(see Fig.). Occasional papillary proliferations and solid cell masses were present. The abnormal tissue tailed off peripherally into foetal thyroid, though there was a very incomplete, inconspicuous fibrous layer between the tumour and normal foetal thyroid. Centrally the tumour had grown through the eroded tracheal ring and into the submucosa of the trachea, causing the latter to be greatly thickened. The mucosal epithelium itself was absent on the affected side. The presence of colloid-filled vesicles in the tracheal portion of the tumour confirmed the thyroid origin. The lung showed alveolar distension with rupture of some alveolar walls.

Comment

Although the tumour had a generally follicular appearance, the presence of papillary elements indicated that this was a papillary adenocarcinoma. The diffuseness of the lesion is in keeping with this diagnosis.

The failure to cry at birth, together with the histological appearance of the lungs, indicates that the tracheal obstruction impeded expiration of air and resulted in obstructive emphysema, resulting in death.

The mother of this infant lived 100 miles (161 km.) from the Kaonde Hospital and had not had antenatal supervision, let alone radiological examination. She comes from an area of Northern Rhodesia where endemic goitre is prevalent, and the enlargement of her thyroid was not unusual. Thyroid carcinoma is said to be associated with endemic goitre, but Winship (1956) refutes this. However, Crile (1959) has pointed out that the effect of thyroid-stimulating hormone (T.S.H.) on thyroid malignancies is deleterious. It could not be shown that the mother's thyroid was dysfunctional and there was no evidence that T.S.H. caused malignant change in the thyroid, but the general enlargement of the foetal thyroid in this case may have been connected with some such factor.

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Fatal Infarction of Brain in Migraine

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A number of cases have now been reported in which there has been clinical evidence of permanent damage to the brain by ischaemia in the course of an attack or series of attacks of migraine, the latest report being by Connor (1962). It has therefore seemed inevitable that ultimately a case would occur in which an attack was associated with sufficient damage to cause death. So far, however, only one such case has been reported (Peters, 1934), and no mention was made of the state of the carotid arteries, although the intracranial vessels, aorta, heart, and coronary arteries were carefully examined. The present case is of great importance, as not only was there widespread ischaemic damage to the brain, but it was felt necessary to carefully examine the internal and common carotid arteries, the internal carotid artery on the side with the greatest ischaemic damage to the brain being followed right through the carotid canal.

Clinical History

The patient, a man aged 28, had been well for the last year, but for two years prior to this had suffered from "very bad migraines." His wife thought he had had about five attacks in the two years. During the attacks, which lasted two to three hours, he had headache over both eyes together with nausea. He had twice been brought home from work "with very bad attacks." A year ago, at work, he had been found unconscious in one of the toilets. Radiographs of the skull and chest appeared normal. He was admitted to Selly Oak Hospital at 10.20 p.m. on 14 July 1961. His wife said he was quite well until 8.40 p.m. when he was eating a meal. He suddenly went blank and unresponsive and slumped on to a couch. He was cold and pale, and she noticed that the corners of his mouth were not level. On examination he was restless and semicomatose with a right hemiparesis ; the fundi were normal. Lumbar puncture gave clear C.S.F. under 100 mm. pressure, containing 5 red cells per c.mm., 1 leucocyte per c.mm., and protein 10 mg./100 ml. ; the chlorides, sugar, W.R., and Lange curve were all normal. The blood Hb was 12 g./100 ml. and the E.S.R. 2 mm. in one hour.

At 2.50 a.m. he was still restless and in semicoma. The eye movements were normal. The right hemiparesis affected the face and upper and lower limbs, and there was a right extensor plantar response. He was given penicillin injections.

The coma deepened and he was transferred to the Midland Centre for Neurosurgery, where two emergency burr-holes were made, but he died an hour later. (The time from onset to death was 20 hours 15 minutes.)

Post-mortem Examination.

External Examination.—The body was that of a young, well-nourished man. Apart from the burr-holes in the frontal region there were no external abnormalities. *Nervous system :* The brain was removed and fixed undissected, but it was noted that there was hyperaemia with small pial haemorrhages in the distribution of the left anterior cerebral artery. The tonsils had perhaps descended a little way towards the foramen magnum, but were not definitely engaged, nor was there any evidence of uncal herniation. *Respiratory system :* The right lung was adherent to the chest wall and both lungs showed very marked oedema. *Cardiovascular system :* The heart weighed 340 g. but was entirely normal, with only the slightest atheroma of the coronary artery. The internal and common carotid arteries were opened up throughout their course, including on the left side the portion of the internal carotid artery in the carotid canal. The vertebral arteries were filled with neoprene, which passed freely from their origins to the basilar artery. The spleen weighed 175 g. The Malpighian corpuscles were prominent but there was no definite abnormality. *Alimentary system :* The stomach and intestines were normal. The liver weighed 1,750 g. and was normal. The suprarenals were normal. The right kidney weighed 140 g. and the left 130 g., both appeared normal.

Examination of Brain After Fixation.—The brain, which weighed 1,302 g., showed a distinct broadening of the superior and middle frontal convolutions on the left side. The overlying pial vessels showed an engorgement of their finest branches, some of which were surrounded by very small haemorrhages. The vessels at the base of the brain, in particular the internal carotid and anterior and middle

cerebral arteries, appeared entirely normal. On coronal section the cortex of the left superior and middle frontal convolutions at the level of the head of the caudate nucleus was grey and stippled with petechial haemorrhages. The demarcation between cortex and white matter was blurred (Fig. 1).

