failure of the valves.¹ This could not have been the sole cause, or possibly even the main cause. In each of the cases reported by Pierie *et al.* chemical analysis had shown the presence of substantial quantities of cholesterol and other fats in the silicone balls, and rosette type crystals were found in all altered balls. We have now examined several aortic prostheses removed at necropsy a few weeks after insertion, and in each case fat deposits in the rosette form have been seen on the surface of either silicone or metal ball (Figure $\times 285$).

There is a voluminous literature which shows that the levels in the blood of cholesterol and the other fats concerned are raised by stress, which, as well as apprehension and emotional stress, includes physical stress caused by trauma, surgery, or severe illness. Such stress was present in the cases quoted and is certainly present in our short-term examples of major surgery followed by terminal illness. This presence of a raised level of cholesterol in the blood would thus seem to be one of the contributory factors. It seems most probable, therefore, that in many instances transient surface deposits of the fats are formed, but that in cases of prolonged stress sufficient fats may be deposited to penetrate the structure of the ball and alter its properties. The distribution of cholesterol deposits in the body suggests that another contributory factor is a locally high oxygen tension. In the arterial system most cholesterol deposits are found in the aorta and adjoining arteries, while in the skeletal system only damaged pneumatized bones are affected.^{2,3}

The combination of a raised blood cholesterol and a high oxygen tension cannot, however, be the whole answer. In five of the cases reported by Pierie *et al.*, where both these factors would seem to have been operating, and in which both aortic and mitral valves had been replaced, the silicone rubber ball in the aortic valve was affected, but not the ball in the mitral valve. One possible difference between these two is in the pressures exerted on the components, but another more probable difference is the surrounding turbulence. This latter is associated with haemolysis, usually at a sub-clinical level.⁴ In those pneumatized bones in which cholesterol deposition occurs there is also red cell destruction. The most probable hypothesis, therefore, at the present time, is that the contributory factors necessary for fat deposition are an excess of the fats concerned, a high oxygen tension, and some chemical from the erythrocytes.

It would be of interest if more information were to be made available of the proportion of silicone-containing prosthetic valves removed at operation or necropsy which have

had their properties significantly altered by deposited and dissolved fats, and the extent to which such observations can be correlated with the previous clinical state. We would be grateful for any information on this and other plastic materials used in heart valves which have had their properties modified in a similar manner.—We are, etc.,

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REFERENCES

- ¹ Pierie, W. R., Hancock, W. D., Koorajian, S., and Starr, A., *Ann. N.Y. Acad. Sci.*, 1968, **146**, 345.
- ² Beaumont, G. D., M.S. Thesis, 1965, Adelaide.
- ³ Beaumont, G. D., *J. Oto-Laryngological Soc. of Australia*, 1967, **2**, 28.
- ⁴ Roeser, W. H. P., Powell, L. W., O'Brien, M. F., *Brit. med. J.*, 1968, **4**, 806.

Traumatic Oedema of the Spinal Cord

SIR,—In dislocations of the cervical spine, even where the cord is not transected, neurological signs of damage frequently appear and progress as oedema occurs in the traumatized area of spinal cord. This oedema leads to ischaemia, and if prolonged to a degree of permanent cord damage.

The use of hypertonic solutions is almost taken for granted in the treatment of cerebral oedema, but is not emphasized in the treatment of the damaged spinal cord. The use of intravenous mannitol infusion in the acute treatment of traumatic paraplegia may produce gratifying results, but obviously early treatment is essential.

A 29-year-old man was brought to casualty, having been found by the police lying unconscious on the ground below the roof of a jeweller's premises. He regained consciousness, and because he developed seesaw respiration with indrawing of the intercostals the anaesthetist was sent for to discuss his respiratory obstruction. X-ray confirmed the clinical diagnosis of dislocated cervical spine, which rapidly became more obvious as quadriplegia with anaesthesia developed.

Mannitol 20 g. was given rapidly intravenously in 20% solution, which elicited a complaint of pain in his other arm. This was the initial return of his sensation, which continued to improve. His cervical dislocation was then reduced under inhalational anaesthesia induced while head halter traction was applied. Cricoid pressure was used to forestall oesophageal regurgitation, and in this situation assisted reduction of the dislocation. Intubation was avoided, as experience with a previous case had shown the difficulty of intubating the larynx while head halter traction is maintained.

His subsequent recovery was complete, and he was discharged fit to return to his occupation.

I feel that the improvement in this man's condition appearing within minutes of starting the mannitol infusion cannot be discounted. Delay would obviously increase the degree, extent, and duration of consequent ischaemia, with irreversible neurological damage. I would advocate the early use of mannitol in the acute treatment of spinal injury, without prejudice to the routine orthopaedic procedures.—I am, etc.,

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Bones in Broadwick Street

SIR,—On 30 January 1969 some workmen engaged in demolition work on a site in Broadwick Street and Marshall Street, London, discovered six human skulls and a number of bones about 9 ft. (2.7 m.) below street level. During the next few days a further three skulls and more bones were brought to light. Broadwick Street, formerly Broad Street, was the centre of the epidemic of cholera which devastated Soho in September 1854, an epidemic which has been described as the most terrible of its kind this country ever knew.

At the time the local people believed that they were being stricken by plague, for they knew that somewhere in the area was a pit in which victims of the Great Plague of 1665 had been buried. Moreover, shortly before the irruption of the cholera a drainage system had been laid through the area, and it was held that the consequent disturbance of the soil had released the plague which had been locked underground for nearly 200 years. It was then that John Snow¹ carried out his well-known investigation and located the cause of the cholera in the water of the well which at that time stood in Broad Street. The removal of the pump-handle, at Snow's request, was symbolic of a new understanding of the nature of disease and marked an important milestone in the history of epidemiology and public health.

At first it seemed likely that the bones which have just been found were those of people who succumbed to the cholera. A study of the site suggests that this is probably not so, for the bones lay beneath the footings of the houses, and the records show that these were originally built between the years 1735 and 1750. Have we found the site of the reputed plague pit? A map of the area prepared for the Vestry of St. James's,² shortly after the cholera epidemic, indicates the extent of what is called "the pest field." This field was bought by the Earl of Craven in 1665 and given for the burial of those who died of the plague. The bodies would have been collected in the City and carted along Oxford Street, through the open countryside, to the field, where they would have been buried in pits dug for this purpose. The location of the pits themselves would probably not have been marked. The site where the bones were found lies within the area of the pest field as shown on the map.

The bones have now been deposited with the British Museum (Natural History), where Dr. K. P. Oakley, deputy keeper of the anthropology subdepartment, is arranging for radiocarbon and other tests to be carried out. These, it is hoped, will determine whether the bones date from the cholera epidemic of 1854 or, as now seems more likely, from the Great Plague of 1665. In either case, their discovery in the heart of London is of great historical and medical interest.—I am, etc.,

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REFERENCES

- ¹ Snow, J., *On the Mode of Communication of Cholera*, 1855. London.
- ² Report to the Vestry on the Cholera Outbreak in St. James, Westminster, by the Cholera Inquiry Committee, London, 1855.