hypertrophic gastritis (Menetrier's disease), in which hypoproteinaemia and a considerable protein loss in the stomach (Schwartz and Jarnum, 1959) is very common. It seems likely that in both cases the loss of protein occurs through secretion of protein-containing fluid into the gastric lumen. In actual cancer of the stomach, hypoalbuminaemia is a common finding and is due, at least partially, to gastro-intestinal loss of protein (Jarnum and Schwartz, 1960).

A conclusion to be drawn from our case is that the gastro-intestinal tract must be examined in all obscure cases of hypoproteinaemia.

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Recurrent Rheumatic Chorea and Optic Atrophy after Occlusion of Central Retinal Artery

The following case is presented to illustrate the association of rheumatic chorea and C.R.A. occlusion in the absence of clinical evidence of carditis.

The patient, a 21-year-old married woman, was admitted to hospital at the beginning of July, 1960, suffering from severe chorea gravidarum. She was in the 23rd week of her second pregnancy and had felt nervous, and had been noticed to be excessively restless, for about two months. She had suffered from mild breathlessness on exertion since childhood, but this was no worse at the time of admission. Her left wrist had been swollen and painful for one week and she complained of a crop of ulcers in her mouth. Foetal movements had been felt for three weeks. Her right eye had been blind since sudden loss of vision in 1957. She had a history of several attacks of acute rheumatism and rheumatic chorea since childhood (see below). One of her sisters had died of acute rheumatism at the age of 7½ years, and a brother had had erythema nodosum as a child. The remainder of her family were healthy.

On admission the patient showed gross choreiform movements of the face, hands, and legs, with some dysarthria, and had a slight, painful swelling of the left wrist and a crop of aphthous ulcers. She was clinically in the 23rd week of a normal pregnancy. The pulse was 80–100 and B.P. 110/70. There was slight clinical cardiac enlargement and a loud apical systolic bruit transmitted up the left sternal border, where it was accompanied by an early diastolic third sound. The second sound was closely split at the base.

The visual acuities were not accurately assessed on admission, but refraction on recovery from the chorea revealed that right vision was reduced to vague hand movements and perception of light in the temporal and inferior fields only. The left vision was improved from 6/60 to 6/6 with $+4.0/+0.50\times90°$. The pupils were equal in size but the right direct and left consensual light reflexes were diminished, the left direct and right consensual reflexes being normal. Fundal examination revealed bilateral hypermetropia and right optic atrophy. The retinal vessels were equal in size in the two eyes and of normal calibre, allowance being made for the hypermetropia.

Nothing significant was found on investigation other than a raised E.S.R. of 54 mm. in one hour (Westergren). The chorea was relieved by bed rest, sedation with phenobarbitone, and full doses of salicylates. The wrist became free from pain and the swelling subsided.

PAST HISTORY

I am grateful to Professor C. B. Perry, of Bristol University, and Mr. B. J. Bowden, of the Bristol Eye Hospital, for supplying the following details of the patient's past medical history and for permitting me to make use of them.

December, 1943 (aged 5 years): Attack of acute rheumatism; no clinical evidence of carditis; 1948: attack of rheumatic chorea; no clinical evidence of carditis; 1954: further attack of rheumatic chorea, again with no clinical evidence of carditis.

March, 1957: Sudden loss of right vision, when seen three days later by an ophthalmologist she was found to have the typical fundal changes of occlusion of the central retinal artery. The condition failed to respond to a retrobulbar injection of tolazoline hydrochloride. A presumptive diagnosis of embolism of the artery was made, though again no clinical evidence of carditis or other embolic phenomena was found. The patient gave a history, at this time, of periodic attacks of transient visual loss, affecting either eye for about a minute at a time, for six years, occurring as often as twice a day or at intervals of several weeks.

October, 1958: Normal delivery of first child after a normal pregnancy, though the patient thinks in retrospect she may have been rather restless during this pregnancy also.

January, 1960: Professor Perry discovered a loud apical systolic murmur for the first time.

COMMENT

The visual loss in the above case did not occur during an attack of chorea, but the rarity of C.R.A. occlusion in a patient of her age (18 years) points to an aetiological association of the condition and her rheumatic diathesis.

The most likely cause of the arterial occlusion would be embolism, but several factors weigh against this possibility: (1) the complete absence of clinical evidence of any form of carditis until almost three years after the visual loss, (2) the absence at the time of the arterial occlusion of other signs of embolic phenomena, and (3) the history of transient attacks of visual loss, culminating in the right C.R.A. occlusion, for a period of six years. The last factor points to these being attacks of spasm of the central retinal artery possibly associated with local arterial disease rather than multiple emboli, as recovery of vision between the attacks was said to be complete.

I thank the Director-General of Medical Services, Royal Air Force, for permission to publish details of this case.

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A new British Standard specification for anti-static rubber flooring (B.S.3398) has recently been issued. Copies may be obtained from the British Standards Institution, Sales Branch, 2 Park Street, London W.1. (Price 4s. each, postage extra to non-subscribers.)