second twenty-four hours. Her condition, however, became worse, and reflex spasms began to occur six hours after admission (thirty hours after the onset of trismus). These were satisfactorily controlled with rectal paraldehyde in doses of 4 drachms in normal saline, and this treatment was repeated when the reflex spasms tended to recur. In spite of the fact that the reflex spasms were comparatively easily controlled her condition steadily deteriorated, her pulse rate and temperature rose, and she died eighty hours after the onset of trismus. She never had a really severe reflex spasm, and death seemed to be due to cardiac failure.

Post-mortem examination showed slight congestion of the brain, lungs, and other organs, but no changes were found to suggest that the cause of death was anything but uncomplicated tetanus. The scar of the old wound was explored but no pus was found.

COMMENTARY

In this case an incubation period of seven days suggested a severe attack. On the other hand, the age of the patient, the slight wound on the foot, and the fact that there was only tonic rigidity without reflex spasms on admission twenty-four hours after the onset were all favourable signs. When, however, reflex spasms began to come on six hours later the prognosis became grave. It has been shown previously that if reflex spasms occur regularly within forty-eight hours of the onset of trismus the chances of recovery are slight and that there is a direct relation between the period of onset (the time between the first onset of trismus and the onset of reflex spasms) and the duration of life. Observations on the 16 fatal cases in a previous series treated only with intravenous antitoxin and sedatives indicated that a period of onset of thirty hours would give a duration of life of between three and five days from the first onset of trismus (Cole, 1940). In the above case death took place eighty hours from the onset. The chances of recovery, however, seemed sufficiently borderline to suggest that an additional effective therapeutic measure might tip the scales in favour of recovery. The fact that sulphapyridine not only failed to do this but did not seem to have any effect whatever on the course of the disease or the duration of life suggests that it is not of much value in tetanus.

It is unwise to draw conclusions from a single case. Further observations are needed, but a small number of cases carefully observed on the above lines would give reliable information. It is important to remember that lobar pneumonia is an occasional complication of tetanus and that sulphapyridine would then be of great value. This case also illustrated the fact that, with present treatment, once a lethal dose of toxin has been absorbed death will take place at the appointed hour, whether or not the reflex spasms are controlled, probably from the effect of toxin on the medullary centres.

LESLIE COLE, M.D., F.R.C.P.,

Physician to Addenbrooke's Hospital, Cambridge.

REFERENCES

- Bryant, J., and Fairman, H. D. (1940). *Lancet*, 2, 263.
Cole, L. (1940). *Ibid.*, 1, 164.
Singer, E. (1940). *Med. J. Austral.*, 1, 796.

Hemiplegia as a Complication of Diphtheria

The following case records the sudden onset of a complete hemiplegia in a child suffering from nasopharyngeal diphtheria, with subsequent recovery.

CASE HISTORY

A girl aged 9, with no previous illnesses of any significance, was admitted to Kendray Isolation Hospital on April 21, 1941, with thick membrane extending from the swollen tonsils on to the soft palate and posterior pharyngeal wall, membrane inside the excoriated nares, sero-sanguineous rhinorrhoea, typical bull-neck, and the usual features of a third-day case of severe nasopharyngeal diphtheria. The heart sounds, though poor, were closed, and the temperature remained below 99° throughout the illness. She received 80,000 units of antitoxin intravenously, cardiac stimulants, and intravenous drips, and, apart from transient palatal paresis from the seventh to the eleventh day in

hospital and gallop rhythm from the eleventh to the fourteenth day, progress was uneventful for seventeen days.

The eighteenth day witnessed the sudden onset of a flaccid paralysis which affected the right side of the face and right limbs, and which was accompanied by double incontinence. There was no coma, but the child was dazed and very weak. Auscultation revealed a loud, blowing systolic murmur, maximal at the pulmonary area. Detailed clinical examination, deferred for thirty-six hours because the girl was so desperately ill, showed that the paralysis was becoming spastic: brisk ankle-jerks, biceps-jerks, and triceps-jerks, ankle clonus, and Babinski's sign; motor function and fine sensation absent, but gross sensation present in the hand; no athetosis; no aphasia, although the child was right-handed. Both knee-jerks were absent, presumably indicating diphtheritic neuritis.

Treatment consisted in nursing care, strapping the side of the face, the provision, later, of a cock-up splint and an adjustable Thomas splint with a foot-rest to combat wrist-drop and drop-foot, and, finally, exercises. Sensation returned speedily; organic reflexes were restored in nine days; the motor nerves recovered slowly, the leg movements, which did not begin until the nineteenth day, being the last to return. The pulmonary murmur soon disappeared. During the period of gradual restoration of function, which was complicated by the recurrence of palatal paresis and by right internal strabismus (both transient), radiography and electrocardiography revealed no cardiac abnormality. After 125 days in hospital the patient was discharged, with all movements present, though weaker than on the unaffected side, and the heart normal to clinical examination.

DISCUSSION

Hemiplegia is usually a sequel to cardiac disease; therefore the pulmonary murmur must be considered first. As this was absent before and after the illness, and as the subsequent normality of the heart was confirmed by radiograph and electrocardiogram, it is necessary to exclude all organic causes, congenital and acquired. It might perhaps be suggested that an acute simple endocarditis, such as occurs after streptococcal infections, had developed at the pulmonary valve, and that subsequent absorption of vegetations had left the heart apparently normal. But to postulate an endocarditis which is completely apyrexial and which clears up in a matter of days carries us beyond the limits of human credulity. Clearly, despite its loudness, the murmur was merely one of those functional murmurs about which much has been written and little is really known. Possibly it was akin to the soft apical systolic bruits often audible in febrile illnesses, or, more probably, since in diphtheria the blood volume is reduced by the passage of plasma into the tissue spaces, it was akin to the haemic murmurs of anaemia. Anyway, it lacked pathological significance.

We must now find a connexion between diphtheritic toxæmia and a lesion of the internal capsule. Hemiplegia can develop after encephalitis (which disease has been described in association with diphtheria and other infections), but there is no evidence on which to base such a diagnosis. Again, it can occur as a complication of meningitis, but meningeal symptoms could hardly pass unnoticed in a hospital for infectious diseases. Moreover, encephalitic and meningitic hemiplegias are typically slow in onset.

Cerebral haemorrhage and tumour being excluded by the child's recovery, the diagnosis seems to lie between embolism and thrombosis, with absence of coma perhaps favouring thrombosis. Since the left side of the heart was not diseased, and since, even if the pulmonary valve had been diseased, an embolus from that valve would have had to traverse the entire pulmonary circulation before entering the systemic blood stream, the most feasible explanation of embolism would seem to be that a minute fragment of aspirated membrane in some way gained entrance to the lung capillaries and travelled onwards with the blood: not impossible, but, surely, most unlikely. The grossly enfeebled circulation and the increased viscosity of the blood as a result of the extrusion of plasma to the tissue spaces make cerebral thrombosis a much more probable diagnosis.

I. A. G. MACQUEEN, M.A., M.B., Ch.B., D.P.H.,
Assistant Medical Officer of Health, Barnsley.