in practice this does not always lead to trouble, the precaution may reasonably be taken of leaving sutures in place for longer periods than usual when operations are performed on patients under the influence of these hormones.

Conclusions

It is clear from these considerations that cortisone and A.C.T.H. are not the ideal therapeutic answers to chronic disabling diseases. The greatness of their discovery lies rather in the future possibilities of therapy which they have revealed. Yet in some diseases and especially in rheumatoid arthritis, they provide for some cases the most effective therapy at present available. When their prolonged administration is tolerated they may, by restoring working capacity, even now prove an economical form of treatment. In other cases their administration raises more problems than it solves. In any individual it is to be remembered that the assessment of their usefulness and practicability as therapeutic agents can only be made

by experiment; the possibility that the experiment may have to be terminated must be faced by both doctor and patient from the start. Thus at the present time the decision whether or not to use cortisone or A.C.T.H. as a means of therapy in an individual case is not an easy one. Much fundamental work needs to be done before a rational basis for their use in chronic diseases is achieved.

BIBLIOGRAPHY

- BOLAND, E. W. (1951), *Brit. med. J.*, **2**, 191.
 HART, P. D., and REES, R. J. W. (1950), *Lancet*, **2**, 391.
 HENCH, P. S. (1952), *Ann. intern. Med.*, **36**, 1.
 HENCH, P. S., KENDALL, E. C., SLOCUMB, C. H., and POLLEY, H. F. (1949), *Proc. Mayo Clin.*, **24**, 181.
 HENCH, P. S., KENDALL, E. C., SLOCUMB, C. H., and POLLEY, H. F. (1950), *Arch. intern. Med.*, **85**, 545.
 KASS, E. H., INGBAR, S. H., FINLAND, M. (1950), *Ann. intern. Med.*, **33**, 1081.
 LE MAISTRE, C. A., TOMPSETT, R., MUSCHENHEIM, C., MOORE, J. A., and McDERMOTT, W. (1951), *Proceeding of the Second Clinical A.C.T.H. Conference*, Vol. 2, p. 363. J. A. Churchill, Ltd., London.
 TROTTER, W. (1935), *Brit. med. J.*, **2**, 609.
 WARD, E., SLOCUMB, C. H., POLLEY, H. F., LOWMAN, E. W., and HENCH, P. S. (1951), *Proc. Mayo Clin.*, **26**, 361.

DELAYED HAEMORRHAGE FOLLOWING TRAUMATIC RUPTURE OF THE NORMAL SPLEEN SIMULATING SPONTANEOUS RUPTURE

By R. W. BAILLIE, M.B., F.R.C.S.E.

In writing of spontaneous rupture of the normal spleen, Hamilton Bailey stated: 'Forgotten trauma should be accepted as the cause. If that theory is discarded, we are plunged into a morass of speculation in which it is unprofitable to linger.'

The following case is recorded because it illustrates so well the truth of the above statement. In it rupture of the normal spleen followed upon an injury so insignificant as to be immediately forgotten by the patient, not associated by him with the subsequent abdominal catastrophe and, indeed, which he could only recollect after some days' reflection.

The patient, an athletic-looking young man of 31 years, was admitted in the early hours of the morning of January 8, 1950, as a perforated peptic ulcer. He complained of waking up on the

morning of the previous day at 8 a.m., i.e. 20 hours previously, with severe central abdominal pain which spread all over the abdomen and radiated into the base of the neck posteriorly. The pain eased off somewhat by midday, but became worse again in the evening. He had vomited twice during the course of the day and in the evening he fainted once.

There was no previous history of indigestion, but he had suffered from right renal colic two years before and his appendix had been removed at about the same time.

On examination, the temperature was 98.2° F. and the pulse varied between 80 and 92 per minute. The patient showed no gross pallor, but was extremely distressed, the pain being so severe that he was unable to lie down flat on the bed, and

so abdominal examination was very difficult. Palpation revealed absolute rigidity of the upper abdomen, but no marked tenderness; the lower abdomen could be got to relax a little and then also showed little tenderness. Liver dullness appeared to be diminished, but there were well-marked peristaltic sounds over the abdomen.

