1955; Grytting, 1954; Hartnett, 1955; Loftus *et al.*, 1955; Lomas *et al.*, 1955; Van Ommen and Brown, 1955; Whitfield, 1955; Zatuchni and Miller, 1954). The main points on which comment seems justified at present are:

1. The jaundice is apparently *not* a direct toxic action of chlorpromazine on the liver. This is supported by (a) the demonstrated lack of correlation between the amount of chlorpromazine ingested and the occurrence of jaundice; (b) the absence of liver damage in patients who have taken large amounts of chlorpromazine in suicidal attempts (Deniker, 1954; Aubertin *et al.*, 1954; Vallat and Lepetit, 1954); (c) the failure to induce liver damage in experimental animals given large doses of chlorpromazine over long periods (Courvoisier *et al.*, 1953; Smith, Kline, and French, 1954, personal communication); (d) the failure to demonstrate reduction in the oxygen consumption of isolated rat-liver slices exposed to chlorpromazine (Peruzzo and Forni, 1953); (e) the observation that in some patients jaundice is not apparent until some time after the drug has been stopped.

2. Older people are more likely to develop jaundice than young ones, and females more than males.

3. The mechanism of jaundice in our Case 2 and in other recorded cases is intrahepatic biliary obstruction (Sherlock, 1955). In some cases the bile ducts are apparently compressed by swollen fat-filled liver cells, and the cause of fat storage in these cases might be worth investigating. It seems necessary to explain why, on the one hand, jaundice does not appear until after chlorpromazine therapy has been stopped, and, on the other hand, why the jaundice can recover even although chlorpromazine is continued. The sex incidence of this complication also awaits explanation.

Conclusion

The incidence of jaundice in patients receiving chlorpromazine therapy is about 1%, and many cases are mild. However, in circumstances not clearly delineated severe or fatal liver disease can occur. It would seem wise to avoid chlorpromazine therapy in the following circumstances: (1) when infective hepatitis is endemic; (2) in the presence of liver disease; (3) in malnourished patients; (4) when other potentially hepatotoxic agents have been used; and (5) when oestrogens have been given. Should a pyrexial illness occur during chlorpromazine therapy evidence of liver damage should be sought. If it is found, chlorpromazine and all other drugs should be withdrawn and a high-protein high-calorie diet given.

This advice is largely empirical, and more clinical and experimental information is required before the problem of jaundice and chlorpromazine is elucidated.

Summary

During the treatment of 26 patients with small doses of chlorpromazine for relief of vomiting, three cases of jaundice occurred; a fourth case was referred from a psychiatric unit. The clinical features of these cases are analysed together with 39 additional cases, information about which was obtained by questionnaire. The incidence of jaundice in chlorpromazine therapy was about 1%. Jaundice was more common in females than in males, but bore no relation to the dose and duration of therapy. Cases varied in severity from mild and transient jaundice to severe, prolonged, and occasionally fatal illness. It is advised that chlorpromazine be avoided in the presence of epidemic hepatitis, in patients with liver disease, and in combination with other potentially hepatotoxic agents.

We wish to thank Professor S. Alstead, Mr. J. P. Galloway, and Dr. A. S. Rogen, under whose care many of these patients were treated; Dr. Hunter Gillies, Dr. W. Mayer-Gross, Dr. A.

MacNiven, Dr. A. Milne, and Professor Ferguson Rodger, who kindly replied to our questionnaire; the many physicians who completed the questionnaire from Messrs. May and Baker for their permission to use the data in this analysis; and Dr. J. Harper, Dr. R. Forgan, and Mr. Baines, of Messrs. May and Baker, for their courtesy and co-operation throughout.

REFERENCES

- Aubertin, A., Martin-Dupont, C., and Tavernier, J. (1954). *Presse méd.*, 62, 752.
 Boardman, R. H. (1954). *British Medical Journal*, 2, 579.
 Courvoisier, S., Fournel, J., Ducrot, R., Kolsky, M., and Koltschet, P. (1953). *Arch. int. Pharmacodyn.*, 92, 305.
 Deniker, P. (1954). Quoted by H. Azima, and W. Ogle, *Canad. med. Ass. J.*, 1954, 71, 116.
 Earle, B. V. (1955). *British Medical Journal*, 1, 1156.
 Grytting, G. (1954). *Nord. med.*, 52, 1699.
 Hartnett, B. S. (1955). *British Medical Journal*, 1, 1458.
 Loftus, L. R., Huizenga, K. A., Stauffer, M. H., Rome, H. P., and Cain, J. C. (1955). *J. Amer. med. Ass.*, 157, 1286.
 Lomas, J., Boardman, R. H., and Markowe, M. (1955). *Lancet*, 1, 1144.
 Peruzzo, L., and Forni, R. B. (1953). *Presse méd.*, 61, 1463.
 Sherlock, S. (1955). *Diseases of the Liver and Biliary System*. Oxford.
 Vallat, J.-N., and Lepetit (1954). *Presse méd.*, 62, 752.
 Van Ommen, R. A., and Brown, C. H. (1955). *J. Amer. med. Ass.*, 157, 321.
 Whitfield, A. G. W. (1955). *British Medical Journal*, 1, 784.
 Zatuchni, J., and Miller, G. (1954). *New Engl. J. Med.*, 251, 1003.

