pleuritic pain, which subsided in two days with chlortetracycline (1 g. daily for four days). Three days before admission she felt less well and in the evening developed intense headache, nausea, and vomiting, which persisted until the onset of coma. The doctor examined her the following morning. Her temperature was 103° F. (39.4° C.) and pulse 100. She had mild photophobia, but no neck stiffness was present. Chlortetracycline was recommenced. On the morning of admission she was semiconscious and very restless and had been incontinent of urine.

On admission the patient, a well-built girl, was unconscious and extremely restless. She was picking at her lips and nose. Her temperature was 98.4° F. (36.9° C.), pulse 80, and respirations 20. She was incontinent of urine. There was no skin rash. She had conjugate deviation of the eyes to the left. The pupils were dilated and did not react to light. Nothing abnormal was seen in the optic fundi. There was trismus. The teeth appeared to be normal. The

Photograph of section taken from white matter of right occipital lobe. Stained with haematoxylin and eosin.

tongue, so far as could be seen, was coated and moist. There were sordes on her lips. Both eardrums appeared normal. The lungs were clinically normal. The pulse was regular and the blood pressure 160/90. A soft mitral systolic murmur was heard. There was no sacral or ankle oedema and the jugular venous pressure was not raised. A corneal reflex was present on both sides. The reflexes and tone in both arms were normal. The abdominal reflexes were absent. The reflexes and tone in both legs were increased. There was ankle clonus. The left plantar response was extensor. Neck rigidity, mild opisthotonos, and a positive Kernig's sign were present. The patient failed to respond to nociceptive stimuli on the left side of the body. A provisional diagnosis of pneumococcal meningitis was made.

Lumbar puncture showed a pressure of over 300 mm. and 5 ml. of slightly yellow opalescent fluid was removed, crystalline penicillin (20,000 units) was injected into the theca and 2 g. of sulphadiazine was given intravenously, followed by 2 g. intramuscularly every four hours.

Despite treatment she lapsed into deeper coma. The corneal reflexes were unobtainable and the arms and legs became flaccid. A gallop rhythm was heard over the praecordium. The patient died 17 hours after admission.

Investigations.—C.S.F.: pressure, 300+ mm.; cells, 2,000 (polymorphs 90%); protein, 620 mg./100 ml.; chlorides, 720 mg./100 ml.; sugar, 80 mg./100 ml.; no organisms seen on direct examination. The 24-hour culture was sterile. Post-mortem blood urea, 124 mg./100 ml. The urine contained albumin.

Post-mortem Examination.—Apart from the kidneys and brain, nothing abnormal was found. The kidneys were enlarged one and a half times by thickening of the cortex,

which was a pale yellow colour with some red streaking. There was a thin layer of subarachnoid haemorrhage round the mid-brain, especially in the cerebro-pontine angles. The vessels of the circle of Willis and its branches were not obstructed. There was a large area of ischaemic softening involving almost the whole of the right occipital lobe. Finger-like processes of a similar nature extended along the wall of the lateral ventricle on both sides, especially at the junction of the lateral, posterior, and inferior horns, and the hippocampus. The right basal ganglia were similarly softened and there were isolated zones in the right parietal white matter and in the mid-brain.

Histological examination of the white matter of the right occipital lobe beyond the edge of an area of softening showed lesions of a patchy nature separated by brain tissue which was apparently normal. The affected parts were centred on blood vessels, particularly the small venules. These showed a curious loss of structure of the vessel wall which was converted into the structureless eosinophilic material usually described as "fibrinoid," and, although the endothelium was absent and the fibrinoid material somewhat fragmented, there was no effusion of red corpuscles through the wall. Polymorphs, however, were present in large numbers in the lumen, in the wall, and in the tissue surrounding the vessels. Capillaries were hardly affected, but small flame-shaped and ring-shaped haemorrhages were seen (see Fig.), the latter surrounding small areas of necrotic brain tissue. Serous exudate was not found in the sections examined, but fibrinoid material appeared to have diffused out of most of the damaged vessels in the surrounding tissue.

