniectomy	6 days	1 year	Improved, mild deficit
27	Suboccipital craniectomy	7 weeks	1 week	Post-op clot on CT scan
28	Suboccipital craniectomy	12 hours	2 years	Improved, moderate deficit
29	Suboccipital craniectomy	1 day	7 months	Improved, mild deficit
30	Infratentorial supracerebellar approach	1 day	7 months	Improved, normal
31	Suboccipital craniectomy	1 week	1 week	Improved, normal
32	Suboccipital craniectomy	1 week	1 week	Improved, moderate deficit
33	Suboccipital craniectomy	1 week	1 month	Improved, mild deficit
34	Suboccipital craniectomy	1 week	3 years	Improved, normal
35	Suboccipital craniectomy	1 week	2 years	Improved, normal
36	Suboccipital craniectomy	1 week	1 year	Improved, moderate deficit
37	Suboccipital craniectomy	15 days	4-25 years	Improved, mild deficit
38	Suboccipital craniectomy	4 days	5 weeks	Improved, mild deficit
39	Suboccipital craniectomy	4 days	1 year	Improved, normal
40	Suboccipital craniectomy	1 week	6 months	Improved, mild deficit
41	Suboccipital craniectomy	1 week	6 months	Improved, mild deficit
42	Suboccipital craniectomy	?	9 months	Improved, normal
43	Suboccipital craniectomy	?	3 months	Improved, normal
44	Suboccipital craniectomy	?	2 years	Improved, normal
45	?	?	3 months	Improved, normal
46	Suboccipital craniectomy	?	3 months	Improved, mild deficit
47	Suboccipital craniectomy	?	3 weeks	Expired
48	Stereotactic aspiration	1 day	1 month	Improved, mild deficit

Table 1 (b) Continued

Case	Operation	Time to operate	Follow-up	Results
49	Suboccipital craniectomy	?	9 months	Improved, mild deficit
50	Stereotactic aspiration	?	6 months	Improved, moderate deficit
51	Suboccipital craniectomy	1 day	3 months	Improved, mild deficit
52	Suboccipital craniectomy	3 days	1 year	Improved, moderate deficit
53	Suboccipital craniectomy	7 weeks	4 years	?
54	Ventriculo-peritoneal Shunt	1 year	5 years	Expired
55	Suboccipital craniectomy	1 year	1 year	Unchanged, moderate deficit
56	Suboccipital craniectomy	1 day	1 year	Improved, normal

Fig 2 (a) Non-contrast CT scan discloses a blood density subependymal mass of the fourth ventricle on the right side, extending towards the brachium pontis (CASE A). (b) MRI of the same subependymal haematoma. Arrows point to two separate areas of haematomas (CASE A).

small haematomas in strategic locations (Cases 21, 6) may cause major clinical symptoms. However, the severity of bleeding most often correlates with the degree of brainstem compromise.

In those cases where the bleed is of sufficient force to cause autodestruction of the primary lesion, the process is self limiting. This explains why the subgroup of brainstem haematomas may do so well clinically, even with conservative management. In cases where the vascular malformation persists, recurrent bleeding may occur even after surgical evacuation of the haematoma (Case 22). Recurrent bleeding usually leads to more severe symptomatology.

In Teilmann's review of pontine haemangiomas,² 20% were asymptomatic during life and were incidental findings at necropsy. Thirty percent had no focal symptoms, but showed signs of diffuse cerebral dysfunction (dementia, personality changes, seizures, etc.) The remainder had focal symptoms suggestive of brainstem disease, with or without diffuse cerebral symptoms. In patients with brainstem findings, a characteristic pattern was encountered (usually in young persons and children) of acute or subacute episodes of pontine dysfunction, typically improving between episodes, but incompletely so. Often, a final common pathway of deterioration would be followed, either due to increased intracranial pressure (secondary to obstructive hydrocephalus), or recurrent haemorrhage. Zeller²³ described a similar picture in children.

Pouyanne *et al*²⁴ stressed the inexorably progressive course that some of these lesions take, mimicking brainstem tumours.

Arseni and Stanciu⁵ described a two-stage evolution of the pontine haematomas, marked first by an ictal onset, and followed by a relatively rapid progression of symptoms. They also noticed the occasional "pseudo-onset" which could precede ictal symptomatology by months to years. This was interpreted as representing the effect of the primary process (cryptic malformation with associated vascular phenomena within the tight quarters of the brainstem) rather than the formation of an early haematoma.

