of fracture may also be elicited by Vaughan's knuckle percussion test, which is described by Todd.

"The metacarpal joints are all flexed to a right angle, or if possible the patient is made to clench the fist; he is then told to keep his eyes shut, whilst the surgeon taps the knuckles smartly, one after another. If the fracture of the scaphoid is present sharp pain will be elicited when the head of the second metacarpal is struck, but not when any of the others is struck."

I have not found this sign of great value in diagnosis. In some cases it is not present, and in others it would appear to be present although no fracture is seen on x-ray examination.

The movements of the wrist, both in flexion and extension, are markedly limited, and the latter movement causes pain referred to the dorsal aspect of the navicular. Pain on flexion is rarely localized to a definite point.

The clinical features may be summarized as follows:

1. Swelling on the dorso-radial aspect of the wrist.
2. Obliteration of the tendons bounding the anatomical snuff-box.
3. Tenderness over the dorsum of the navicular.
4. Tenderness at the base of the snuff-box.
5. Limitation of flexion and extension of the wrist, the latter causing pain over the site of fracture.

While these symptoms are always prominent in recent cases they persist in untreated cases for long periods. I have recently seen a patient who had had an untreated fracture of the navicular for two years, and at the end of that period, when he came to hospital, he still showed all the characteristic signs which have been described.

I would stress the fact that diagnosis can be established without an x-ray examination, but, when available, plates should be taken to confirm the diagnosis. It is of the utmost importance that the skiagram be taken with the hand in marked ulnar deviation, for only in this position will all fractures of the navicular be detected.

FIG. 3.

FIG. 3.—Note the swelling in the anatomical snuff-box with masking of the extensor and abductor tendons.

FIG. 4.

FIG. 4.—Photograph taken five weeks after a fracture of the navicular, which had been treated in a plaster case. The patient was entirely free from pain, and full dorsiflexion was possible.

TREATMENT.

Immobilization is the keynote to success in the treatment of fracture of the navicular. The value of active movement in upper limb fractures is widely recognized, but the number of patients who suffer from a chronic disability following what has been erroneously diagnosed "sprained wrist," and treated by active movement, is sufficient proof that in this type of fracture, at least, it is not to be recommended. When the patient is seen

early the following treatment has given excellent results. A plaster case is applied to the forearm, wrist, and hand, the wrist being dorsiflexed and the fingers and thumb maintained in the position of normal grip. In most cases it is necessary to give an anaesthetic to enable sufficient dorsiflexion to be obtained. The parts are immobilized in this

plaster for a period of three weeks, but at the end of a fortnight the upper half of the plaster is removed to allow of massage. At the end of the third week the plaster is discarded and free movement encouraged. All the patients who have been treated in this way have obtained perfect results, both as regards movement and freedom from pain (Fig. 4).

When the patient is not seen until a month or two have elapsed immobilization has not proved of value. In some of these cases the proximal fragment has been removed by operation, but the results are not very satisfactory, and equally good results have been obtained by a prolonged course of massage, together with exercises for the wrist and hot and cold douching of the part. In my experience, however, the vast majority of these patients are left with a chronic disability, pain and stiffness in the wrist, and with a marked tendency for osteo-arthritic changes to occur (Fig. 1).

REFERENCE.

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TWO CASES OF ACUTE YELLOW ATROPHY OF THE LIVER FOLLOWING ADMINISTRATION OF ATOPHAN.

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IN 1922 Schroeder¹ first drew attention to the toxic properties of the drug variously known as phenylcinchoninic acid, cincofen, quinophan, atophan, agotan, and phenoquin. His seventeen cases—chiefly of mild poisoning—consisted of headache, gastro-intestinal disturbances, and transient jaundice; while Worcester-Drought² a year later reported a similar case. Following a communication from Langdon-Brown³ in 1926, a dozen fatalities were reported by Evans,⁴ Glover,⁵ Willcox,⁶ and Wells.⁷

In his presidential address to the Section of Therapeutics of the Royal Society of Medicine Graham⁸ emphasized the fact that every fatal case recorded was the result of uninterrupted use of this compound for a more or less lengthy period. Individual susceptibility plays a certain

part in determining the production of poisoning, as may also previous hepatic derangement, but it is noteworthy that there is no record, except in our second case, of any untoward result of this form of therapy where the drug is given in the correct manner. By this is meant its administration for periods of not longer than four days with intervals of not less than four days, as advised by Graham. This method brings about the maximum excretion of uric acid; the extension of the drug beyond these bounds, therefore, is not only therapeutically useless, but may be positively harmful.

The following are notes of the authors' two cases:

CASE I.

Read by L. J. A. L. before the Liverpool Medical Institution on February 23rd, 1928.