HYSTERICAL ABDOMINAL PROPTOSIS

RECORDS OF A CASE

BY

M. R. EWING, F.R.C.S.

Senior Lecturer in Surgery, Postgraduate Medical School of London, W.12

AND

J. H. DEREK MILLAR, M.D., F.R.C.P.Ed.

Consulting Physician, Grimsby and District Hospital

Our attention was recently drawn, for the first time, to a most interesting paper by Roussak (1951) on a clinical condition which he called "hysterical abdominal proptosis." On reading it we were reminded vividly of a patient who was under our care two years ago and who presented an interesting problem in diagnosis and in management.

Case Report

A short, thick-set, rugged, placid trawlerman aged 55 first came under the care of one of us (J. H. D. M.) on account of shortness of breath and of abdominal distension. Although he was acutely dyspnoeic there was no cyanosis. His heart was apparently normal, as was an x-ray film of his chest, although on clinical examination there was poor aeration at the bases. The most striking feature on physical examination, however, was the prominence of the abdomen, which was grossly distended and tympanitic all over. Despite a diligent search, no cause for this distension was discovered, and at a subsequent laparotomy there was no hint of intestinal obstruction. However, as soon as anaesthesia was induced the abdominal swelling collapsed in a most dramatic fashion, only to reappear with the return of consciousness. A second anaesthetic was administered during his convalescence and with precisely the same result (Fig. 1).

He was later admitted to Hammersmith Hospital, where it was thought possible that his dyspnoea might be due to a slight degree of left ventricular failure. While a variety of investigations were being completed he continued to complain of abdominal distension, which remained unchanged for days on end. There was no disturbance of bowel function or any colic or vomiting. A straight x-ray film of the abdomen showed only the normal amount and distribution of intestinal gas, and the diaphragm was at its proper level and moved reasonably well. However, as soon as

FIG. 1.—Photographs to show appearance of abdomen before (A) and after (B) induction of anaesthesia with intravenous thiopentone sodium and scoline to complete respiratory paralysis.

FIG. 2.—Tracings of lateral x-ray films of the abdomen before (A) and after (B) the induction of general anaesthesia. Coincident with the collapse of the distension, there is an appreciable lessening of the degree of lumbar lordosis. (The lateral films were so dark as not readily to lend themselves to reproduction.)*

anaesthesia was induced (using thiopentone) the diaphragm was seen to ascend, and at the same time, and without the discharge of any gas from either end of the intestinal canal, the abdominal girth quickly lessened from $40\frac{1}{2}$ to 37 in. (103 to 94 cm.) (Fig. 2). As consciousness returned, so did the distension.

While we were still debating how best to manage this distressing complaint, and before any treatment could be instituted, he suddenly announced that his abdomen was quite flat, for the first time for over a year. It was, in fact, as soft and flabby and easy to examine as it had been earlier under general anaesthesia. He seemed to be as delighted as we were, and forthwith took his discharge home.

We were much intrigued by this illness, which seemed clearly to be psychosomatic in nature, and in searching

round for a possible precipitating cause it was discovered that he had five months previously received an injury at the fishing grounds which he believed to have induced an inguinal hernia. This was repaired surgically and at the same time an operation was carried out on the opposite testis. Some complication ensued, for five weeks later he made a second trip to the operating theatre. On direct questioning he freely admitted that he was greatly dissatisfied with the terms of the settlement which had been offered to him in compensation for this injury: it was also clear that his inability to work was denying him the share of the rich earnings which may (in good times) be enjoyed by deep-sea fishermen.

Comment

Cases of this sort are by no means uncommon: most clinicians of experience will be able to call several to mind. There has been recently a precisely similar case in the surgical practice of Hammersmith Hospital. There is a considerable literature on "pseudocystitis," on "phantom tumour," and on "tumeur en accordéon," and the writings of Alvarez (1949) on this subject are familiar to most specialists in gastro-enterology. He called it "the hysterical type of non-gaseous abdominal bloating." We still feel justified, however, in presenting this single case, in the belief that the condition is not nearly so well known as it should be—a belief that receives some support from the large number of the recorded cases that have been submitted to one, two, or even very many unnecessary surgical explorations.

The important clinical features are as follows: (1) The presence of abdominal distension for which there is no obvious cause. (2) There is no excess of intestinal gas. (3) The distension disappears under anaesthesia, and as it subsides there is no discharge of gas upward or downwards. (4) Often there is an obvious lumbar lordosis, so that a hand can be slipped freely under the spine when the patient is recumbent. (5) Even when the diaphragm is not obviously at a low level it will almost always be seen to rise as the distension subsides. (6) The presence of one or more laparotomy scars is a common finding. (7) The male enjoys no immunity to this complaint. (8) The giving of an anaesthetic (as in pseudocystitis) is immensely valuable in diagnosis: it may also have some merit in treatment. (9) A diligent inquiry will often bring to light some possible precipitating cause.

The abdominal protrusion is due, no doubt, to contraction of certain groups of muscles of the abdominal wall and to relaxation of others. This is often accompanied by the development of a lumbar lordosis and by descent of the diaphragm. Most muscular healthy males can push out the abdominal wall, but the effort can seldom be sustained for long even in the presence of the quietest respiration. In the hysterical subject it is unaffected by a cough or by a sneeze, nor by emptying the bladder or by straining at stool.

On treatment we have no comment to make other than to draw attention to the obvious shortcomings of laparotomy as a cure. The first is all too often the prelude to a second.

Our attention was first drawn to this clinical syndrome by Mr. Ralph Shackman and Dr. M. D. Milne. For the photographs and x-ray films we are indebted to Dr. K. O. Harrison and Dr. R. E. Steiner respectively.

REFERENCES

- Alvarez, W. C. (1949). *Arch. intern. Med.*, **84**, 217.
Roussak, N. J. (1951). *Gastroenterology*, **17**, 133.