Ascaris ova were found in the stools in all cases; *ancylostoma* were also present in 13 cases; *Giardia lamblia* in four cases, and *Hymenolepis nana* in three cases.

The following three dosage schedules were applied: (1) 0.3 g. (one tablet) a day for each year of life up to 6 years, and 1.8 g. (six tablets) a day from 6 years upward. This was given to 25 cases. Five children received the dosage for seven days, six for five days, and 14 for three days. (2) 0.6 g. a day for each year of life up to 6 years, and 3.6 g. a day from 6 years upwards—that is, double the dosage administered to Group 1. Twenty-one cases, including two failures from the previous group, were treated for one day. Eleven cases, including four failures from the preceding cases, were treated for two days. (3) 0.75 g. a day for each year of life up to 6 years, and 4.5 g. a day from 6 years upwards. Thirty-five cases, including one failure from the second group, were treated for one day.

Treatment consisted solely of the administration of tablets of piperazine adipate as described above; no special diet was recommended during treatment. No pre-treatment measures were undertaken. The daily dose of the drug was given in three or four equal portions during the day, at four-hourly intervals, after meals. A mild saline purge was given on the day after the completion of treatment.

Results

Group Treated with Schedule 1.—The five cases treated for seven days and the six cases treated for five days were completely cured. Most of the worms were expelled on the second and third days of treatment. The worms were intact; some were still living. *Ancylostoma* infestation persisted in four cases. Twelve of the 14 cases treated for three days were completely cured, giving a cure rate of 85.7%. In one case the first stool examination two days after treatment still showed a few *ascaris ova*, but was subsequently negative. This patient passed only one *ascaris* worm on the fourth day from the start of treatment. Another patient passed many *ascaris* worms from the third to eighth day; stools, however, still contained *ascaris ova*. A second similar course was given seven days after the first course, but no worms were expelled and stools were still positive for *ascaris ova*. In two cases *ancylostoma* and *Giardia lamblia* infestations were not affected.

Group Treated with Schedule 2.—Of the 21 cases given the treatment for one day 17 were completely cured, including the two failures from the previous group, showing a cure rate of 80.9%. The same course was repeated after seven days in three out of the four failures in this series, and though they again passed *ascaris* worms the stools still contained *ascaris ova*. The fourth case in which the infection persisted passed 72 *ascaris* worms on the first and second days of therapy. In six cases *ancylostoma* and *G. lamblia* infestations were not affected. Among those treated for two days (11 cases) there was only one failure, the cure rate being 90.9%. The failure responded to schedule 3 dosage (see below). In two cases *ancylostoma* and *G. lamblia* resisted treatment.

Group Treated With Schedule 3.—All the 35 cases in this group were completely cured, including the resistant case which had previously received three courses. In seven cases *ancylostoma* and *Hymenolepis nana* were not affected.

Side-effects.—No toxic effects or side-reactions were noted in any of the cases, although some of them received repeated courses of treatment. There was no demonstrable effect on the kidneys, the circulatory system, or the blood picture.

Summary and Conclusions

A series of 85 children (47 males and 38 females), aged from 1½ to 12 years, suffering from *ascaris* infestation were treated with piperazine adipate tablets, each containing 0.3 g., according to the plan described.

As a result it can be definitely stated that piperazine adipate is a very effective and safe drug for the treatment of *ascaris* infestation in children. The recom-

mended schedule, especially for wide use, is a dose of 0.75 g. for each year of life up to 6 years and 4.5 g. from 6 years upwards, to be given for one day in four equal doses at four-hourly intervals, after meals.

Piperazine adipate was given in the form of tablets, which were easily taken by children. The tablets were administered whole in most cases, but in some it was necessary to crush them; this presented no difficulty. The tablets were not objected to by the children whether given whole or crushed.

The piperazine adipate used in these investigations was kindly supplied by British Drug Houses Ltd. as "entacyl" tablets.

REFERENCES

- Brown, H. W. (1954). *J. Pediat.*, 45, 419.
— and Sterman, M. M. (1954). *Amer. J. trop. Med.*, 3, 750.
Davies, M. T., et al. (1954). *J. Pharm. Pharmacol.*, 6, 707.
Fayard, C. (1949). *Thèse Paris*, No. 889.
O'Brien, D. P. (1954). *British Medical Journal*, 2, 246.
Rearden, J. (1954). *Ibid.*, 2, 872.
Savaton-Pillet, J. (1954). *Nouveautés méd.*, 3, 283.
Sims, S. R. (1953). *British Medical Journal*, 2, 1432.
Sloan, J. E. N., et al. (1954). *J. Pharm. Pharmacol.*, 6, 718.
White, R. H. R. (1954). *Lancet*, 2, 315.
— and Standen, O. D. (1953). *British Medical Journal*, 2, 1272.

