genic organisms (Smadel and Jackson, 1947, 1948; Payne, Knaudt, and Palacios, 1948; Payne, Sharp, and Knaudt, 1948; Smadel, Woodward, *et al.*, 1948; Smadel, Leon, *et al.*, 1948; Pincoffs *et al.*, 1948), including the viruses of lymphogranuloma inguinale, psittacosis, and primary atypical pneumonia, the rickettsias, the enteric, Brucella, and relapsing-fever organisms, as well as a large number of others.

It is therapeutically effective after being given orally, but as it rapidly disappears from the blood frequent doses or high intermittent doses seem required; therapeutic dosage appears free from toxic or undesirable side-effects on man (Ley *et al.*, 1948).

As in the present case, positive stool cultures may occasionally be found in typhoid after a course of chloromycetin, and sometimes relapses with positive blood cultures may occur. Such relapses seem to respond readily to further courses of the drug, and no evidence that the organism becomes resistant has yet been reported.

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Medical Memoranda

A Clinical Note on a Vasovagal Attack of Gowers

Sir William Gowers (1907) described prolonged seizures the symptoms of which consisted "chiefly in disturbance of some of the functions of the hypogastric nerve," usually accompanied by more or less vasomotor upset. Of sudden onset, the symptoms, at first slight, gathered strength rapidly and then slowly receded, an attack seldom lasting less than half an hour in all. To these episodes Gowers gave the name "vagal" (or, when the vasomotor disturbance was much in evidence, "vasovagal") attacks. He added that the word "vagal" was used "only as a convenient descriptive term, bringing together symptoms which cannot be easily included in a useful designation." He believed that these attacks were to be regarded as a protracted form of minor epilepsy, "drawn out, lengthened, as it were, and thereby made less intense, though not less distressing," by the absence of any tendency to termination by loss of consciousness.

Although vasovagal attacks of Gowers's type have an established place in neurological literature they are relatively uncommon, and for this reason may at times be perplexing to those unfamiliar with the clinical picture. The following case report may therefore be of some interest, since it illustrates both the nature of the symptoms and their characteristic pattern.

CASE HISTORY

Shortly after sitting down to dinner one evening early in March, 1947, a healthy man aged 30 became aware of a vague sense of uncertainty and apprehension, which was at first not severe enough to prevent him taking his soup. It became more pronounced, and a feeling of swimminess was added. Thinking he was going to faint, he left the table and lay down on a sofa. Despite this the attack continued to grow in intensity, with mounting mental and physical distress. There was profuse sweating and audible intestinal gurgling, and he felt as though his bowels might be moved even against his will. He observed that his pulse rate had fallen to about 30 a minute (roughly half its normal resting value), the pulse being weak, and there was extreme pallor and coldness of face and hands. At the height of the attack, which occurred a quarter of an hour after its onset, angor animi was a marked feature. At this time the radial pulse was very slow and almost impalpable, and there was profound disinclination to make any movement. A curious hoarseness and weakness in phonation was also experienced, as though, it seems, he was unable to expire forcibly enough to set the vocal cords in full vibration. Parenthetically it may be remarked that no respiratory distress or, apparently, any definite alteration of respiratory rhythm was noticed by the patient at any time in his attack. A slow waning of the attack followed, the terminal stage of its decrescence being accompanied by a subjective feeling of intense cold, with unrestrainable chattering of the teeth and violent shivering, lasting fully ten minutes. The total duration of the attack was between half and three-quarters of an hour.

COMMENT

Unfortunately the patient was not seen during the attack. However, his full and careful account unquestionably established its identity with those described by Gowers: indeed, the similarity to Gowers's second case is strikingly close. Full physical examination the following day revealed no abnormal cardiovascular or neurological signs, and the blood pressure was normal (125/80). An electrocardiogram taken two days later showed a heart rate of 70, with sinus arrhythmia but otherwise normal. The patient experienced infrequent extrasystoles for about a fortnight afterwards, and then these disappeared.

It should be added that there was no personal history of similar attacks in the past, though ten years before, as a medical student on a ward round, he had once nearly fainted. The family history was essentially negative.

It is perhaps significant that the patient had returned from the Tropics only eight weeks before, to meet with the unusually severe cold of the 1946–7 winter. Further, he had had three bouts of coryza in quick succession, the last less than a week before the attack. So far as is known there have been no recurrences.

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Death following Injection of Neostigmine

Neostigmine ("prostigmin") is known to slow the pulse markedly even in the fit patient, and in view of the widespread use of this drug in present-day anaesthetics its effect on a gravely ill patient, as exemplified in the following case, must be seriously considered.

CASE REPORT

A well-built but gravely ill man, aged 38, was admitted with a history of abdominal pain for three or four days. His abdomen was distended, and there was bronchial breathing, with rales at both lung bases. His pulse was 140 and respirations 50 per minute. A careful and experienced house anaesthetist induced anaesthesia with thiopentone 0.35 gr. (23 mg.), and continued with cyclopropane. Despite the patient's poor general condition, tubocurarine, 15, 10, and 5 mg., had to be given to obtain satisfactory relaxation throughout an operation which lasted 75 minutes. Four pints (2.27 litres) of purulent fluid was evacuated through an upper abdominal incision. The stomach was found to be normal, but a perforated gangrenous appendix was revealed, making a second incision necessary. After the peritoneum was sewn up the general condition was no worse than at the beginning of the operation and certainly not immediately alarming. The pulse rate was 100, but because of the curare the tidal exchange was not adequate and respiration had to be "assisted." At this stage 2.5 mg. of neostigmine, together with 1/100 gr. (0.65 mg.) of atropine, was injected through the tubing of the intravenous drip. After one or two minutes, instead of the expected respiratory response, slight cyanosis was noticed, and the pulse, which before the injection was reasonably good, was no longer perceptible. Adrenaline 0.5 ml., injected into the tubing, and cardiac massage were ineffective.

Post-mortem examination revealed collapse of the lower lobes of both lungs, dilatation of the right side of the heart, and generalized peritonitis of a very severe degree. It is improbable that the man would have survived the operation long in any case, but it is not possible to dissociate the unexpectedly sudden death with the intravenous injection given two minutes before.

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