conclude that there was an increased protein exudation into the bowel in Cases 1 and 3 (see Table).[1]

The normal values for faecal excretion of ^{131}I and ^{51}Cr quoted in the Table agree with those reported by Gordon (1959) and Schwartz and Jarnum (1959) for ^{131}I-P.V.P. and by Rubini and Sheehy (1961) and Rubini *et al.* (1961) for $^{51}CrCl_3$. However, in the former two reports faecal recovery of ^{131}I-P.V.P. in control subjects was occasionally as high as 1.5% and 1.03% respectively, while in the study by Rubini and Sheehy (1961) faecal recovery of ^{51}Cr was up to 1%. The excretion of 1.3% of the injected isotope in Case 4 cannot therefore be regarded as abnormal. However, the excretion of 2.4% of the administered dose in Case 5 may well be abnormally high, although the serum albumin level was normal. In this patient albumin production had presumably increased enough to prevent hypoalbuminaemia, as in some of the patients studied by Jeejeebhoy (1964).

It is very unlikely that loss of blood into the bowel entirely accounted for the recovery of isotope in the faeces. Bleeding did not occur in Case 1 during the ^{131}I-P.V.P. test, daily tests for occult blood in the stools being consistently negative. Hypoalbuminaemia was present in Case 2 before gastrointestinal bleeding was detected. No bleeding occurred in Case 5 but the faecal recovery of isotope was elevated. Moreover, far larger haemorrhages due to diseases such as peptic ulcer do not usually produce hypoalbuminaemia.

Although these results indicate that protein-losing enteropathy contributed to the hypoalbuminaemia in Cases 1–3, two other possible factors have not been excluded. Albumin production may have been reduced in these patients. None had clinical evidence of liver disease, and liver-function tests in Case 3 gave normal results. Jeejeebhoy (1964) has suggested that in certain gastrointestinal diseases excessive faecal losses of nitrogen may eventually limit albumin synthesis. It seems unlikely that this mechanism was important in these patients, whose gastrointestinal disturbances were of short duration before hypoalbuminaemia was detected. Finally, it is conceivable that an abnormal distribution of albumin between the intravascular and extravascular compartments was present, as described by Weinbren *et al.* (1965), in one patient with systemic lupus erythematosus.

The fact that gastrointestinal symptoms were most marked in those patients with hypoalbuminaemia (see Table) lends support to the view that this deficiency was due to abnormal protein losses into the gut. Such losses are presumably associated with the gut lesions described in anaphylactoid purpura, which include thickening of the bowel wall with localized or extensive haemorrhages (Bailey, 1930 ; Bywaters *et al.*, 1957). It is of interest to note that in five of the cases described by Bywaters *et al.* (1957) oedema was the presenting symptom, whereas only two patients had signs of renal involvement at the onset of the disease, and both these had previously suffered from nephritis. It seems possible that when oedema antedated renal disease in the latter series hypoalbuminaemia due to protein-losing enteropathy was the cause.

[1] The validity of $^{51}CrCl_3$ and ^{125}I-P.V.P. in the qualitative assessment of gastrointestinal protein loss is further supported by a recent study (Rootwelt, K., *Scand. J. clin. Lab. Invest.*, 1966, **18**, 405).

Summary

Hypoalbuminaemia may occur in anaphylactoid purpura when urinary protein losses are trivial.

Evidence is presented to suggest that excessive protein losses into the gut may contribute to the hypoalbuminaemia.

We are grateful to the physicians of St. Thomas's Hospital who allowed us to study patients under their care.

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Non-tumoral Stenosis of the Aqueduct in Adults

TUSHAR K. NAG,* F.R.C.S.ED., M.R.C.P.ED. ; MURRAY A. FALCONER,† M.CH., F.R.C.S., F.R.A.C.S.