Histology.—Half-hemisphere blocks were taken for low-viscosity nitrocellulose embedding from the following areas on both sides: dorsal and ventral halves of the frontal lobes at the level of the head of the caudate nucleus ; the frontal and temporal lobes at the anterior limit of the hippocampus ; parietal and temporal lobes at the level of Ammon's horn and occipital lobes at a level passing through the posterior horn of the lateral ventricle ; sections of the lateral lobes of the cerebellum on both sides, passing through the dentate nucleus, the pons in its upper and middle thirds, and the medulla through the lower third of the fourth ventricle. Sections from all the blocks were stained with Woelcke for myelin, haematoxylin-van Gieson, phosphotungstic-acid-haematoxylin, and cresyl violet.

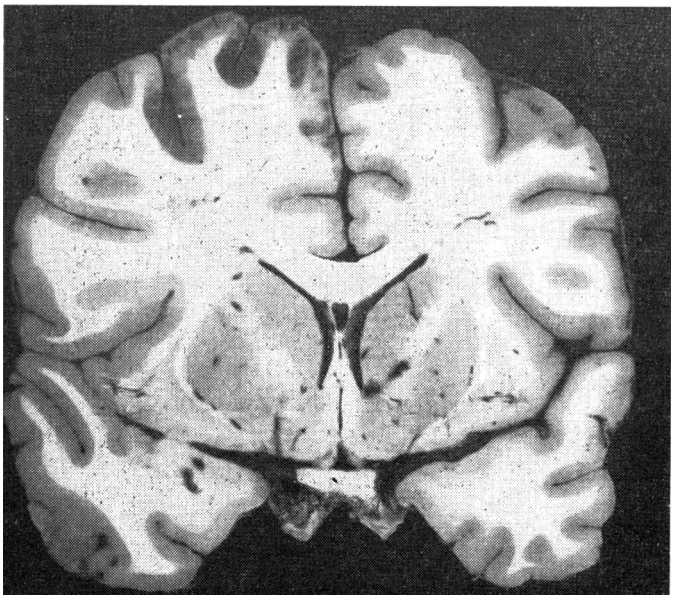

FIG. 1.—Coronal section through the cerebral hemispheres showing haemorrhagic infarction of the left superior and part of the middle frontal convolutions and small haemorrhages in the internal capsule.

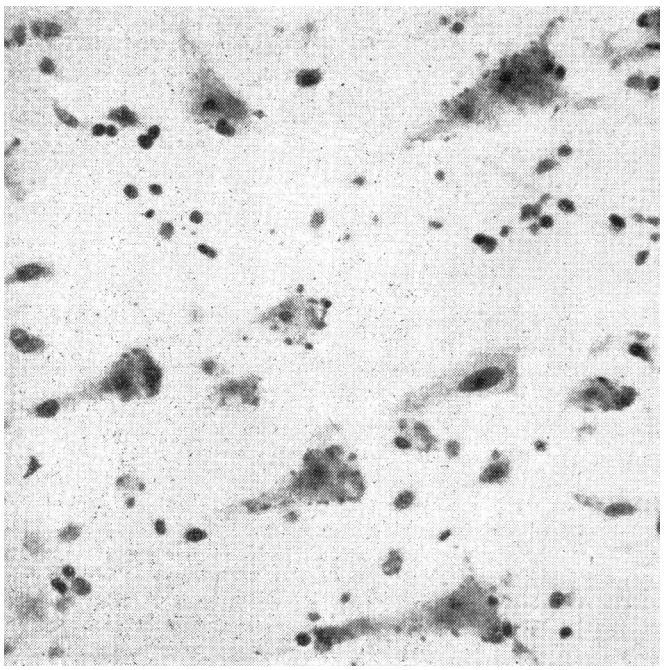

FIG. 2.—Ischaemic and encrusted nerve cells from the left superior frontal convolution. (Cresyl violet. ×437.)

FINDINGS

The cortex of the *left superior and middle frontal gyri* showed ischaemic or severe cell change of the nerve cells in all layers at both levels examined. Many of the affected cells showed very marked encrustation (Fig. 2). There was little reaction in the glia, but the nuclei of the astrocytes showed commencing pyknosis. Small haemorrhages were scattered throughout the affected cortex, but not obviously related to blood-vessels. The cortical myelin sheaths showed fusiform or spherical swelling, which in some areas was very marked. In some areas formalin pigment was present on the surface of the nerve cells. The pial vessels showed no abnormality. Severe and widespread ischaemic or severe cell change was also seen in the nerve cells of the cortex of the left insula, paracentral lobule, and post-central convolution. Ischaemic change was also seen in the cells of Sommer's sector of the left Ammon's horn and the subiculum of the right Ammon's horn. Less widespread areas of cortex with ischaemic or severe cell change were seen in the right post-central convolution, the cortex of almost the whole of the convexity of the dorsal half of the left occipital lobe and the ventral half of the right and left occipital lobes, the substantia nigra especially on the left, both nuclei reticularis pontis, and the left nucleus of the spinal tract of the fifth nerve. There were two pinhead-sized haemorrhages in the anterior limb of the right internal capsule and satellitosis of nerve cells in the right caudate nucleus.

COMMENT

It is unfortunate that this patient's migrainous attacks lacked the more characteristic visual features ; the fact remains that he suffered from attacks of headache associated with nausea, that he had lost consciousness in one of them, and finally succumbed to the last, with histological evidence of ischaemia largely in the territory of the left anterior cerebral artery, and this irrefutable evidence of focal disturbance of cerebral blood flow was unassociated with the slightest structural abnormality of the parent vessels or heart. We have recorded the case under the above title to emphasize that fatal infarction of the brain can occur in one of a series of headaches presumably due to spasm of one or more cerebral arteries. The cause of death was ischaemia of the brain stem, and we have presented histological evidence of this. It indicates that the disturbance of cerebral blood-flow was not confined to the anterior cerebral artery.

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