Medical Memoranda

Ferrous Sulphate Poisoning with Gross Stricture of the Stomach

To illustrate the destructive effects following ingestion of ferrous sulphate pills, and to stress further the criminal negligence in leaving such pills within the reach of small children, the following case is briefly reported.

CASE REPORT

A male child of 20 months swallowed approximately 50 "fersolate" tablets and was admitted to hospital two hours later. An unrecorded proportion of the pills was recovered by immediate gastric lavage. There was repeated and severe haematemesis. The initial collapse was treated successfully by blood transfusion, but during the next few days there was a steady deterioration in the general condition, and it was not possible to maintain nutrition adequately by mouth because of repeated vomiting. When seen seven weeks after the onset of illness the child was found to be emaciated and dehydrated.

A barium meal showed a smooth tubular stricture affecting the middle third of the stomach (see Fig.).

The child was transferred to a paediatric unit in the hope of improving his nutrition and of preparing him for operation. Despite feeding by an intragastric tube, which was passed beyond the obstruction, and by the intravenous

route, there was a progressive decline, and at no time did he seem fit for surgery. A further barium meal showed progressive and irregular stricture formation. He died 16 weeks after poisoning.

The necropsy showed dense adhesions in the upper peritoneal cavity. A small pouch of healthy stomach proximally

and a normal pyloric antrum were found. The intervening stomach showed gross inflammatory changes, and a narrow channel was lined with granulations, ulcerated and adherent to surrounding organs. A large chronic ulcer penetrated the liver at one point. The muscle wall was replaced by fibrous tissue. The liver and kidneys were normal. These appearances confirmed that excision of the stomach would have been almost impossible in the last weeks of the illness.

COMMENT

There have been numerous reports in recent years of the corrosive effects of ferrous sulphate poisoning. Forbes (1947) and Thomson (1947) drew attention to the hazard of leaving attractive sugar-coated pills accessible to infants and children. Further cases have been described by Prain (1949), Spencer (1951), Crosskey (1952), Ross (1953), and Elliot-Smith and Davies (1954). Forshall and Rickham (1954) have discussed fully the complication of pyloric stenosis after ingestion of ferrous sulphate pills. Stricture of the mid-portion of the stomach, as found in the present instance, is less commonly encountered, and presents a more difficult problem of management than does a localized pyloric lesion.

If a child survives the immediate prostration and toxicity due to a heavy dosage of ferrous sulphate the mucosal changes in the stomach are apt to be severe and the subsequent contracture and ulceration progressive. Repeated haematemesis is probably an indication of excessive mucosal damage. It would appear that early operation is necessary, and it is suggested that jejunostomy should be performed as soon as possible. This should be followed by early resection of the stomach unless repeated radiological examination shows rapid improvement in the local condition. These contentions are borne out by the experience of Ross (1953), who described a somewhat similar case.

Dr. C. H. Stewart-Hess referred the child to me, and I am indebted also to Miss Isabella Forshall, to whom I transferred him for further treatment, for details of the later stages of the illness.

REFERENCES

- Crosskey, P. H. (1952). *British Medical Journal*, 2, 285.
Elliot-Smith, A., and Davies, P. A. (1954). *Ibid.*, 1, 156.
Forbes, G. (1947). *Ibid.*, 1, 367.
Forshall, I., and Rickham, P. P. (1954). *Brit. J. Surg.*, 41, 379.
Prain, J. H. (1949). *British Medical Journal*, 2, 1019.
Ross, F. G. M. (1953). *Ibid.*, 2, 1200.
Spencer, I. O. B. (1951). *Ibid.*, 2, 1112.
Thomson, J. (1947). *Ibid.*, 1, 640.

JOHN A. SHEPHERD, V.R.D., M.D.,
Ch.M., F.R.C.S.Ed.,
Surgeon, Broadgreen Hospital, Liverpool.

A Case of Priapism

Priapism is defined as continuous erection of the penis without sexual stimulation. It is an uncommon condition and is not very adequately dealt with in the textbooks. As the patient attended on a Sunday morning one was denied access to the literature.