Brit. med. J., 1966, **2**, 1168–1170

Though non-tumoral stenosis of the aqueduct has long been described as a common cause of hydrocephalus in infants and young children, its occurrence in adults has been recorded infrequently. In childhood the general picture of the clinical features (Dandy, 1920, 1945 ; Pennybacker, 1940 ; Torkildsen, 1947, 1948, 1960) is of an infant or child whose head is steadily enlarging from hydrocephalus, who is overweight and usually slow at school, and who has developed increasing headache with slight clumsiness of movements, together with a low-grade papilloedema. Plain *x*-ray films of the skull show, in addition to enlargement of the skull vault, pressure changes in the dorsum sellae and a shallow posterior fossa. The diagnosis is confirmed by ventriculography with or without lumbar encephalography. Cases of aqueduct stenosis in adults have been described (Spiller and Allen, 1907 ; Petit-Dutaillis *et al.*, 1950 ; Paine and McKissock, 1955). These authors, however, did not delineate any specific syndromes or patterns of symptom-presentation in adults. Our own findings suggest that certain patterns do occur which are variants of the presentation of chronic hydrocephalus in adults from other causes. Among these patterns are the presentation of impaired memory, of epilepsy, of unsteady gait, of headaches and other features of increased intracranial tension, and of endocrine disorders and features indicative of hypothalamic involvement. Below we report a series of 10 cases in patients over the age of 25 years, in whom this lesion seemed responsible for symptoms.

* Registrar, Guy's-Maudsley Neurosurgical Unit, London.
† Director, Guy's-Maudsley Neurosugical Unit, London.

The Patients

The salient features of our 10 cases are listed in Table I. In only two (Cases 9 and 10) did the symptoms date from childhood. The duration of symptoms varied from 1 to 39

years, the average being 16 years. In all cases the diagnosis was based upon radiological findings, principally on those of ventriculography and/or lumbar encephalography (Table II). The principal points noted in the straight *x*-ray films of the skull are: first, that, although the head was enlarged in four cases, the posterior fossa was relatively shallow in six; secondly, erosive changes affecting the dorsum sellae indicative of raised intracranial pressure were present in all but two cases; and, thirdly, the calvarium was unduly thin in two cases. Ventriculography with air was performed in all cases, and in one a positive contrast medium was also used. All 10 showed symmetrical enlargements of the lateral ventricles with enlargement of the third ventricle. In three cases air passed through a very narrow aqueduct into the fourth ventricle, which was of normal size, the aqueduct itself not being displaced. Only the orifice of the aqueduct at the posterior end of the third ventricle could be visualized in five cases, while in the remaining two it was not seen. To supplement these findings lumbar encephalography was then performed in four cases, and in three of them the air filled a normal but small fourth ventricle without passing upwards into the aqueduct. The diagnosis of aqueduct stenosis was then substantiated by superimposing the findings of the lumbar encephalogram on those of the ventriculogram.

Aetiological Factors

Possible aetiological factors were sought in all cases. In three (Cases 1, 2, and 4) there had been an antecedent history of meningitis, 5, 7, and 13 years, respectively, prior to the onset of symptoms. All cases seemed to have had a non-traumatic birth (Case 8 had a caesarean birth) and no consanguinity was noted in the parents. The maternal age and the pregnancy order did not seem significant. None of the cases had clinical or radiological evidence of spina bifida. No other aetiological factors were forthcoming. The assumption is that, apart from the three cases which had had meningitis, the stricture of the aqueduct was probably of developmental origin. There was no evidence in any case that it was neoplastic.

The symptoms and signs shown by our patients are those of chronic hydrocephalus appearing in adults, and varying in their pattern of presentation from one patient to another. As regards symptomatology, four modes of presentation could be made out: (1) cases presenting with impaired memory and intellectual deterioration (Cases 1, 2, 3, and 8); (2) cases presenting with epilepsy, usually of psychomotor type (Cases 2, 3, 7, and 9); (3) cases presenting with unsteady gait (Cases 4, 8, and 10); and (4) cases presenting with headache indicative of raised intracranial pressure (Cases 1, 4, 5, 6, and 9).

