direct pressure on the optic chiasma from a dilated third ventricle (Brain, 1962), the exact mechanism cannot be categorically stated. The exact mechanism of recovery also cannot be fully explained, though it is known that recovery of vision can follow prolonged blindness in children with hydrocephalus (Lorber, 1967). Shaffer and Hetherington (1969) suggested that the neuroglial framework is more susceptible than the neurones to an increase in surrounding pressure, and this, of course, can regenerate when the pressure is relieved.

For the help given in the care of this case we are grateful to Miss U. J. Hull, public health nurse, and to Dr. Christian Belcourt, visiting radiologist from the Children's Hospital, Boston, U.S.A.

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Purpura Fulminans Treated with Hyperbaric Oxygen

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Purpura fulminans is an acute and often fatal disease of unknown aetiology. It is characterized by the sudden occurrence of massive ecchymotic lesions on the lower extremities that rapidly proceed to necrosis and gangrene. Other parts of the body may be affected, and visceral involvement may also occur (Hjort, Rapaport, and Jørgensen, 1964).

We report the effects of hyperbaric oxygen in a child with purpura fulminans and suggest that it merits further trial in this serious condition.

CASE REPORT

A boy aged 14 months was admitted to hospital with purpura and bruises on his legs extending up to the knees and some purpura on his face and arms of 3 days' duration. During the week before admission he had had a mild non-specific upper respiratory tract infection. On examination he had extensive and symmetrical purpura and ecchymoses on both legs and a few purpuric spots on the face and arms. There was generalized lymphadenopathy, the spleen was palpable, the throat was infected and inflamed, and there was left otitis media.

Investigations.—Haemoglobin 12 g./100 ml.; white cell count 12,400/mm.³ (polymorphs 25%, lymphocytes 71%, monocytes 3%, eosinophils 1%); platelets 304,000/mm.³ Sedimentation rate 27 mm. in 1 hour (Westergren). Blood culture, electrolytes, urea, cholesterol, immunoglobulins, prothrombin time, and kaolin/

cephalin time normal. Excessive fibrinolysis absent. Serum proteins 6.7 g./100 ml. (albumin 3.85 g., and globulin 2.85 g.). Urine and faeces examination negative.

Despite treatment with cloxacillin (50 mg./kg./24 hours), fusidic acid (Fucidin) (20 mg./kg./24 hours), and prednisolone 5 mg. six-hourly (increased after 24 hours to 10 mg. six-hourly), his condition deteriorated greatly during the next 48 hours. Fresh ecchymoses appeared on his legs. Pedal pulses were almost palpable, and all his toes had become tensely swollen and black, giving the appearance of cutaneous gangrene. He became oliguric, his face became puffy—the eyes being almost closed by oedema of the eyelids—and he developed macroscopic haematuria. Occult blood was present in the stools. In view of the early signs of gangrene it was decided to try the effect of hyperbaric oxygen.

On his third day in hospital he was premedicated with 180 mg. of chloral hydrate and placed in the oxygen chamber. The pressure was raised to 23 lb./sq. in. (1.6 kg./cm.²), or 2½ atmospheres absolute. He remained asleep, and after 10 minutes his toes became pink. He awoke after 60 minutes and the pressure was lowered at 75 minutes. The next day there were only a few new purpuric spots on his knees and he was given two further treatments of 75 minutes at 2½ atmospheres absolute pressure. On the fifth day there were no new purpuric areas, the puffiness had almost gone, and the feet remained pink. He was given two further treatments of 75 minutes. On the sixth day he had one more treatment, during which he crawled up and down the chamber, and as his feet were normal and all his spots were fading no further hyperbaric oxygen treatment was given. The only adverse effect of the vascular occlusion of his feet was a small area of scabbing, 0.25 cm. diameter, on the skin of both big toes. The total time in the chamber was eight hours five minutes at 2½ atmospheres absolute. There were no side effects.

COMMENT

We feel that hyperbaric oxygenation was largely responsible for the dramatic recovery in this patient. It certainly prevented progression of ischaemic changes resulting from poor tissue perfusion and also allowed satisfactory oxygenation of ischaemic tissues until such time as recovery occurred, possibly aided by corticosteroids. It would seem that it shortened the duration of the disease, thus possibly preventing the development of many of the coagulation abnormalities which often appear later (Hjort *et al.*, 1964). We have found only one report in the literature of a child treated in a similar way (Waddell, Saltzman, Fuson, and Harris, 1965), but we used higher and shorter periods of oxygen tension than these workers.

We thank Dr. G. A. Beck, consultant dermatologist, for referring this patient to us, and Dr. Marguerite King, consultant haematologist, for the haematological investigations.

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