persisted for some days after the beginning of vitamin-C administration. In the remaining three of the nine patients the donor red-cell survival times appeared to be normal, but two of these cases could not be studied adequately, and even the one who was adequately followed showed a rapid fall in the number of transfused red cells one week after vitamin-C therapy was started. It is possible that the blood transfusion materially increased metabolic demands for vitamin C, and this factor may have contributed to the rapid destruction of donor cells. With this reservation, the results of this investigation support the view that a haemolytic process is an important factor in the pathogenesis of the anaemia of scurvy.

ADDENDUM.—Since this paper was prepared an additional nine Bantu male patients with scurvy and normoblastic bone marrow have been studied by the same methods. Three of these, who were severely anaemic, were transfused with sedimented red cells from 1 pint of blood. All three patients eliminated the transfused red cells very rapidly. In the remaining six patients 1 pint of blood was first removed; subsequently they were transfused with 1 pint of blood. Red-cell survival was greatly decreased in five of these patients, the sixth patient having a normal red-cell survival time. The sixth patient (and one of the five with diminished red-cell survival) was not anaemic. The other four were anaemic. It appears that the proviso inserted in the main body of the paper that overtransfusion might be playing some part in the abnormal red-cell survival in untreated scurvy is unnecessary. Red-cell survival was abnormal even when a replacement transfusion was substituted for simple transfusion. An increase in the protein content of the diet by the addition of thoroughly stewed meat also did not alter the diminished red-cell survival in the last four patients studied. In the combined series there were thus 14 anaemic patients and 2 non-anaemic patients who were maintained on the vitamin-C-free diet for more than seven days. Of the anaemic cases 13 had greatly diminished red-cell survival. Of the non-anaemic cases, red-cell survival was normal in one and diminished in the other.

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The Association of Universities of the British Commonwealth has published a useful list of postgraduate fellowships, scholarships, grants, etc., for advanced study and research, tenable at universities in the United Kingdom, together with details of some of the more important awards tenable overseas by British graduates. The list is published by the Association at 5, Gordon Square, London, W.C.1. Academic interchange between the universities of Britain and those of other European countries has been sponsored by the British Council for several years. The Council has now published a pamphlet (*University Interchange between the United Kingdom and other European Countries, 1948–1953*) listing all such interchanges.

CEREBELLAR NECROSIS IN A CASE OF SUBARACHNOID HAEMORRHAGE

BY

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The possible dangers from lumbar puncture to patients with increased intracranial tension are well known. The main one is forcible impaction—"coning"—of the brain-stem and cerebellum into the foramen magnum, following excessive and rapid drainage of cerebrospinal fluid from the lumbar theca. This may or may not be accompanied by uncalled herniation through the tentorium cerebelli. These risks are mentioned only briefly in the standard neurological textbooks, but their mechanisms have received some close attention in the literature (Jefferson, 1938; Cairns, 1939).

A less frequently cited complication is the precipitation of cerebral haemorrhage, and Schube and Raskin (1936) mention 14 examples occurring in the literature. Two of these were cases of cerebral aneurysm. Androp (1939) reports a case in which death occurred, following lumbar puncture, from haemorrhage into the spinal cord and brain-stem.

Necrosis of the cerebellar tonsils was present in the following case of subarachnoid haemorrhage due to rupture of a cerebral aneurysm. Lumbar puncture was performed three times in the course of the illness. This necrotic lesion is considered to have resulted from an extreme and possibly prolonged degree of coning of the cerebellum. So far as I am aware, such a lesion has not been described before.

Case History

A 41-year-old housewife was admitted in an unconscious state to a country hospital in Northern Ireland on November 14, 1951. She had complained of headache since November 9, and had collapsed on the 13th. On examination there was pronounced neck stiffness, and a subhyaloid haemorrhage was present below the right optic disk. At lumbar puncture the fluid was blood-stained and under obviously greatly increased pressure. The supernatant fluid was xanthochromic.

The patient's condition improved somewhat until November 17; she was more conscious, but now complained of diplopia, although an external ocular paresis could not be demonstrated. On the 19th lumbar puncture was repeated, and yellow fluid not under increased pressure was obtained. Following this, she complained of headache, and became drowsy. A right lower facial weakness was noticed at this time.

Again there was some improvement, and on November 24 lumbar puncture was performed for the third time. A few drops of clear fluid, not under pressure, were obtained. In a matter of minutes she became shocked and lapsed into semicomatose.

The patient was transferred to the neurosurgical unit at the Royal Victoria Hospital, Belfast, where she arrived at midnight of the same day. She was deeply comatose on admission, with marked neck stiffness, extensor plantar reflexes, and B.P. 150/80 mm. Hg. The pupils were unequal, and fixed in dilatation. Papilloedema was not thought to be present. X-ray examination of the skull failed to reveal any fracture, and the pineal gland was not visible.

Operation was undertaken immediately. In view of the previous right-sided signs, a left temporal burr-hole was made under local analgesia. The dura was found to be

tense, but needling did not produce any fluid, bloody or otherwise. Three burr-holes were then made on the right side, and dark blood clot and old blood were evacuated from both the subdural and the subarachnoid spaces on that side. A pronounced but only transient improvement took place in the patient's condition, and death occurred at 5 p.m. on November 25.

