ment is a random process. The Chart shows the increase in specific activity with time for replacement at 1, 2, 5, and 10% per year. It also shows observed specific activities for various bones (femora, ribs, vertebrae, ilia, calvaria, patellae). The observations for 1959 and 1961 are for normal subjects killed in accidents. The plots suggest that replacement is 1–2% per year for calvaria, 2–5% for femora, ribs, and patellae, and 7–10% for ilia and vertebrae. Kulp and Schulert (1961) calculate a figure of 2.5% per year for the whole skeleton.

CONCLUSION

The conclusion from the present paper is that strontium built into bone (as distinct from that on bone surfaces) is more tenaciously held and recycled than strontium entering the plasma from diet. Estimates of the annual rate of replacement in the adult are thus only a few per cent. per year, when calculated from long-term ingestion of strontium-90, somewhat more according to short-term experiment involving ingestion of strontium-85 for a month, and much less than the total turnover of bone mineral.

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In the July issue of *F.P.A. Journal*, the journal of the Fire Protection Association, appears the following extract: "One hundred years ago deaths from clothing burns among women presented a serious problem. The following is extracted from *Scientific American*, July, 1861: The public has been painfully startled by the sudden death of Mrs. Longfellow—wife of the distinguished poet—which took place at Cambridge, Mass., on the 10th inst. The cause of this afflicting event is thus related: Mrs. Longfellow was seated in her library, and in the act of making seals with sealing wax. A bit of paper lighted and fell upon her dress, which caught fire, and before the blaze could be extinguished she was terribly burned. She was attended by Drs. Wyman and Johnson of Cambridge, but their efforts were vain to alleviate her intense suffering or to save her life. The dresses commonly worn by ladies in warm weather are composed of muslin and such-like inflammable materials. Although we have, on several occasions, urged the preparation of ladies' dresses with non-inflammable agents, we regret to state that very little attention has been given to our admonitions. We trust that the subject of safety-clothing will hereafter receive more attention from ladies. Their safety and good sense demand this."

Medical Memoranda

Cerebral Damage in Whooping-cough

[WITH SPECIAL PLATE]

Though the occurrence of neurological complications in whooping-cough is well recognized, the mechanism of their production is not so clearly defined. The incidence of these complications ranges from less than 1% to more than 15% in the larger series in the literature (Litvak *et al.*, 1948; Berg, 1961). Hemiplegia is a frequent finding, occurring in about half the cases that develop neurological signs (Valentin, 1902; Sears, 1929; Grant and Williams, 1943). Amaurosis of cerebral origin, though rare, has been described (Lazarus and Levine, 1934; Woywitka and Riches, 1950). In the following case both hemiplegia and a visual field defect developed during the illness. Subsequent neurosurgical treatment enabled a pathological examination of the damaged cerebral hemisphere to be carried out, and the results throw some light on the controversial problem of the pathogenesis of these lesions.

CASE HISTORY

A 9-year-old girl with infantile hemiplegia and severe behaviour disorder was referred to the Neurosurgical Department of the Sheffield Royal Infirmary for investigation and treatment. She was the third child in a healthy family, being born at full term after normal delivery. Development was satisfactory till the age of 18 months, when she had a severe attack of whooping-cough, during which she became hemiplegic on the right side with a right homonymous hemianopia. This was considered at the time to be due to either encephalitis or thrombosis. Though she could say some words before the illness, afterwards she could only mumble a few sounds. At the age of 2 she started to have fits in which she cried out, turned to the left, fell down, and became incontinent. These persisted at irregular intervals, and were not controlled by phenobarbitone, phenytoin sodium, and primidone. Though she started school when 5 years of age, by the time she was 7 her speech was almost incomprehensible so that she had to be removed and given home tuition. The fits were still frequent despite anticonvulsant therapy, and her behaviour was extremely bad, with restlessness, swearing, and temper tantrums. Her behaviour gradually got worse.

Physical examination confirmed the right hemiplegia, with small, poorly developed limbs on this side and a right homonymous hemianopia. The optic fundi were normal.

An electroencephalogram (E.E.G.) showed frequent bursts of high-amplitude slow spike and wave complexes occurring bifrontally and extending posteriorly on the right. A lumbar air encephalogram revealed a dilated left lateral ventricle, while the right side was relatively normal. In view of her deteriorating behaviour and the results of the investigations a left hemispherectomy was performed.

In the subsequent five years she has gradually improved, especially in her behaviour and language, so that she can now attend a school for backward children. She is able to read and to write with her left hand. Her fits are well controlled with phenobarbitone gr. ½ (32 mg.) and primidone 125 mg. once a day. The E.E.G. now shows a flat record on the left side and some dysrhythmia on the right.

Macroscopic Examination of Left Hemisphere.—The specimen measured 13 × 9 × 3 cm. and weighed 250 g. The lateral ventricle was grossly dilated, with marked congestion of subependymal veins and thinning of the white matter, especially in the corpus callosum. The cortical ribbon was narrowed over most of the hemisphere, and on the lateral

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 Zimmerman (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

R. F. FLETCHER AND A. C. FRAZER: RENAL FAILURE AND OSTEOSCLEROSIS

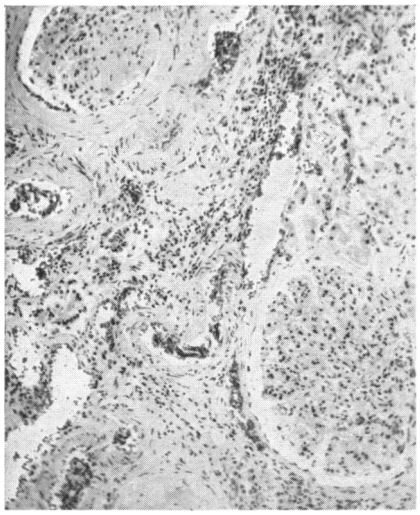

FIG. 1.—Histological appearance of left kidney at necropsy. (H. and E. ×60.)