A fifth mode of presentation reported in the literature (Parker and Kernohan, 1933; Paine and McKissock, 1955) but not exemplified in our series is that of a female patient with hypothalamic symptoms, such as amenorrhoea, due to third ventricular distension. It is possible that in patients with such a presentation menstrual periods may return after a successful surgical intervention, for in a case of non-tumoral aqueduct stenosis in an 18-year-old girl, not included in our series, who had never menstruated, the menses appeared a year later after ventriculocisternostomy.

As regards signs, seven showed objective evidence of mental impairment, although only four complained of this. Five showed low-grade papilloedema. Two (Cases 7 and 8) had a slight unilateral sixth-cranial-nerve weakness, while the pupillary reactions to light in these two cases were absent. One (Case 8) showed limited upward gaze of the eyeballs. Five of the patients showed mild bilateral ataxic features or disequilibrium of gait; while four showed pyramidal signs, which were unilateral in only one. Four of them had an enlarged head (more than 60 cm. in circumference).

Illustrative Cases

The following three cases illustrate the differing modes of presentation of aqueduct stenosis in adults.

Case 2.—A labourer aged 30 was admitted with a history of impaired memory of recent events, irritability, outbursts of temper, and psychomotor epileptic seizures dating back for one year. Apart

TABLE I.—*Salient Clinical Features*

Case No.	Age and Sex	Age at Onset of Symptoms	Presenting Symptoms					Principal Signs				Head Circumference, above 60 cm.	Abnormal E.E.G.
			Impaired Memory	Epilepsy	Unsteady Gait	Headache	Others	Mental Impairment	Papilloedema	Ataxia, Dysequilibrium	Pyramidal Signs		
1	26 M	19	Present			Present	Dragging left foot	Present	Present	Present	Present	60·5	Temporal spikes
2	30 M	29	,,	Psychomotor				,,					,,
3	56 M	47	,,	Grand mal				,,					Abnormal
4	31 M	29			Present	Present				Present	Present		Not performed
5	46 M	21				,,	Vertigo	Present	Present	,,		61·5	,, ,,
6	38 F	36				,,		,,					,, ,,
7	37 M	20		Psychomotor				Present	,,			63.5	Temporal spikes
8	60 M	32	Present		Present			,,	,,	Present	Present		Not performed
9	45 M	Childhood		Psychomotor		Present	Vertigo						Normal
10	46 M	7			Present			Present		Present	Present	65	Not performed
Total			4	4	3	5		7	5	5	4	4	4

TABLE II.—*Radiological Findings*

Case No.	Plain Skull *X*-ray			Ventriculogram			Air Encephalogram	
	Skull Vault	Sellar Pressure Changes	Posterior Fossa	Ventricular Dilatation	Aqueduct	Fourth Ventricle	Fourth Ventricle	Aqueduct
1	Thin	Present	Shallow	Gross	Proximal part narrowed	Normal	Not performed	Not performed
2	Normal	,,	,,	Moderate	Only orifice seen	Not visualized	,, ,,	,, ,,
3	,,	,,	,,	,,	Narrowed	Normal	,, ,,	,, ,,
4	,,	,,	,,	,,	,,	,,	,, ,,	,, ,,
5	Thin	,,	,,	Gross	Not visualized	Not visualized	Not visualized	Not visualized
6	Normal	,,	Normal	,,	Only orifice seen	,, ,,	Normal	,, ,,
7	Thin		,,	Gross with diverticulum	Not visualized	,, ,,	Not performed	Not performed
8	Osteoporotic	Present	,,	Gross	Only orifice seen	,, ,,	,, ,,	,, ,,
9	Normal		,,	Moderate	,, ,, ,,	,, ,,	Normal	Not visualized
10	Thin	Present	Shallow	Gross	Only upper 3 mm. seen	,, ,,		,, ,,

from a history of meningitis eight years previously, he had had no serious illnesses and his birth had been normal. His wife confirmed that he had difficulty in remembering ordinary everyday events, while it was noted that his replies to a simple questionary were defective. His seizures began with an aura of *déjà vu* associated with a brief absence or short automatism. Neurological examination was negative. Electroencephalography showed bilateral spike discharges over the temporal lobes. Bilateral ventriculocisternostomy was performed. This resulted in subjective and objective improvement of recent memory but had no effect on his seizures during the follow-up period of 10½ years.

