aspect, especially in the insular region, multiple elongated cavities were present in the cortex of many convolutions. Though the head of the caudate nucleus and lenticular nucleus was present in the specimen, the thalamus and hippocampal uncus had not been excised.

Microscopical Findings.—There was extensive cystic laminar necrosis of the cortex, maximal in the frontal lobe and insular region (Special Plate, Fig. 1). The cystic cavities were lined by dense glial tissue, with severe gliosis of the overlying molecular layer and an interlacing glial network in some of the cavities (Special Plate, Fig. 2). A few scattered histiocytes and lymphocytes were present in the spaces. Smaller cysts were present in the intervening cortex, and these were seen to occupy the middle cortical laminae (Special Plate, Fig. 3). In the more intact cortex the nerve-cells in the second and third laminae had disappeared, being replaced by a band of dense glial tissue (Special Plate, Fig. 4). The underlying white matter showed severe demyelination with replacement gliosis. The medial aspect of the hemisphere was much less severely involved, with patchy loss of nerve-cells in the middle cortical laminae and replacement gliosis without cyst-formation. There was preservation of the subcortical white matter in these areas. The occipital-lobe cortex was the least affected, though there were cysts on the lateral aspect but only patchy nerve-cell loss with glial scars on the medial aspect. There was severe subependymal gliosis around the dilated ventricle, with scattered areas where the ependyma was missing. The basal ganglia showed diminution in nerve-cells with increased cellularity due to astrocytic increase, while many of the vessels in this region showed severe gliosis of the walls of their perivascular spaces. The overlying pia-arachnoid was markedly thickened, but was devoid of inflammatory reaction apart from scanty lymphocytes and histiocytes. The pial vessels were free from thrombus, though several of the arteries, especially posteriorly, showed slight intimal proliferation.

COMMENT

The possible causes of eclampsia in pertussis have been reviewed by Ellison (1934). It seems very likely that all the cases with brain damage are not due to the same cause. Evidence of encephalitis with inflammatory cells was found in the cases reported by Askin and Zimmermann (1929), Worster-Drought (1940), and Woolf and Caplin (1956). Dolgopol (1941), however, found inflammatory reaction to be rare, and the main changes to consist of oedema, ischaemic cellular degeneration, and small haemorrhages, which were thought to be of circulatory origin. Nelson (1939) attributed the damage to minute haemorrhages or encephalitis, while Ford (1929) found haemorrhages in four out of 11 cases.

Many observers, however, have noted that haemorrhages are sometimes absent, or, if present, are not extensive enough to explain the symptoms. Hiller and Grinker (1930) found cortical softening of varying degrees of severity, though no organic obstruction to vessels could be demonstrated, suggesting stasis as the basis of tissue damage. Neubürger (1925) attributed the areas of cell damage to hypothetical air embolus. In recent years anoxia has become more widely regarded as the cause of complications in some cases (Berg, 1961).

The findings described in the excised cerebral hemisphere in the present case consisted of severe and diffuse cortical damage with cyst-formation and intense gliosis maximal on the lateral surface. The degeneration of the underlying white matter was secondary to this. There was no evidence of any inflammatory reaction, haemorrhage, or thrombosis.

The diffuse loss of nerve-cells in the cortex, not confined to the territory of any particular vessels, could be the result of either air embolism from ruptured alveoli

or severe anoxic damage. In air embolism Lhermitte and Barrelet (1934) described spongy transformation of molecular and external granular layers of the cortex with scattered small, irregularly shaped foci, often perivascular, from which all nerve-cells had disappeared. No changes were seen in the white matter, though Courville (1945) found numerous punctate ring haemorrhages associated with infarcted areas in his cases. In the present case the changes in the white matter were regarded as secondary to the cortical destruction. The absence of focal lesions in both the cortex and white matter and the diffuse damage to the middle cortical laminae would be more consistent with severe anoxic damage to which spasm of vessels may have contributed. This anoxia presumably occurred initially during a severe paroxysm of coughing, though subsequent epileptic attacks starting six months later may have augmented the damage. There was evidence of some involvement of the hemisphere on the opposite side, as the right lateral ventricle was slightly dilated, and the E.E.G. was not entirely normal, suggesting a lesser degree of damage by a diffusely acting process such as anoxia.

My thanks are due to Mr. J. Hardman, F.R.C.S., under whose care the patient was admitted, for allowing me full access to his clinical notes and for the details of the operation.

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Long-delayed Complications of Closed Abdominal Trauma

The appearance of long-delayed complications after closed abdominal trauma has been sporadically reported but is not widely recognized. The following case is reported because it exhibited two such complications—the development of a lesion resembling Crohn's disease and delayed perforation occurring 10 weeks after the original injury.

