TABLE V

	Grade A	Grade B	Grade C	Grade D
Genital cancer	1 (1.5%)	25 (37.3%)	25 (37.3%)	16 (23.9%)
No genital cancer	8 (3.4%)	132 (56.4%)	53 (22.6%)	4 (1.7%)

was not a source of bias, for the average age of the patients with genital cancer was 57.0 years and that of the other women 56.1 years.

Summary

An investigation has been made into the post-menopausal production of oestrogen on a series of 353 women.

The production of oestrogen was found to be high, 53% of women showing evidence of considerable oestrogenic activity and only 20% showing none at all.

The oestrogen production was not affected by the manner in which the menopause was achieved.

Neither the ovary nor the adrenal was found to be a significant source of oestrogen after the menopause.

Oestrogen did not appear to have an important part to play in either breast or female genital tract cancer.

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CEREBELLAR SOFTENING

A SURGICAL EMERGENCY

BY

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Although cerebral haemorrhage is so common cerebellar haemorrhage is rare; Courville (1950) found only 117 examples in 1,484 necropsies of patients dying from spontaneous haemorrhage in the brain (less than 8%). Despite the rapidly fatal nature of cerebellar haemorrhage (Mitchell and Angrist, 1942) a few patients have been saved by timely surgery (Guillaume *et al.*, 1949; Dickmann and Zimmann, 1949).

Softening of the cerebellum is even rarer than haemorrhage. Thus Grinker and Bucy (1949) state that "because of the many anastomoses, softenings of the cerebellum are almost pathological curiosities"; and search of the literature has disclosed only three references to the condition (Germain and Morvan, 1938; Fattovich and Lenti, 1940; Fattovich, 1950). Hitherto, diagnosis has been made at necropsy and no well-defined syndrome has been established. No reference to

posterior fossa decompression having been carried out for this lesion has been discovered.

In this article, records are presented of three patients whose symptoms and signs indicated a space-occupying lesion, and evidence is given that softening and swelling of one cerebellar hemisphere—probably due to vascular occlusion—was the cause in each case.

Case 1

The patient was a man aged 40. On April 23, 1953, while at work he felt faint and vomited several times. He did not lose consciousness, but felt so ill that he had to be sent home to bed. He continued to vomit, and was therefore admitted to hospital on April 25, when he was drowsy and had signs of meningeal irritation. Lumbar puncture showed a raised pressure (reading not recorded). The cerebrospinal fluid contained 60 mg. of protein per 100 ml. His condition deteriorated during the next two days. He became comatose and developed Cheyne-Stokes respiration and transient Babinski responses. Another lumbar puncture revealed turbid fluid containing 260 white cells per c.mm. (89% polymorphs) and 80 mg. of protein per 100 ml. The fluid was sterile. He gradually regained consciousness and was transferred to us on May 1, nine days after the onset of the first symptoms. On examination he was found to be fully conscious but could not remember the first three days of his illness. His only complaint was of mild headache and pain in the neck. A complete clinical examination showed no abnormal physical signs apart from a little neck stiffness. Radiography of the skull and chest and examination of the blood showed nothing abnormal.

Ventriculography (May 19) was performed with air and ethyl iodophenylundecylate. The ventricular pressure was 60 mm. There was moderate dilatation of all the ventricles and also a small irregular filling defect in the fourth ventricle on the left side.

The posterior fossa was explored on the same day. Both cerebellar tonsils were herniated through the foramen magnum. The right tonsil descended further than the left, reaching the lower border of the axis. It was very shrunken, frayed, and yellowish, and was adherent to the medulla oblongata. The dura mater was adherent to the right cerebellar hemisphere, which was also very shrunken and soft. An incision into it revealed yellowish diffuent tissue. Retraction of the right cerebellar lobe and tonsil showed atheromatous occlusion of the peripheral part of the posterior inferior cerebellar artery. The left side of the cerebellum was of normal appearance and consistency, and there was no atheroma of the left posterior cerebellar artery. The cerebello-pontine angle was clear on both sides and a fine catheter was passed upward into the fourth ventricle through the foramen of Magendie without obstruction. The small fragments of cerebellum taken from both hemispheres showed no histological evidence of tumour. It was concluded that infarction of the right cerebellar hemisphere had occurred from atheromatous occlusion of the distal part of the posterior inferior cerebellar artery (occlusion of the proximal part would have produced the classical syndrome of infarction of the medulla oblongata). The patient made a rapid and complete recovery and when last examined on January 11, 1956, was symptom-free and showed no abnormal physical signs.

Case 2

A man aged 69 was admitted to hospital on May 29, 1954, having the day before had an attack of giddiness and sweating followed by frontal headache and repeated vomiting. During the next 24 hours the headache became worse and was also felt in the occipital region. The vomiting continued. He had had two similar attacks 12 and 9 months previously, when repeated vomiting with slight headache had occurred.

A complete physical examination showed brisk tendon jerks, bilateral positive Babinski responses, and retention of urine (the bladder was distended up to the level of the umbilicus); the pulse rate was 60 a minute.