CASE HISTORY

On August 23, 1953, a boy of 13 was admitted to hospital. While playing football about 40 hours previously he had been kicked accidentally in the perineum. A few hours later he was taken to his doctor's surgery complaining of lower abdominal pain and vomiting. He did not at that time mention the kick, and apparently it was thought then that he probably had mild appendicitis. He was not referred to hospital until the following day, by which time he had had intense priapism for about 16 hours, with dysuria.

On examination his penis was erect and extremely tender, as was the perineal region. No evidence of bruising was found, but direct questioning disclosed the history of trauma. His temperature was 103° F. (39.4° C.) and pulse rate 104. The bladder was distended to about half-way between the symphysis pubis and umbilicus at the beginning of the examination, but he managed to pass 13 oz. (370 ml.) of urine after rectal examination, which was very painful

owing to the congestion of the corpora cavernosa. The urine was clear. The spleen was not palpable and a blood examination did not disclose leukaemia. No positive findings were made on routine examination of other systems. A diagnosis of priapism due to mechanical obstruction of the efferent veins from the penis caused by oedema or thrombosis due to the trauma was made.

A low spinal anaesthetic was administered without any effect on the priapism. It did, however, relieve the pain temporarily. Any element of venous spasm thus being eliminated, the fact of mechanical obstruction was established.

Aspiration or incision of the corpora was decided against, as we considered that as the priapism had been present for only 16 hours thrombosis in the corpora should not have occurred and could be logically prevented by anticoagulants.

Treatment by heparin and ethyl biscoumacetate was begun, being controlled by daily prothrombin activity estimation. Adequate sedation was also administered in view of the severity of the pain.

The patient had considerable difficulty with micturition, but once the stream started the bladder emptied. Catheterization was avoided. On the second day he volunteered that micturition was easier, though there was no apparent alteration in the priapism. By the third day the temperature had settled spontaneously. On the sixth day he said that he now thought the penis was softer, and this was confirmed. By the twelfth day the penis was quite flaccid but still larger than it had been before the accident; micturition was normal. On the fourteenth day he was allowed up, but it was noticed that, although the corpora were quite soft, there was a tender spot in the right corpus in the perineum. It was considered that this was probably the site of the original trauma and would resolve spontaneously. Anticoagulants were discontinued. By the seventeenth day the prothrombin activity was within normal limits and the patient was discharged home. He was seen at the follow-up clinic on October 8, when it was found that the penis was normal in size and shape. As he has not reached the age of active sexual life no effect on this important function could be recorded.

COMMENT

Search of the literature has shown that only one case of priapism treated by anticoagulants (dicoumarol) has been recorded (Smith, 1950). Prior to this, aspiration and incision of the corpora were advised by several surgeons. Calloman (1950), in his exhaustive review of the condition, gives a good prognosis, whether or not active surgery is employed. Some authors (Kemp and Hosay, 1950) report impotence due to organization of clots in the corpora cavernosa. In one patient operative release of fibrous tissue at the base of the penis was required owing to persistent partial priapism and lack of erection during sexual stimulation. It would appear that this distressing sequel has been avoided in the case recorded, as no thickening can be felt in the perineum which might result in impotence.

The treatment of priapism with anticoagulants seems to have been entirely successful and would certainly appear to be the most rational despite Calloman's remarks.

My thanks are due to Mr. F. Mitchell Heggs, under whose care the patient was admitted, for permission to publish the case.

W. J. FRASER, M.B., F.R.C.S.Ed.,

Senior Registrar in Surgery, City General Hospital, Sheffield.
Late Senior Registrar in Surgery, Bury and Rossendale Hospital Group.

BIBLIOGRAPHY

- Aaron, G., and Robbins, M. A. (1949). *J. Urol. (Baltimore)*, 62, 328.
Bailey, H. (1948). *Brit. J. Surg.*, 35, 298.
— (1953). *Emergency Surgery*. Bristol.
Calloman, D. (1950). *Urol. cutan. Rev.*, 54, 144.
Campbell, J. H., and Cummins, S. D. (1951). *J. Urol. (Baltimore)*, 66, 697.
Freedman, S. Z. (1946). *Urol. cutan. Rev.*, 50, 728.
Henderson, D. St. C. L. (1950). *Brit. J. Urol.*, 22, 223.
Kemp, N. F., and Hosay, J. J. (1950). *Urol. cutan. Rev.*, 54, 69.
Kimsbrough, J. C., Worgan, D. K., and Denslow, J. C. (1950). *Ibid.*, 54, 53.
Smith, K. H. (1950). *J. Urol. (Baltimore)*, 64, 400.