I wish to thank Dr. G. R. Steed for advice and permission to publish this case, and Dr. M. R. Thomas for performing the necropsy and preparing the histological sections.

H. E. ALDRIDGE, M.B., B.S.,

Late House-physician,

South Devon and East Cornwall Hospital, Freedom Fields.

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Case of Acute Haemorrhagic Leuco-encephalitis

Acute haemorrhagic leuco-encephalitis is a rapidly fatal disease of the central nervous system in which haemorrhagic lesions are found in the white matter of the brain. Crawford (1954) described three cases and found a further seven in the literature up to that time. Adams, Cammermeyer, and Denny-Brown (1949) described four similar cases under the name of acute necrotizing haemorrhagic encephalopathy.

Clinical Description.—The condition is commonest in young adults, but the youngest in Crawford's series was aged 2 years 10 months, and the oldest reported case was aged 51. It is commoner in males than in females. In most cases an acute upper respiratory infection precedes the neurological signs by a few days. The latter are acute in onset, with high fever, neck rigidity, sometimes convulsions, and paralyses with no characteristic distribution. The onset of coma is early and death follows in a few days. The cerebrospinal fluid usually shows an increase in cells, the maximum in Crawford's series being 220 per c.mm., but Adams *et al.* reported up to 5,000 per c.mm. Most of the cells are polymorphs. The protein is slightly increased in about half the cases; it was 140 mg. per 100 ml. in one of Crawford's. The sugar and chlorides were normal in three cases. Those cases in which the blood count was recorded showed a polymorphonuclear leucocytosis.

Pathology.—The meninges may appear normal or congested. The convolutions are flattened. The cut surface of the brain shows one or more pinkish oedematous areas, with numerous small haemorrhages of varying size, not more than a few millimetres in diameter, scattered through the white matter. The distribution is usually mainly in the cerebral white matter of either or both sides, often asym-

metrical, and may extend down into the cerebral peduncles, pons, and, less commonly, into the white matter of the cerebellum. The microscopical findings are described in detail by Crawford and are a combination of haemorrhage, vascular lesions, demyelination, and neurofibril degeneration. Fibrin impregnation of the walls of small vessels, and often the surrounding tissue, is characteristic. Perivascular collections of cells, especially polymorphs, are also seen. Adams *et al.* describe similar lesions.

CASE REPORT

A carpenter aged 50 was admitted to hospital on December 12, 1952, complaining of vomiting blood. He had had two episodes of gastro-intestinal haemorrhage, in 1945 and 1951, and pneumonia in 1951. He was treated by blood transfusion, and his condition on December 29 was satisfactory. On January 7, 1953, he had seven septic tooth stumps removed under penicillin cover. On January 10 he complained of headache and cough, his temperature rose to 101° F. (38.3° C.), and there were signs of bronchitis. Penicillin, 500,000 units twice daily, was prescribed. Next day the headache was worse; he complained of slight neck stiffness and was coughing up a little blood-stained sputum. A few rales were heard at the base of the right lung. Lumbar puncture gave a turbid cerebrospinal fluid at a pressure of 110 mm. with 1,400 cells (almost all polymorphs) per c.mm.; no organisms were seen or cultured. Penicillin, 500,000 units four-hourly, and sulphadiazine, 1 g. four-hourly, were prescribed.

On the 12th he was semiconscious, neck rigidity had increased, but there were no other neurological signs; he was incontinent of urine. Lumbar puncture gave a turbid C.S.F. at a pressure of 100 mm. The treatment was changed to intravenous chlortetracycline, 300 mg. twice daily, intrathecal streptomycin, 100 mg., and intramuscular streptomycin, 1 g. twice daily. Later in the day he was unable to swallow, and at 7 p.m. he suddenly became very pale, sweated profusely, and died within a few minutes. A tentative diagnosis of septicaemia and meningitis following the dental extractions was made.