Examination of the blood and radiography of the skull and chest were normal. The cerebrospinal fluid was at a pressure of 160 mm. and contained 40 mg. of protein per 100 ml., and 4 polymorphs and 3 lymphocytes per c.mm. On June 3 air and ethyl-iodophenylundecylate ventriculography was carried out. The ventricular pressure was atmospheric. There was symmetrical dilatation of the lateral ventricles. The third ventricle and aqueduct were dilated and the fourth ventricle was displaced to the right.

Exploration of the posterior fossa the same day revealed a very swollen left cerebellar lobe with widened folia and tonsillar herniation down to the lower border of the atlas. The lower half of this lobe was smooth and yellowish. This part was incised, exposing soft necrotic tissue which involved most of the lower half of the left cerebellar lobe, including the tonsil. This tissue was aspirated with the sucker, leaving a cavity about 1½ in. (3.8 cm.) in diameter. Cerebrospinal fluid then flowed freely from the foramen of Magendie. Immediately after the operation headache and vomiting ceased.

Examination of tissue removed at operation showed appearances suggestive of early softening, although the sections were too fragmentary for a dogmatic report. The Purkinje cells were eosinophilic, and scattered polymorphs and some macrophages were present. Vascular congestion and some extravasation of blood were found. There was no evidence of tumour.

The patient made a complete recovery from the intracranial lesion but died from coronary thrombosis on October 11, while in a convalescent home.

Case 3

A man aged 56 was admitted to hospital with a three-weeks history of aching and stiffness in the left occipital region. He was then complaining of severe generalized headache radiating to the vertex, worse on the left than the right, and associated with vertigo (surrounding objects rotating to the right) and photophobia.

A complete physical examination showed nothing abnormal except for a slight rise of blood pressure (170/95). Radiography of the skull and chest was negative. Three days after admission the headache became associated with vomiting. He then gradually improved until May 24, nine days after admission, when he again had a severe headache with nausea and vomiting. He also had diplopia. The next day he was found to have paralysis of the left third, fourth, and seventh cranial nerves.

We were asked to see him on May 26, two days later. He was then complaining of severe generalized headache and pain in the left side of the face, shoulder, and arm ("as though it were on fire").

On examination the following abnormal physical signs were found: slight bilateral papilloedema; dilatation of the right pupil with retained pupillary reflexes; coarse nystagmus to the right (not to the left); impaired convergence; paralysis of the left abducens nerve; diminished left corneal reflex; hypalgesia of the left trigeminal area; marked left facial weakness (lower neurone type); hypotonia of the left upper and lower limbs; and ataxia of the left limbs. There was no neck stiffness, and the plantar reflexes were normal. The diagnosis of posterior fossa tumour was made. Ventriculography and operation were arranged for the following day, but the patient died suddenly during the night.

At necropsy the left cerebellar hemisphere was found to be very swollen and soft. Partial disintegration of both grey and white matter had occurred. There was coning of the corresponding cerebellar tonsil.

Histology.—"The section consists of a segment of cerebellum showing an irregular infarct affecting both the grey and the white matter. The lesion is patchy and of varying severity in different areas. In some patches brain is completely replaced by collections of compound granular corpuscles with swollen granule-containing cytoplasm. In regions of incomplete destruction the white matter is of a foamy appearance and pale colour, the molecular layer oedematous, and both are infiltrated by cerebral phagocytes which are often of a perivascular distribution. In areas of still less damage there are infiltrating polymorphs and scanty cerebral phagocytes. The Purkinje cells and cells of the molecular layer are similarly affected, with absence in the severely damaged portions and early signs of degeneration in those portions least affected. The capillaries are prominent and lined by swollen endothelial cells. The vessels are congested and there is some extravasation around the thin-walled veins. No thrombus is seen. The lesion is about two to three weeks old, and is of ischaemic origin—due to either thrombosis or vascular spasm. There is no evidence of tumour."

Discussion

All three patients had clinical evidence of acute compression of the posterior fossa, and ventriculography indicated a space-occupying lesion in the two patients on whom it was carried out. One patient died before ventriculography could be done. The first patient was operated on when the acute phase had passed, and is therefore regarded as having recovered spontaneously, although he had nearly died (at one stage he was in coma and there was Cheyne-Stokes breathing). The second patient was operated on during the acute phase when there was marked cerebellar swelling. Although medullary compression was immediately relieved, it is not possible to say definitely what the outcome would have been without decompression. However, the third patient died from medullary failure while awaiting operation. The danger to the vital centres of the medulla oblongata from a sudden rise of pressure within the rigid boundaries of the posterior fossa is well known.

Cerebral swelling following cerebral arterial occlusion is a recognized phenomenon (King, 1951; Clarke *et al.*, 1955). Therefore, in the absence of a tumour, there is good reason for attributing acute swelling of a cerebellar hemisphere to vascular occlusion. This explanation is suggested by the finding of atheromatous occlusion of the peripheral part of the posterior inferior cerebellar artery in Case 1 and by the histological evidence of infarction in Case 3.

Summary

Three examples of cerebellar softening causing sudden compression of the brain stem are described. Vascular occlusion was the probable cause. Immediate decompression of the posterior fossa is the treatment advised.

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