Case 5.—A labourer aged 46 was admitted with a history of headache dating back 25 years. These headaches were frontal and used to appear shortly after waking in the morning. Physical development was normal, but his mental development had been retarded (I.Q. 54). The optic discs were blurred. He had moderate ataxia in his limbs and an unsteady gait. The head circumference was 61.5 cm. (24¼ in.). After the operation he has been followed up for six and a half years, and some improvement has occurred in his mental functions. Although he is still working at his former job, his reading and other abilities have improved, while his papilloedema has subsided. His headaches have also improved.

Case 10.—A postal sorter aged 46 had had an unsteady gait since the age of 7 years. His schooling had been normal but he had always been clumsy at games. His head circumference was 65 cm. (25½ in.). In addition to ataxia of his limbs and disequilibrium of gait, he presented increased spasticity of the lower limbs with bilateral extensor plantar responses. Since ventriculocisternostomy he had been followed up for three and a half years. His gait was steady and there were no pyramidal signs. His work performance has improved.

TABLE III.—*Results of Treatment*

Case No.	Follow-up Period (years)	Overall Results	Working Ability	Mental Functions	Epilepsy
1	10½	Improved	Improved	Improved	
2	10¼	,,	Unchanged	,,	Persists
3	7	Unchanged	,,	No improvement	,,
4	Untraced	Slight immediate improvement	Not known		
5	6½	Improved	Unchanged	Improved	
6	5	,,	Improved		
7	4	,,	Unchanged	Slightly improved	Persists
8	3	,,	Improved	Improved	
9	3½	,,	Unchanged		Persists
10	3½	,,	Improved	Improved	

Discussion

The pathological features of non-tumoral aqueduct stenosis have been exhaustively described by Russell (1949) and were reviewed by Alvord (1961). Beckett *et al.* (1950) also made a series of necropsy studies, mainly in adult cases. Russell classified aqueduct stenosis as follows: (1) simple stenosis, in which the aqueduct is histologically normal but abnormally narrow: (2) forking or atresia, in which the aqueduct is represented by two or more narrow distinct channels lined by intact ependymal epithelium and separated by normal nervous tissue without any gliosis; (3) cases with a neuroglial septum obstructing the hinder part of the aqueduct; and (4) cases of gliosis of the periaqueductal tissues resulting in stenosis of the aqueduct. She considered that simple stenosis or forking was caused by malformation, while the septum formation was due either to malformation or to inflammation. The cause of periaqueductal gliosis is a matter of controversy, and inflammation, maldevelopment, and neoplastic lesions have each been postulated.

All cases in this series were treated by ventriculocisternostomy. Most authors nowadays seem to prefer this operation (Paine and McKissock, 1955). Other procedures, however, have had their advocates, including third-ventriculostomy (Krayenbühl *et al.*, 1950; Scarff, 1963) and even decompression of the posterior fossa (Pennybacker, 1940). Petit-Dutaillis *et al.* (1950) employed either third-ventriculostomy or ventriculocisternostomy, preferring the latter operation when the hydrocephalus was marked. Our results seem to suggest that Torkildsen's ventriculocisternostomy is superior to other procedures in this condition. From the follow-up studies it would appear that most of the symptoms and signs can be improved by this operation (Table III). It would also appear that, although the other features are improved, epilepsy remains unchanged.

Summary

Reports are given of 10 cases of non-tumoral stenosis of the aqueduct causing symptoms and signs in patients over the age of 25 years. There were five patterns of clinical presentation: impaired memory and intellectual deterioration; epilepsy, usually of psychomotor type; unsteady gait; headaches and other features of increased intracranial tension; and endocrine disorder. The follow-up studies indicated that most of the symptoms and signs could be improved by ventriculocisternostomy, though, if epilepsy was present, it persisted despite relief of the hydrocephalus.

Our thanks are due to Mr. P. H. Schuur, who kindly allowed us to include cases under his care, and to Dr. R. D. Hoare for the radiological studies.

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