Correspondence

Ogilvie's Syndrome

SIR,—Five cases of intestinal ileus due to sympathetic deprivation have now been described in the *Journal*. Both in Ogilvie's¹ original case and in the later cases^{2,3} the interfering lesion was malignant invasion of the posterior abdominal wall. Although "Ogilvie's syndrome" has not been precisely defined, it is suggested that the essential feature is the production of intestinal ileus by sympathetic involvement rather than the malignant nature of the causative lesion. The following case is therefore suggested as an example of the condition, although the "invasion" of the posterior abdominal wall was due to a haemorrhagic, and not a neoplastic, process.

The patient, a man of 60 years, was submitted to herniotomy for a right inguinal hernia of two years' duration. The operation was performed under spinal anaesthesia. An indirect sac was found and excised. Immediate post-operative progress was uneventful, but on the fifth day he commenced vomiting copiously, and this continued with rapid deterioration of his general condition until death occurred 11 days after operation. During this time the patient was constipated, but passed much rectal flatus. Blood urea on the day preceding death was 364 mg. per 100 ml.

Post-mortem Findings.—The body was that of a very obese man, with a barrel-shaped chest and severe finger clubbing. The heart showed moderate left ventricular hypertrophy. The lungs showed terminal congestion and oedema superimposed on chronic bronchitis and emphysema. The pleural cavities were almost obliterated by old fibrous adhesions. A large extravasation of blood was present under the peritoneum throughout the lower half of the right anterior abdominal wall, spreading round into the posterior abdominal wall and also projecting downwards into the right side of the pelvis and passing upwards to form a large retroperitoneal haematoma 6 in. by 1.5 in. (15 by 3.75 cm.) in front of the right psoas sheath. The operation site appeared to be normal, there being no collection of blood in the subcutaneous, fascial, or muscular layers. The stomach showed slight congestion. The small intestine showed great dilatation of its entire length, with the exception of the terminal 6 in. (15 cm.) of ileum, which was involved in the extravasation into the posterior abdominal wall but which showed no organic obstruction. The colon showed moderate dilatation down to the lower sigmoid portion, the distension being gaseous, with only a small amount of faeces present. No twisting, bands, masses, stricture or faecal collections were present to account for organic obstruction.

This case occurred in June, 1947, before Ogilvie's syndrome was described. At this time the ileus was attributed to interference with the nerve supply to the intestine, but it was not decided whether this was due to sympathetic involvement by the haematoma of the posterior abdominal wall or to local interference with the nerves of the terminal part of the ileum which were directly involved in the extravasation. In view of the viability of the affected portion of gut and the absence of apparent interference with local blood supply it may be considered unlikely that the local nerves would have suffered selective mechanical damage, and it is now thought more probable that the "invasion" of the posterior abdominal wall by the haematoma might have been responsible for the ileus by a similar mechanism to that described by Ogilvie. Unfortunately no detailed dissection of the abdominal autonomic system was performed.—I am, etc.,

Grimsby.

L. M. GERLIS.

REFERENCES

- ¹ *British Medical Journal*, 1948, 2, 671.
- ² Dunlop, J. A., *ibid.*, 1949, 1, 890.
- ³ Handley, R. S., *ibid.*, 1949, 1, 891.

Research in Renal Disease

SIR,—Writing as an obstetrician with a keen interest in renal problems, I wish to add to the support already given to Dr. Osman's major thesis (May 21, p. 910). Specializing in clinical nephrology is in general justified, (1) by the vastness of the subject and of its literature, and (2) by the wide prevalence and often serious character of renal disorder.

In my own particular field I have long been concerned with the renal changes of pregnancy, but despite extensive reading

and consultation with others I still lack final answers to many of my questions in the fields of renal anatomy, physiology, pathology, and so on. This experience, which I must share with many others, is in part due to the fact that there are few with sufficiently full time for the renal work, and in part to the fact that many gaps still remain in our knowledge which are all too slowly being closed by research.

An improvement can readily be made and maintained if knowledge is synthesized and further research encouraged in such a centre as, I imagine, Dr. Osman envisages. Then the lonely furrow which he himself has ploughed for so many years will be joined by several new ones, and the consequent harvest should be great. With all due deference I should like to suggest that the three Royal Colleges could find in the synthesis of knowledge about the kidney, and encouragement of research on the problems presented by it, a very worthy field for a joint effort. The synthesis of existing knowledge, as a beginning, could result in the production of a very much-needed book, in which the artificial separation of the three aspects of the subjects could be overcome in the ultimate interest of the patient. The encouragement of research would be a longer-term policy, but the initial consideration of it could be undertaken without much delay.—I am, etc.,

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JOHN SOPHIAN.

Death following Neostigmine

SIR,—In the cases described by Professor R. R. Macintosh (May 14, p. 852) and Dr. J. Clutton-Brock (June 4, p. 1007), though it was a mixture of neostigmine and atropine which was injected, death appears to have been attributed exclusively to the neostigmine. The atropine is presumably incorporated in such injections to diminish the cardiac and other untoward effects of the neostigmine. In fact, however, it is quite likely to do the reverse, and we feel that this should be pointed out.

Though we have no experience of intravenous atropine there is no doubt from numerous controlled experiments on healthy people that the primary effect of a subcutaneous dose of atropine sulphate (0.5 mg. per 50 kg. b/w) is to slow the pulse, sometimes very markedly, by what we have always supposed to be medullary (cardiac vagus centre) stimulation. This effect is well known to pharmacologists. It appears quickly and is at its maximum about 10–15 minutes after the injection. The pulse rate then either returns to normal and remains there, or it may rise significantly above normal for a variable period. The rise above normal is, of course, due to the peripheral blocking effect of the drug on the muscarinic action of the acetylcholine liberated by the tonic activity of the cardiac vagus : this effect, with the dose used, is usually slight.

Thus atropine administered subcutaneously first increases the effect of the cardiac vagus : this action either ceases or, more likely, is masked by the subsequently established anti-acetylcholine effect of the drug. Though there is considerable individual variation in the degree of response in such experiments there can be no doubt about the sequence of events.

It therefore seems likely that a similar primary vagus stimulation with secondary partial vagus blocking may occur when atropine is given intravenously and that the primary effect in these circumstances would begin almost at once. If such central stimulation does occur on intravenous injection of atropine, then, clearly, its effects will be markedly potentiated by neostigmine. This would also apply to any central action of atropine tending to increase the tone of bronchiolar muscle. Hence atropine, administered simultaneously with neostigmine, far from preventing the unwanted effects of the latter drug may, at first, anyhow, be more likely to increase them. This may have been an important factor in causing death in the two cases cited.

Apart, however, from the possibility of such a potentiation of the effects of neostigmine arising from the central action of atropine, the peripheral anti-acetylcholine effects of therapeutic doses of atropine take some twenty or thirty minutes to be fully established. It is, therefore, not to be expected that the unwanted cardiac and bronchial action of neostigmine would be prevented by giving the atropine along with it. The importance of this point is increased by the known liability of both tubocurarine and neostigmine to produce bronchoconstriction by peripheral mechanisms.