E. H., aged 55, had suffered from rheumatoid arthritis for many years. In July, 1927, on the advice of a neighbour, she began taking atophan at the rate of three 7½-grain tablets daily and without intermission. During the second week of January, 1928, she developed mild jaundice, apparently without any other symptoms. The final catastrophe occurred suddenly. The jaundice became more pronounced, on January 20th coma followed delirium, and she died on the following day.

The faeces had retained their normal colour throughout, and on this account a diagnosis of "acute yellow atrophy of the liver" was made. In view of the self-administration of atophan a coronor's inquest was held and a post-mortem examination performed.

Pathological Report.

(By the courtesy of Dr. W. Howel Evans of the University of Liverpool.)

Macroscopically.—The liver was the typical yellow-ochre colour, with small reddish cell areas. It weighed about 19 ounces, and resembled in consistency a fluid jelly contained in a thin capsule. The gall-bladder was normal, and contained a small quantity of bile. There were no gall-stones present.

Microscopically.—Sections showed a moderate degree of multilobular cirrhosis, which probably antedated the acute yellow atrophy. The great majority of the liver cells were completely necrotic; some areas showed fatty degeneration. There seemed to be practically no attempt at compensatory regeneration. The picture is that of almost complete necrosis, with the addition of the areas of fibrous tissue and the round-celled infiltration due to the multilobular cirrhosis.

CASE II.

Reported by W. A. M.

Mrs. R., aged 55. Previous history—nothing pertinent. On April 28th, 1927, the urine had been tested, and gave specific gravity 1010, reaction acid, no sugar or albumin. In May, 1923, she had sustained a fracture of the skull, with wound (compound fissured fracture). In the second week of December, 1927, she was ordered, for neuritic pains, atophan (7½ grains) three times a day for three days a week, and to take each morning a teaspoonful of sodium bicarbonate. According to her daughter's evidence at the inquest she never exceeded the dose, and ceased taking the drug about the end of January, 1928—five weeks before death. This was about a fortnight before the onset of jaundice.

On February 15th I found her jaundiced to a slight degree. She was put to bed, but became deeply jaundiced, and evidently was going downhill. There was some tenderness over the gall-bladder, and, owing to the uncertainty of the diagnosis, exploration was determined upon. A small cirrhotic-looking liver was found, but no dilatation of the ducts. She died within twenty-four hours on March 5th, 1928.

The urine had contained bile pigment. The faeces could not be got free from urine, and the examination of them gave equivocal results as regards the evidence of bile. *Post mortem*, a biphasic van den Bergh reaction was obtained on the blood. No obvious cause of the atrophy was found, and it was thought better to report the suspicion to the coroner, who held an inquest.

The extreme quantity she could have taken is 540 grains spread over eight weeks, and four days' interval followed each 67½ grains. At first she told her family that the drug was doing her good; later she had expressed a doubt.

Post-mortem Report (E. C. L.).

Macroscopically.—The liver was considerably smaller than normal, weighing about 26 ounces; it was soft in consistency, its capsule showing typical shrinkage. The cut surface was of the typical yellow-ochre colour and studded with small reddish areas. The gall-bladder and bile ducts were normal and patent. No gall-stones or cholecystitis were present. The pancreas was normal in size and consistency. The spleen was not enlarged, but rather softer in consistency than normal. The kidneys were normal in size, but somewhat soft in consistency.

Microscopically.—Liver sections showed marked necrosis of the hepatic tissue, interspersed with round-celled infiltration, without the development of any multilobular cirrhotic changes, the picture being typical of acute yellow atrophy. Sections of the spleen and pancreas showed no abnormal findings. Sections of the kidneys showed marked parenchymatous degeneration of the tubular epithelium, and suggest a considerable amount of subacute nephritis.

Conclusions.

1. Poisoning by phenylcinchoninic acid and its congeners is a condition which the medical practitioner should always bear in mind.
2. There may be no premonitory symptoms, although urticaria, albuminuria, gastro-intestinal disturbances, and transient jaundice have been noted. Acute yellow atrophy ushers in the fatal termination.
3. For the present we would suggest that the presence of albuminuria or any evidence of nephritis should be considered as a contraindication for the administration of this drug.
4. The slightest sign of intolerance, such as nausea or loss of appetite, should be an indication for the immediate stoppage of administration of the drug.
5. The estimation of a van den Bergh reaction and icteric index of the patient's serum will probably give the earliest evidence of any pathological effect upon the liver cells, and present the opportunity for omitting treatment,

long before any evidence of clinical jaundice could be observed.

6. Even if administered in the correct intermittent manner phenylcinchoninic acid or any of its congeners may be a dangerous drug. The authors feel that its indiscriminate use is very undesirable.