Occasionally the course is stereotyped, there being an ictus, partial remission and subsequent recurrence. Rebleeding from the primary cryptic malformation might explain such a course, as suggested by Abroms.²⁵ These cases have been misdiagnosed as multiple sclerosis on occasion.²⁶

Purely acute symptom courses occur more rarely and are usually related to sudden occlusion of the aqueduct with rapid neurological demise due to increased intracranial pressure and incipient tentorial herniation.

Some cases present only with the syndrome of obstructive hydrocephalus, undergoing shunting procedures alone with-

Fig 3 Sagittal MRI on same lesion as fig 2 (CASE A).

out suspected brainstem disease (Case 21, 6). Misdiagnosis is relatively common: tumour, multiple sclerosis, or brainstem infarction are most frequently suspected.

Diagnostic measures

Computed tomography^{27 28 29} may accurately help in the diagnosis of the brainstem haematoma. A high attenuation mass within the brainstem densitometrically consistent with blood, which does not enhance after contrast administration, is strongly suggestive of a brainstem haematoma, especially if subsequent studies show resolution of the density and persistent failure to enhance. A fluid : fluid interface may be seen within the haematoma (fig 5). On occasion, the computed tomogram may disclose some enhancement of the haematoma wall and it may be impossible to make the correct diagnosis preoperatively.²

Magnetic resonance imaging (MRI) accurately discloses location in sagittal view (fig 3). It may also show more than one haematoma, suggesting recurrent haemorrhage (fig 2b).

Lumbar puncture Bloody or xanthochromic fluid may be seen in cases where the haematoma has leaked through the ependymal floor of the fourth ventricle, suggesting a vascular rather than neoplastic aetiology. Frank bloody CSF may help distinguish hypertensive haemorrhage from subependymal haematoma. Of the 22 cases that underwent lumbar puncture in this series, 10 showed evidence of blood in the spinal fluid.

Cerebral posterior fossa angiography is not capable of providing adequate resolution to disclose cryptic malformations.^{30 31 15} Of the 30 patients who were studied angiographically in this series, no preoperative study was reported to have disclosed a vascular anomaly. The angiogram may show evidence of a pontine mass and may outline local vascular anatomy, but it does not differentiate between the haematoma and primary intra-axial tumour.

Results of surgical management (table 1)

The first case of brainstem haematoma to be approached surgically was that of Finkelnburg¹, who in 1903, explored a 14 year old boy with signs and symptoms of a posterior fossa obstructing mass lesion. In none of the first five cases^{1 12 33 34 35} was the diagnosis of brainstem haematoma

made operatively, and the lesions were subsequently found at necropsy. Bailey's case,³⁴ presented 8 years later by Cushing and Bailey,¹² is interesting in that the patient was felt to be suffering from a severe psychoneurotic disorder.

Dandy³⁶ was the first to make the correct operative diagnosis and to evacuate a brainstem haematoma successfully. Since that time, the results of surgical treatment for these lesions has been remarkably good (table 2). Only two of the 51 cases, since Dandy's case, became worse after operation, although five were transiently disabled or acquired temporary new post-surgical deficits. At the time of last observation, 14 patients (27%) were normal or nearly normal, 30 patients (58%) had mild to moderate neurological deficits but were carrying out their usual activities and/or were working, one patient had severe deficits and was bedridden. Four patients (8%) expired.

Timing of surgery

In cases where the clinical presentation is precipitous, early operation has been suggested.³⁷ Some authors^{5 22 24} have recommended timing surgery from 2 weeks to 1 month after the onset of the patient's most recent ictus, so that time may be allowed for local oedema to resolve and for delimitation of the haematoma by local reactive processes.⁵ The time from admission to operation in this series is listed in table 1.

Conservative versus surgical therapy (table 2)

Numerous authors have recommended conservative management for brainstem haematomas, as some patients may follow a relatively benign course. The best case in point would be that of the discrete haematoma which has destroyed the primary malformation from whence it arose. A review of reported brainstem haematomas which have been managed conservatively is summarised in table 3. Comparison between the conservatively and surgically managed cases discloses an apparent, although not statistically significant, difference in outcome. Eighty-five percent of the surgical cases were normal or had mild-moderate neurological deficits, while only 30% of the conservatively managed group had similar results.