Morphia, gr. 1/6, was then administered by the intravenous route; even after this the patient found it very difficult to lie supine and further examination only served to confirm the above findings.

In diagnosis it was considered that there was no general peritonitis present and that the most likely cause was a volvulus of small intestine or a high small intestinal obstruction from adhesions. Acute pancreatitis was thought very unlikely in view of his age.

Operation was performed about an hour after admission, a general anaesthetic being given by Dr. Emerson using curare, pentothal, gas, oxygen and ether. An upper midline incision was used and the peritoneal cavity was found to be full of blood. A hand was then inserted to palpate the spleen, the only likely source of such a haemorrhage, and a large haematoma was found on its convex border where it felt unduly friable, and the capsule seemed to be stripped in this area. Free bleeding was continuing and the patient's condition became very critical, Trendelenburg's position being adopted and transfusion with Group 4 blood started. The splenic vessels were controlled with the fingers and the spleen mobilized until a mass ligature could be placed round its pedicle. In doing this a small left trans-rectus extension of the incision was found necessary. The vessels in the pedicle were now ligatured individually and the wound closed. The patient's condition was now much improved, two pints of blood having been given and a further pint was given after his return to the ward.

The post-operative course was very satisfactory, penicillin being exhibited for a few days.

When questioned three days after his operation, the patient had no recollection of any accident or injury prior to operation, but two days later he remembered an episode which had occurred on the day prior to his illness; when returning home with some friends about 10 p.m. he walked into a 'Belisha Beacon,' which struck him fairly and squarely down the centre of the trunk and winded him for a few seconds, after which he felt all right and went home to bed.

Pathological Report (Dr. J. Carr Brundrett)

The spleen was little, if at all, enlarged; the capsule had been stripped from the pulp on the external aspect as if by a subcapsular haemorrhage.

There was a 3-in. linear rupture of the pulp running longitudinally and in the centre of this a 1-in. diameter mass of blood clot.

Section shows no pathology of the spleen beyond the effects of trauma. The contained clot may be two to three days old, but this is difficult to assess in view of the autolytic changes from failure of penetration by fixative into intact specimen.

Discussion

Cases of so-called spontaneous rupture of the normal spleen have rarely been diagnosed as ruptures pre-operatively and in this connection, in this patient, there was one very significant symptom, namely, fainting. This symptom has been rarely volunteered by the patient, but it was mentioned by Hamilton Bailey and was present in Black's case and those of Galloway, Watson and Ferdiber, whilst Coleman's patient felt fainty and dizzy, otherwise the literature contains little record of it. Fainting, which is here a sign of rapid blood loss, is probably a fairly common symptom in any acute intraperitoneal haemorrhage and in the female is a well-known and important symptom in ruptured ectopic gestation. When it occurs in the male with acute abdominal symptoms where no history of injury has been given, it should raise the question of splenic rupture as the most common cause of intraperitoneal haemorrhage in these circumstances.

In discussing the aetiology of spontaneous rupture in the normal and pathological spleen, splenic congestion secondary to portal congestion was thought by Susman to be an important factor, and he describes the probable sequence of events leading to rupture. In this connection Besnier's aphorism, quoted by McIndoe, that 'traumatism plays a part in spontaneous rupture of the spleen and the spleen is ready to rupture when the trauma occurs' is applicable, the splenic congestion being the most likely factor that makes the spleen ready to rupture. Portal congestion may be either physiological, e.g. just before a meal or with the stomach very full, or pathological.

Zuckerman and Jacobi reviewed 28 cases and inferred that sex had no significance in the occurrence of spontaneous rupture of normal spleens (16 males to 12 females), but a limited review of the literature since their case was published has revealed 15 cases (Galloway, Thomas, Coleman, Watson and Ferdiber, McLachlan, Brines, Jones, Johns, Babson and Morgan, Silverman and Randazzo, Druitt, Schomaker and Gates Harper and Huertas), of which those of Johns, Jones and one of the two cases of Schomaker and Gates are not definitely non-traumatic in origin. Thirteen of these 15 cases were male subjects, a

