rare, and in this instance there is no indication of how the organisms gained a "bridgehead" in the muscle. It is, however, possible that in some cases of clostridial myositis following a wound or an injection the infecting organism has been derived from the bowel rather than by inoculation from the exterior of the body. There was a rather suggestive latent period of at least eight days in the case reported by Shaw and Evans (1953), though admittedly delays of longer than five days may occur even in grossly contaminated wounds.

Direct evidence that phenylbutazone contributed to death is lacking. However, in view of the rarity of the condition, and the known effects of phenylbutazone on the continuity of the gut mucosa and on the circulating white corpuscles, it is difficult to dissociate the drug from some part in the initiation or spread of the gangrene or terminal septicaemia.

Since these two cases, both treated in general practice, two other fatalities have come to my notice. Both were instances of death due to perforation of peptic ulcer during phenylbutazone treatment. It should be noted that in Case 1 the perforation was unsuspected, nor was the cause of death clinically apparent in Case 2. The possibility of analgesic properties masking signs and symptoms is therefore of some importance.

It seems pertinent to iterate that, apart from the known contraindications, phenylbutazone should never be given without *regular and frequent* clinical assessment of the patient and appropriate white-cell counts.

Assessment of the precise risks attached to new drugs is never easy, and risks are not necessarily as great in hospital as they are in general practice. It is perhaps desirable that statutory post-mortem examinations should be held on all patients dying while treated by any new drug within a specified time of its inception *even if there is no prima facie reason for supposing death to be attributable to treatment.*

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REFERENCES

British Medical Journal, 1952, 2, 1401, 1427.
Davies, H. R., Barter, R. W., Gee, A., and Hirson, C. (1952). *British Medical Journal*, 2, 1392.
Shaw, S., and Evans, W. E. (1953). Ibid., 1, 429.

A Case of Subserous Duodenal Haematoma

A case of this condition due to non-penetrating and apparently trivial injury is reported because a search of the literature has not brought to light any record of a similar case.

CASE REPORT

A thin boy aged 9 was admitted to hospital on April 9, 1952, for abdominal pain and vomiting. Five days before admission he had "gone over" the handlebars of his bicycle; he thought he had fallen mainly on his left side, and had had a small abrasion of the left temple and left leg. From his own and his parents' account of the injury, it seems to have been regarded as trivial, and he was riding his cycle again a short time after the accident. He complained of some abdominal pain, but this was not severe and passed off in a few hours. Two days later he was seized with a sudden abdominal pain of such severity that he had to be put to bed; a hot-water bottle was applied to the abdomen, which apparently relieved him and he slept, but he woke at midnight and vomited food. Pain was felt in the central abdomen and remained in this area. The next day his general condition became worse; the abdominal pain was more severe, and vomiting and retching continued all day. His bowels were not open. His doctor decided that the condition was probably appendicular colic. He was kept in bed and given fluids and "sulphatriad."

On the day before admission he seemed to be slightly better: the pain was less severe, but vomiting and retching continued. There was a slight rise of temperature and pulse rate. His bowels had still not been open. On the day of admission the pain had again increased and vomiting and retching were very frequent. The doctor noted a slightly raised temperature, but the pulse rate had risen further.

Apart from the usual childhood diseases he had always been well.

On admission the patient was slightly flushed and seemed to be in some distress. His temperature was 98.6° F. (37° C.), pulse 74, respirations 22. The tongue was coated and dry, and there was some fetor oris. The abdomen was slightly distended but moved freely with respiration. There was no evidence of bruising or abrasion of the abdominal wall. The boy pointed to the site of pain as immediately below the umbilicus, and there was some guarding and tenderness in this region. Hard faeces could be felt in the descending colon. No other abnormality could be detected on abdominal examination. On rectal examination hard faeces were felt; there was no tenderness or induration. Routine urine examination showed no abnormality. The haemoglobin was 15 g.%; total leucocyte count, 16,200 (polymorphonuclears 84%, lymphocytes 16%); bleeding-time, 5½ minutes; clotting-time, 9 minutes. A straight *x*-ray film of the abdomen showed that the stomach contained a considerable quantity of gas.

The child was placed under observation and given fluids only by mouth. He vomited twice soon after admission; each vomit was of a few ounces and contained mucus and flecks of altered blood. An intravenous drip of glucose–saline was set up, a Ryle's tube passed, and gastric suction started; about 1 pint (570 ml.) of a "coffee ground" type of gastric fluid was aspirated. Further examination of the abdomen about eight hours later revealed a fixed ill-defined mass in the midline above the umbilicus.

On the morning of April 10 the boy's general condition had improved, and it was decided to perform a laparotomy to find the cause of what was obviously a high small-intestine obstruction. The abdomen was opened through an upper midline incision. The mass was found to be a gross enlargement of the second, third, and fourth parts of the duodenum and the first 4 in. (10 cm.) of the jejunum. At first sight the mass appeared to be gangrenous, being blue-black. Further examination showed that there was a very large subserous haematoma: 4 oz. (114 ml.) of fluid blood was aspirated from beneath the serous coat, and after incision of the serosa a great quantity of clot was evacuated with the finger. The underlying muscle coat appeared to be intact, but the presence of such a large clot had completely closed the lumen of the duodenum. The serosa was left unsutured and the abdomen closed without drainage.

Convalescence was slow but uneventful, and the patient was discharged from hospital six weeks after operation. When seen one month and three months after discharge he was very well. He had gained weight and was taking normal diet. No mass could be felt on abdominal examination.

COMMENT

Although there is no clear history of injury in this case, it is reasonable to assume that the lesion was caused by abdominal injury at the time of the accident. That the force of the blow was slight is evident from the fact that neither the child nor the parents were definitely aware of it, and from the absence of bruising of the anterior abdominal wall. The injury seems to be related to the blast injuries of the abdomen seen in the late war, and the effects of the injury were maximal in that area because of the immobility of the second, third, and fourth parts of the duodenum and the relative immobility of the duodeno-jejunal flexure.

I wish to thank Dr. C. D. Agassiz, physician-superintendent of Queen Mary's Hospital for Children, and Mr. A. M. Desmond, who performed the operation, for permission to publish this case.

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