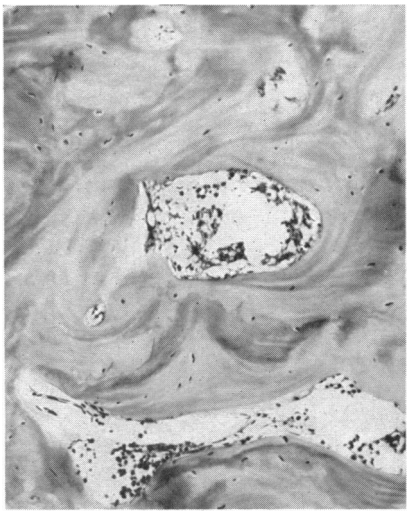

FIG. 2.—Histological appearance of iliac crest bone at necropsy. (H. and E. ×60.)

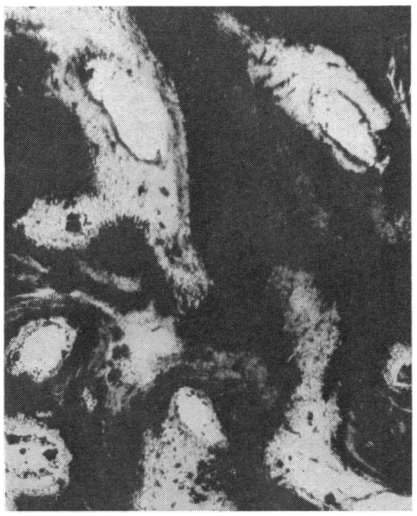

FIG. 3.—Iliac crest bone at necropsy. (Lillie preparation. ×60.) Black areas indicate calcified tissue, except that centres of trabeculae cannot be stained with this technique.

L. WOLMAN: CEREBRAL DAMAGE IN WHOOPING-COUGH

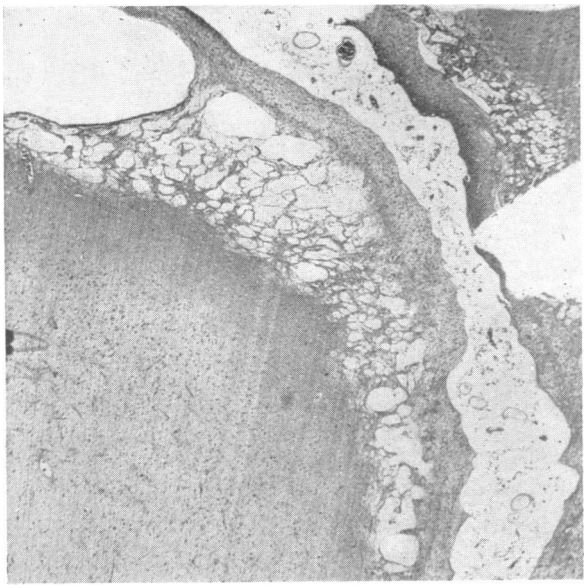

FIG. 1

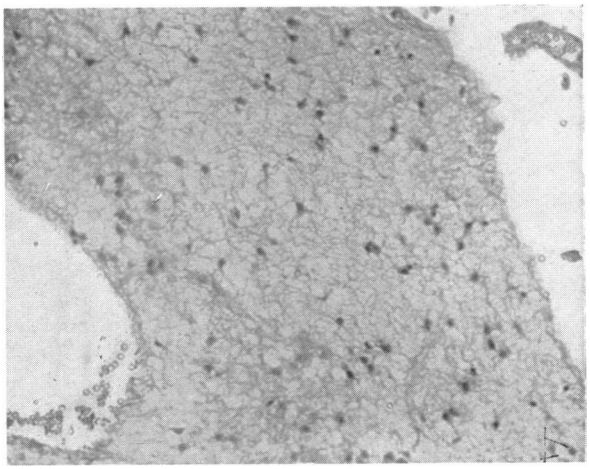

FIG. 2

FIG. 3

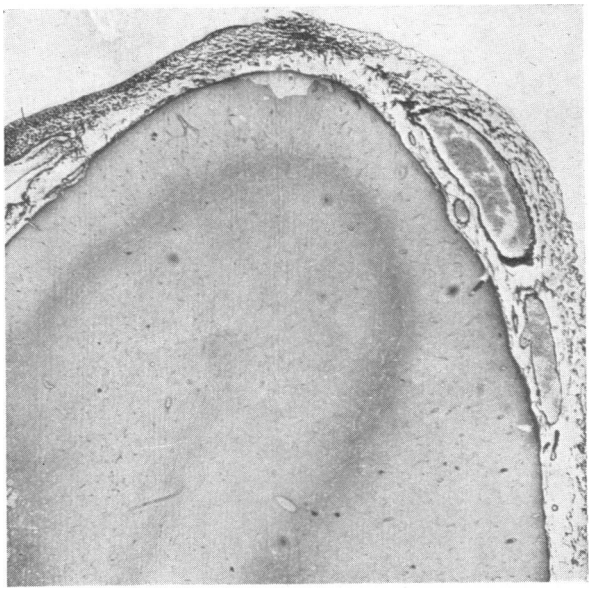

FIG. 4