CASE REPORT

A woman aged 53 was admitted to hospital on November 14, 1960, after a road accident. She had previously been healthy and had had no bowel symptoms whatever. On admission she was severely shocked and had compound fractures of humerus, ulna, tibia, and fibula, with multiple lacerations. She did not appear to have any intra-abdominal

injury but was semi-comatose at this stage. After resuscitation her fractures were dealt with and her condition was satisfactory for two days.

On the third day she developed abdominal distension and bilious vomiting, with absence of bowel sounds. Treatment with gastric aspiration and intravenous fluids improved her condition and her distension settled with the passage of flatus. Suction and parenteral fluids were discontinued, but two days later her ileus returned and they were recommenced. After a further four days' treatment her abdomen had returned to normal. Three days later she had an attack of diarrhoea followed by a short period of melaena which was thought to be due to sloughing of contused mucosa. This settled after transfusion.

The patient was later discharged and followed up at the fracture clinic. When seen on January 31, 1961, she was well but stated that she occasionally had slight abdominal distension relieved by passage of flatus. On February 4 she was readmitted to a surgical ward as an emergency case, complaining of severe abdominal pain and vomiting for eight hours. Her clinical picture was that of diffuse peritonitis and after a short period of resuscitation she underwent laparotomy.

At operation an inflamed lower ileum was found and a length of 15 cm. was thickened and oedematous, and showed mucosal ulceration. At one point there was a perforation 1.5 cm. in diameter from which ileal contents were leaking. The perforation was partially sealed by omentum. The terminal 2 ft. (60 cm.) of ileum was resected and continuity restored by end-to-side ileocaecal anastomosis. Convalescence was punctuated by hypotassaemia requiring intravenous correction and a pelvic abscess which discharged spontaneously.

Histological Report (Dr. Knudsen).—Sections show ulceration of ileal mucosa with replacement by inflammatory granulation tissue (see Fig.). The submucosa and muscle coats show marked congestion, oedema, sparse inflammatory cell infiltration, and early fibrosis. The serosa is congested and oedematous with minimal fibrosis. The related lymph

Section of mid-zone of wall of ulcerated ileum, showing granulomatous change in submucosa. ($\times 8$.)

nodes show reactive hyperplasia and distension of sinuses. No areas of sarcoid reaction seen. The mesenteric vessels are patent. The histological appearances are compatible with Crohn's disease but the lesions are non-specific and in view of the previous history an unusual response to trauma cannot be excluded.

DISCUSSION

Development of a Lesion Resembling Crohn's Disease.—This, again, has been reported at intervals but is not widely recognized. Aird (1957), in an intensive summary of a possible aetiology, does not mention trauma as a cause, nor does Hadfield (1939) in his

classical description of the histological picture of Crohn's disease. Crohn *et al.* (1932) failed to mention it in their own original description. However, in 1931 Mock described a heterogeneous collection of bowel granulomata which obviously included diseases now recognized as Crohn's disease and diverticulitis. He also defined a traumatic group and reported several cases very similar to the one here described.

In most such cases, including the two Corbett (1957) described, the lesion has manifested itself by the development of intestinal obstruction. This has occurred after a shorter interval than is usual with Crohn's disease—namely, an average period of about three months, compared with one of several years of intermittent symptoms in the classical disease. Cases described have involved ileum, transverse colon, and descending colon. One case has been described following pregnancy, and although it is more difficult to exclude coexistent Crohn's disease in such a case it is interesting that other traumatic conditions—for example, spontaneous rupture of spleen—have been described following pregnancy. There is little doubt that the post-traumatic lesions are separate from classical Crohn's disease, as none of the reported cases have shown multiple lesions with skip areas, and we have been unable to find a report of recurrence after resection.

There has been no obvious mechanism in the development of the lesion in most cases, although in one of Mock's cases a transverse mesenteric tear was found opposite the lesion, which appeared to be due to interference with the blood supply at that level.

Delayed Perforation of Intestine.—Perforation delayed for 6 to 24 hours after closed abdominal injury is a well-recognized and relatively common occurrence. Perforation delayed longer than this is much less common, although Gummer and Ranking (1953) described a perforation of the caecum occurring six days after injury. We have been unable to find an account in recent literature of perforation manifesting itself as late as three months after the original injury, although Mock found a perforation sealed with omentum at laparotomy for another condition 20 days after an injury. This may well have reperforated in time, as in our case. Mock's case is also the only one we have been able to find where delayed perforation has been associated with a granulomatous condition, and it may well be that this reperforation was in large part due to the development of obstruction distal to the site of sealed perforation.

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