Necropsy was carried out 16 hours after death; the body was that of a thin middle-aged man. The meninges over the brain and spinal cord were congested; no pus was found. The cut surface of the brain showed large numbers of small haemorrhages throughout the white matter of both cerebral hemispheres, extending down into the cerebral peduncles and in the white matter of the cerebellum. The spinal cord appeared normal. Thick mucopus was present in the larynx, trachea, and bronchi; there was almost confluent bronchopneumonia at the base of the left lung, with patchy bronchopneumonia at the base of the right lung.

Section from the cerebral white matter, stained by phosphotungstic acid haematoxylin, showing exudate of fibrin and red cells in a perivascular space.

The stomach was congested, but no gastric or duodenal ulcer was found. There were no gross lesions in the other organs.

Microscopically the cerebral white matter showed extensive demyelination, haemorrhages round capillaries, and small arterioles, and marked perivascular infiltration, mainly with polymorphs. A few polymorphs were also scattered diffusely through the white matter. Fibrin impregnation was especially noticeable round capillaries, but also as fibrin plugs, and in the walls of some arterioles. Section of the left lung showed bronchopneumonia. The spinal cord was normal.

COMMENT

Diagnosis.—Clinically the present case resembled meningitis, and the failure to culture any organisms from the C.S.F. would not exclude it. In other cases an intracranial abscess or thrombophlebitis might be suspected, and middle-ear and other local infections would have to be excluded. The course of the disease is more rapid than in most cases of brain abscess. Adams *et al.* mention a similarity to epidemic equine encephalitis, which can be excluded in this country. Post-vaccinal and post-infective encephalitis usually run a milder course, polymorphs are not found in the cerebrospinal fluid, and there is a history of vaccination or an acute exanthem.

Necropsy.—Severe equine encephalitis and purpura can be excluded, as they are not confined to the white matter. Fat embolism may present a similar picture, but can be excluded histologically. Haemorrhages are not seen in other forms of encephalitis. There is no difficulty in excluding meningitis, brain abscess, or thrombophlebitis. In polyarteritis lesions will be found in other organs.

The aetiology has not been established; the following points, however, must be considered. (1) Bacterial: no organisms were isolated from the C.S.F. in the present case or any of Crawford's series. (2) Virus: Adams *et al.* were unable to transmit the disease to mice; Crawford could not transmit the disease from one case to a monkey. (3) Sensitivity to drugs is unlikely, as most of the cases occurred in previously healthy patients; the use of sulphonamides has been mentioned in only one case (Adams *et al.*); penicillin has been given only to the more recent cases. (4) The direct action of circulating toxins cannot be excluded. (5) Acquired hypersensitivity to an unknown antigen seems the most likely in view of the resemblance of the vascular lesions to those of polyarteritis and experimental hypersensitivity reactions (Crawford, 1954).

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N. E. RANKIN, M.B., D.Path.,
Assistant Pathologist, Hope Hospital, Salford;
formerly Assistant Pathologist, Gloucestershire Royal Hospitals.

JEAN DANCE, M.B., D.C.H.,
General Practitioner, Gloucester;
formerly Medical Registrar, Gloucestershire Royal Hospitals.

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"Cruel Parents" is the title of a 25-page pamphlet written by two of the staff of the Institute of Psychiatry, London University—Dr. T. C. N. GIBBENS, senior lecturer in forensic medicine, and Mrs. A. WALKER, a psychiatric social worker. The pamphlet reviews the results of interviewing and examining clinically 32 men and 7 women sentenced to prison for cruelty to children which involved violence. As much information as possible about these offenders was also obtained from other sources such as the N.S.P.C.C. The pamphlet is published by the Institute for the Study and Treatment of Delinquency, 8, Bourdon Street, Davies Street, London, W.1, price 1s. 6d.