very high male incidence, and even after adding these cases to those of Zuckerman and Jacobi the incidence is still two males to one female. McIndoe and later Zabinski and Harkins in delayed rupture following trauma noted a very high male incidence, and McIndoe attributed this to the greater liability of males to injury by virtue of occupation, but this would not explain the male incidence in cases of so-called spontaneous rupture where there has been no history of injury or it has been of a very minor nature. A likely explanation would seem to be the fact that males are abdominal breathers to a much greater extent than females and the spleen, being a direct relation of the very mobile diaphragm, may be caught between the abdominal wall, the abdominal viscera and the contracted diaphragm and will suffer accordingly, especially if congested or stuck down by adhesions when subjected to even minor trauma or bodily stresses. Schomaker and Gates think that increased intra-abdominal pressure is a factor and a case was recorded by Druitt following defaecation, though this may have been only the immediate precipitating factor. Babson and Morgan thought that cough may have been the responsible factor in one of their cases. In the case here described a similar mechanism would appear to have been responsible.

In this case it is interesting to speculate on whether the patient, an intelligent active young man, would have recollected the injury at all if the actual rupture had been delayed for a week or two. The period of 'symptomatic silence' or latent period' of Baudet in his case was very short, about 10 hours as compared with the arbitrary period of 48 hours taken as a minimum by McIndoe.

Summary

A case of delayed haemorrhage following traumatic rupture of the normal spleen has been described.

The injury that apparently caused the rupture was so trivial that it was for some time forgotten by the patient and the case was at first considered one of spontaneous rupture. It seems likely that in many cases of apparently spontaneous rupture the injury which caused the rupture has been forgotten.

The aetiology has been discussed and an attempt made to correlate the male incidence with the mechanism of production.

Since writing the above article a further case of spontaneous rupture of the spleen has been recorded (Nicoll, J. A. V. (1952), *Brit. med. J.*, i., 801) in a male patient aged 46, who exhibited fainting after the onset of abdominal pain.

BIBLIOGRAPHY

- BABSON, W. W., and MORGAN, P. (1946), *Amer. J. Surg.*, 72, 97.
 BAILEY, H. (1927-8), *Brit. J. Surg.*, 15, 40.
 BLACK, J. M. (1933), *Ibid.*, 20, 79.
 BRINES, O. A. (1943), *Arch. Path.*, 36, 136.
 COLEMAN, A. H. (1939), *Brit. J. Surg.*, 27, 173.
 DRUITT, A. W. N. (1947), *Lancet*, ii, 652.
 GALLOWAY, W. D. (1935), *Brit. J. Surg.*, 23, 235.
 HALLIWELL, A. C. (1933), *Brit. med. J.*, i, 919.
 HARPER, J. A., and HUERTAS, J. (1950), *Canad. med. Assoc J.*, 63, 169.
 JOHNS, W. A. (1944), *South. med. & Surg.*, 106, 478.
 JONES, L. E. (1944), *Brit. med. J.*, ii, 561.
 MCINDOE, A. H. (1932), *Brit. J. Surg.*, 20, 249.
 McLACHLAN, A. D. (1943), *Ann. Surg.*, 117, 476.
 SHEPHERD, J. A. (1949), *Brit. med. J.*, i, 1083.
 SCHOMAKER, T. P., and GATES, C. Y. (1948), *Amer. J. Surg.*, 76, 319.
 SILVERMAN, I., and RANDAZZO, A. P. (1946), *Arch. Surg.*, 53, 355.
 SUSMAN, M. P. (1927), *Brit. J. Surg.*, 15, 47.
 THOMAS, G. B. (1935), *Brit. med. J.*, ii, 1100.
 WATSON, J. R., and FERDIBER, M. (1942), *J. Amer. med. Assoc.*, 120, 690.
 ZABINSKI, E. J., and HARKINS, H. N. (1943), *Arch. Surg.*, 46, 186.
 ZUCKERMAN, J. C., and JACOBI, M. (1937), *Ibid.*, 34, 917.

HOW TO GET THERE

An Address Book for the Medical profession, showing how to reach the various Colleges, Societies, Institutes and Hospitals in and near London.

Third edition: 1950.

Published by the

FELLOWSHIP OF POSTGRADUATE MEDICINE

60 Portland Place, London, W.1

Price 2s. 6d.