Post-mortem Findings.—Necropsy was performed 17½ hours after death. On opening the skull an extensive subarachnoid haemorrhage became apparent, and it was found to be present chiefly over the base of the brain, more particularly on the right side. Its source was a ruptured aneurysm 1 cm. in diameter on a branch of the right middle cerebral artery, fairly high in the Sylvian fissure. The brain was generally swollen, the right temporal lobe more than the left. On sectioning the brain the subarachnoid haemorrhage was seen to extend for about 3 cm. into the substance of the right temporal lobe from the region of the aneurysm. No other intracerebral haemorrhages were revealed. Gross hernias of the uncus and cerebellum were observed, the latter very marked indeed, so that it was only with difficulty that the cerebellar tonsils and the brain-stem could be delivered from the foramen magnum. The lower 1.5 cm. of the tonsils was swollen, discoloured, and haemorrhagic. Histological sections from these affected areas showed recent haemorrhagic necrosis, in which there was an acute inflammatory cell reaction, together with fibrinoid necrosis of the walls of several small vessels.

Discussion

The clinical diagnosis of subarachnoid haemorrhage, including episodes of unconsciousness, could be satisfactorily, if superficially, confirmed at necropsy on the basis of the findings described above. It is in trying to reconstruct the precise chronological details of these changes that problems arise.

It is probable that a potentially reversible degree of uncomplicated cerebellar coning developed early in the clinical course. However, the age of the necrotic process in the tonsils was only about 24–48 hours. This particular lesion, if precipitated by lumbar puncture, was more likely to be related to the last occasion than to the first two. The intracerebral haemorrhage, too, was of recent occurrence, but its histological details do not permit of such an accurate estimate of its age. It may have been precipitated also by the third lumbar puncture, but equally well may have arisen earlier, either spontaneously or in relation to the previous lumbar punctures. Irrespective of when and how this latter haemorrhagic lesion took place, it seems indisputable that it materially aggravated the degree of cerebellar coning already present. Schaller (1933) and Masserman (1934) pointed out that foraminal herniation is due not so much to a direct "plugging" effect following the withdrawal of cerebrospinal fluid as to oedema of the brain which develops subsequently. An intracerebral haemorrhage could be visualized easily as contributing to this. Clearly, in the present case, at least three distinct factors—subarachnoid haemorrhage, cerebral haemorrhage, and lumbar puncture—interacted in a complex manner to produce the pathological end-result described.

These speculations, academic and inconclusive though they may be, do not invalidate the general warning of the dangers of injudicious lumbar puncture in cases of subarachnoid haemorrhage. It is impossible to escape the conclusion that lumbar puncture in this case contributed at least to the cerebellar coning and necrosis, and possibly further subarachnoid bleeding. It would seem that, once the diagnosis is established by the withdrawal of evenly blood-stained fluid which leaves a xanthochromic supernatant fluid after centrifuging, there are no further indications for lumbar puncture in cases of subarachnoid haemorrhage.

Summary

A case of subarachnoid haemorrhage due to rupture of a cerebral aneurysm is described. Death occurred

after the third lumbar puncture. At necropsy, cerebral haemorrhage, cerebellar coning, and tonsillar necrosis were found. No reports of the last-named lesion can be found.

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ACUTE DERMATOMYOSITIS

BY

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Recent articles (Oppel *et al.*, 1950; Thorn *et al.*, 1950; Suzzman and Rudolph, 1951) have shown the value of cortisone and A.C.T.H. in acute dermatomyositis. A further case of this rare disease responding dramatically to cortisone therapy is described.

Case History

A domestic servant aged 24 was admitted to the Central Middlesex Hospital on May 14, 1952. She had been in good health till three months previously, when her fingers became stiff and swollen at times. A week before admission her left hand felt weak, and the following night a sensation of tightness developed in her throat. She could not swallow properly. When she awoke the following morning (May 10), both hands felt weak. They were not painful. The weakness and stiffness of the finger- and wrist-joints soon involved the elbows and shoulders, and later both her knees and hip-joints and her back. Her legs felt useless and her muscles became increasingly painful and tender to pressure. On the same day a bright red rash started on the dorsal surface of her hands, soon spreading up both arms, and a similar rash involved both legs. On May 12 her eyelids felt swollen and stiff and they were red-rimmed. This condition lasted only a few hours. Throughout this time she felt ill and had lost her appetite completely. She vomited on three occasions. Each movement was an effort, and she ached all over when she attempted to shift herself in bed.

Physical examination showed her to be well nourished but very ill. Her temperature was 104.6° F. (40.3° C.). Her tongue was heavily furred, and her skin was hot and clammy. There was a diffuse confluent erythematous rash on the dorsal aspects of both feet and on the upper arms. In other areas irregular red macules were present. The rash was mainly confined to the limbs, but had a few extensions on the skin of the abdomen and back. She was profoundly weak, being unable to sit up, and only with difficulty could she straighten her arms or bend her knees. All her muscles were tender on palpation and felt firm and full. The superficial reflexes were normal.

On May 15 her temperature was 103° F. (39.4° C.). The rash appeared to be clearing from the forearms and from the dorsal surfaces of her feet. However, she seemed weaker and to have become worse. She was totally unable to move herself and could swallow fluids only, as she had marked dysphagia. She had a troublesome watery diarrhoea and retention of urine developed, which required catheterization. Her general condition was deteriorating so rapidly that it was decided to give cortisone immediately, and 100 mg. was injected intramuscularly at 5.30 p.m. An hour later she could move herself in bed more freely and could bend her