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- ¹ Schroeder, K.: *Ugeskrift for Laeger*, September 7th, 1922, p. 1141. References 2 to 7 are to the *British Medical Journal* of the dates given.
² Worster-Drought, C.: January 27th, 1923, p. 148. ³ Langdon-Brown, W.: July 3rd, 1926, p. 37. ⁴ Evans, G.: July 10th, 1926, p. 93. ⁵ Glover, L. G.: July 17th, 1926, p. 136. ⁶ Wilcox, Sir W. H.: August 7th, 1926, p. 273. ⁷ Wells, C. J. L.: October 23rd, 1926, p. 759. ⁸ Graham, G.: Presidential address, Section of Therapeutics and Pharmacology, Royal Society of Medicine, October, 1926. See also *British Medical Journal*, October 16th, 1926, p. 688.

Memoranda:

MEDICAL, SURGICAL, OBSTETRICAL.

GAS GANGRENE OF SIGMOID.

I VENTURE to think that the following case, resulting in death from toxæmia in twelve hours, must be fortunately of rare occurrence.

On February 27th, at 3.15 a.m., I was called to see a man, aged 51, with violent abdominal pain which had lasted about an hour. He had vomited twice, and had passed a little blood and mucus. The pain had begun in the right iliac fossa, and had become generalized. He was a healthy man, and looked less than his age. There was no history of any abdominal symptoms whatsoever, and of no illness except "rheumatism."

Condition on Examination.—He appeared to be very ill and was in obvious agony, though not collapsed. The temperature was 98° F., and the pulse 88. The tongue was furred, brown, and very dry. The abdomen moved on respiration; it was somewhat distended and extremely resistant, though there was no board-like rigidity. The tenderness was marked and universal. No assistance was given by a rectal examination.

Diagnosis.—This was difficult to fit in with any of the ordinary causes of an "acute abdomen" in a man of his age. It was decided to operate on the assumption that volvulus was present, and a quarter of a grain of morphine was given.

At 4.30 a.m. the pulse was 100 and the temperature was 100° F. There had been no more vomiting, but the pain was quite unrelieved.

Operation.—After a second injection of a quarter of a grain of morphine and 1/100 grain of atropine, the right rectus was displaced outwards below the umbilicus; free peritoneal fluid welled up on opening the cavity. The appendix was found to be normal, the gall-bladder was blue and apparently normal; the intestines were not unduly distended, and there was nothing to suggest an obstruction; there was no sign of duodenal or stomach contents, nor was any ulcer felt. There was no question of acute pancreatitis or of diverticulitis. The only abnormality I could find was in the lowest part of the sigmoid, where it passed into the rectum; this was rather swollen, oedematous, and stained by a few purple petechiae near the mesenteric border. I was frankly puzzled, since I hardly thought a lesion so low down, and so unfamiliar as a cause of an abdominal catastrophe, could account for the acute onset and extreme illness of the patient. His general condition was good and the pulse had hardly risen (120). Mr. Arthur Cooke also carefully examined the abdominal cavity and found everything normal except the sigmoid. He had never seen such a case, but hazarded an opinion of a *B. welchii* infection of the affected loop, and 10 c.cm. of gas gangrene serum was injected immediately. At the end of the operation the pulse rate was 100, and a rectal saline was given. At 9 a.m. the pulse was still only 100. The tongue was more moist, and his colour was better, but the agony was unrelieved. Morphine (1/4 grain) was given. At 12 noon the pulse had risen to 140; he was flushed and there was cyanosis. The breathing was rapid. The patient was evidently still in terrible pain; he had not vomited since the operation. The abdomen was somewhat distended. Another quarter of a grain of morphine was given. At 2.15 p.m. the patient had become cyanosed and grey; he was completely comatose and pulseless, and died a quarter of an hour later.

Post-mortem Findings.—Permission for a limited necropsy was obtained, and at 9 p.m., with Dr. H. E. Nourse, I explored the abdomen through the original incision extended upwards. The *post-mortem* changes generally were very marked for the short time since death, and the odour on reopening the distended suture line was appalling. There was much sero-sanguineous peritoneal exudate, and the changes in the sigmoid had progressed to complete gangrene for about six inches, extending below the reflection of the peritoneum. There was no diverticulum. Minute bubbles of gas were observed below the peritoneal coat. Elsewhere the abdomen was quite normal.

I sent some of the fluid and five inches of the gut to the Bonnett Laboratory; the following is a summary of Dr. C. H. Whittle's pathological report.

1. **Exudate.**—Large numbers of Gram-positive capsulated bacilli were found in films. Culture: (a) aerobic—streptococci and *Staphylococcus albus*; (b) anaerobic—a bacillus of the *B. welchii* group.

2. **Sections of Gut.**—There was intense congestion and gangrene of the whole wall and bleeding into the lumen. The wall was