Conservatively managed cases rebleed with an unacceptably high frequency. When managed conservatively

Fig 4 Operative photographs of brainstem haematoma (CASE B): (a) Prior to evacuation of haematoma, which elevated the floor of fourth ventricle on the left side displacing the median raphe. (b) After removal of haematoma, the cavity is seen after incision to the floor of the fourth ventricle.

Fig 5 Non-contrast CT disclosing a fluid:fluid level in the pons. This lesion did not enhance with contrast.

for prolonged periods, patients eventually deteriorate (as did our Case A).⁴¹⁻⁴³

At surgery, residual vascular malformation may be visualised under high-power magnification (we have observed three), and removed. If complete, such removal should obviate future rebleeding.^{38 39}

Alternatively, surgical removal of the clot does not always preclude rebleeding, even when an obvious vascular malformation is seen in the wall of the clot with the operating microscope. This occurs because incision over the brainstem haematoma may provide only limited exposure, precluding adequate visualisation of the clot cavity and excision of the

entire malformation (fig 4).

Conclusions

Subependymal brainstem haematomas are focal lesions which arise from congenital vascular malformations of the cryptic type within the closely packed confines of the brainstem.

When the history discloses a characteristic pattern of ictus, partial remission and later progression of neurological signs, and there is computed tomo-

Table 2 Comparison between conservatively and surgically managed cases

Conservative management		Surgical management	
33 Cases		1905-1986-56 Cases 1932 (Dandy)-1986-51 Cases	
Follow-up results		Follow-up results	
Average-11 months		Average-14 months	
Range-2 days-5 years		Range-1 week-8 years	
5	Normal (15%)	14	Normal (27%)
5	Mild-mod. deficit (15%)	30	Mild-mod deficit (58%)
6	Severe deficit (18%)	1	Severe deficit (2%)
11	Expired (33%)	4	Expired (8%)
6	Not evaluated (18%)	2	Not evaluated (4%)
33	Total	51	Total

Table 3 Conservative management of brainstem haematomas

Authors	Year	Age (yr)	Time of follow-up	Result
Grotts ⁶⁷	1950	2	2D	Expired
Teilmann ²	1953	36	4Y	Expired
Teilmann ²	1953	47	3Y	Expired
Zeller ²³	1968	10	9D	Expired
Zeller ²³	1968	18	3-5Y	Expired
Zeller ²³	1968	4	2M	Expired
Burns <i>et al</i> ⁶⁸	1980	58	37D	Severe deficit
Payne <i>et al</i> ⁶⁹	1978	?	1M	Mild deficit
Cioffi <i>et al</i> ⁶⁰	1980	43	2Y	Mild deficit
Cioffi <i>et al</i> ⁶⁰	1980	55	7M	Severe deficit
Kase <i>et al</i> ⁶⁹	1980	47	52D	Severe deficit
Kase <i>et al</i> ⁶⁹	1980	42	48D	Severe deficit
Zuccarello <i>et al</i> ⁴⁰	1980	48	3M	"Improved"
Zuccarello <i>et al</i> ⁴⁰	1980	10	2M	Normal
Zuccarello <i>et al</i> ⁴⁰	1980	54	2M	Normal
Zuccarello <i>et al</i> ⁴⁰	1980	57	2W	Normal
Zuccarello <i>et al</i> ⁴⁰	1980	53	2M	Normal
Caplan <i>et al</i> ²¹	1982	81	1M	Expired
Caplan <i>et al</i> ²¹	1982	58	3W	Expired
Caplan <i>et al</i> ²¹	1982	63	18M	Mod-severe deficit
Albright <i>et al</i> ⁴¹	1982	1-5	5M	Expired
O'Laioire <i>et al</i> ³⁸	1983	19	5Y	Severe deficit
Gonzalez <i>et al</i> ⁷⁰	1983			9 patients with discrete lesions: 2 expired 1 normal Others "improved"
Toyama <i>et al</i> ²⁶	1978	16	?	Mild deficit
Sako <i>et al</i> ⁷²	1979	14	7W	Mild deficit

graphic evidence of a discrete intra-axial lesion of blood density which does not enhance, the diagnosis of brainstem haematoma may be accurately made.

It appears from available evidence that these lesions probably fare better with surgical than with conservative management.

The surgical goals in the treatment of these lesions consist of (1) removal of the compressive lesion from within the brainstem, allowing for rapid return of neurological function, and (2) the obliteration of residual vascular malformation when observed, to prevent future rebleeding.

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Addendum: Since submitting this paper, four more patients have been operated on for brainstem haematoma, with similar results to those of the first five patients.

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