steatorrhoea, but it may be worthy of note that the two lowest values obtained were in cases of pancreatic steatorrhoea.

In patients on steroid therapy the plasma activity ranged from 0.3 to 2.3% per litre, but all except two were below 1.4%. The two highest values were obtained in one patient who had been on steroids for only two weeks and in another on 7.5 mg. of prednisolone a day. The lowest value observed was in the case of Cushing's syndrome.

Ten subjects were given calcium-47 carbonate instead of calcium-47 chloride. The results of these tests (Fig. 4) indicate that the absorption of isotope in calcium carbonate carrier was inferior to its absorption in calcium chloride.

DISCUSSION

The procedure we have described is simple and quite suitable for out-patient use. Its validity depends upon the relationship between the two-hour plasma activity and the net calcium absorption calculated from the balance data. The observed correlation warrants the view that the isotopic procedure provides an index of calcium absorption. The 95% confidence limits are rather wide, but we have shown that it is partly accounted for by the long interval (up to one year) between some of the tests and the balances. Even when these latter tests are included the agreement is close enough to suggest that calcium absorption is a fairly constant factor in any given individual. The relatively wide normal range is therefore presumably not a function of the isotopic procedure as such but reflects the wide range of calcium absorption in normal subjects.

The results obtained in osteoporosis are entirely compatible with the results of our balance studies in this type of case (Nordin, 1960), which have shown that the great majority of patients with osteoporosis absorb calcium normally.

The results obtained in steatorrhoea may appear surprising since lower plasma activities might have been expected. However, this is a heterogeneous group of cases, many of them on treatment, and does not include any cases of untreated osteomalacia. Our balance studies (Nordin, 1961) have shown that the faecal calcium content in cases of steatorrhoea is slightly higher than that of normal subjects at the same intake levels, but the difference is a relatively small one.

The results obtained in patients on steroid therapy are also compatible with our own previous observations and with those of other workers (Bunim *et al.*, 1958; Slater *et al.*, 1959). On moderate doses of corticosteroids the only consistent abnormality of calcium balance so far observed is an impairment of calcium absorption. It would be compatible with the view that osteoporosis is caused by negative calcium balance to suggest that this malabsorption of calcium is at least a factor in the pathogenesis of steroid osteoporosis.

The isotopic procedure lends itself to the large-scale comparison of the absorption of different calcium compounds. So far we have only tested the absorption of calcium carbonate, which is of particular interest because of its use in the fortification of bread. The results suggest that calcium carbonate may be less well absorbed than calcium chloride, but clearly further work is required.

In conclusion, we suggest that this simple procedure may lend itself to the study of calcium absorption on a much larger scale than is possible with conventional balance techniques.

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SUMMARY

Calcium-47 in 250 mg. of calcium chloride was given to 34 fasting normal subjects. The maximum plasma activity was reached about two hours later and ranged from 0.3 to 3% of the dose per litre of plasma.

There was a good correlation between the two-hour plasma activity and the net calcium absorption as determined by calcium balance in 19 subjects ($r=0.78$; $P<0.01$).

Cases of osteoporosis and of steatorrhoea and patients on steroid therapy were also studied; absorption by most of those on steroid therapy was low.

Absorption of the isotope appeared to be reduced when it was given in calcium carbonate carrier.

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REFERENCES

- Barnett, E., and Nordin, B. E. C. (1960). *Clin. Radiol.*, **11**, 166.
Blau, M., Spencer, H., Swernov, J., and Laszlo, D. (1954).
Science, **120**, 1029.
Bronner, F., and Harris, R. S. (1956). *Ann. N.Y. Acad. Sci.*, **64**, 314.
Bunim, J. J., Black, R. L., Lutwak, L., Peterson, R. E., and Whedon, G. D. (1958). *Arth. and Rheum.*, **1**, 313.
Nordin, B. E. C. (1960). In *Bone as a Tissue*, edited by K. Rodahl and others. McGraw-Hill, New York.
— (1961). *Proc. roy. Soc. Med.*, **54**, 497.
Samachson, J. (1960). *Radiat. Res.*, **13**, 192.
Slater, J. D. H., Heffron, P. F., Vernet, A., and Nabarro, J. D. N. (1959). *Lancet*, **i**, 173.
Staub, H. (1954). *Schweiz. med. Wschr.*, **84**, 499.

Medical Memoranda

Visceral Scleroderma Without Skin Involvement

Only two reports of visceral scleroderma without skin involvement have been found in the literature. In the following case the patient presented with persistent vomiting, but there were no skin changes suggestive of scleroderma, and the diagnosis was not suspected in life or at necropsy. However, the histological examination of the heart, lungs, and alimentary tract showed changes characteristic of visceral scleroderma.

CASE REPORT

The patient, a man of 20, gave a history of persistent vomiting, almost every day for a year, with consequent loss of weight and, in the last month, generalized prostration. No other suggestive symptoms could be elicited by systematic questioning.

On examination he was grossly emaciated and too weak to sit up. His blood-pressure was 70/50 and his pulse irregular, but no abnormality was found on clinical examination in any other system.

An x-ray film of the chest taken at the bedside was unhelpful. An electrocardiogram showed multiple ventricular extrasystoles. Plasma electrolyte determinations gave the following results: sodium 135 mEq/l., potassium 3.5 mEq/l., chloride 71 mEq/l. Blood urea was 89 mg./100 ml. Total serum albumin was 3.4 g./100 ml., and globulin 1.6 g./

100 ml. Electrophoretic strip showed a normal pattern. On his admission the haemoglobin was 117% and the total leucocyte count 6,550/c.mm. (differential count normal).

Intravenous fluid was given to restore fluid balance. A Ryles tube was passed and a litre of fluid aspirated from the stomach, although immediately before this the patient had vomited 800 ml. of bile-stained fluid.

A radiograph taken after a small barium meal showed a dilated, atonic duodenum with practically no passage of barium in one hour. After two hours the barium had reached the mid-jejunal region. This investigation suggested ileus of the duodenum and first part of the jejunum, but there was no evidence of actual intestinal obstruction. As we were uncertain whether the ileus was neurogenic, mechanical, or biochemical, it was decided to prepare the patient for a diagnostic laparotomy.

Accordingly, a "polythene" tube was passed into the superior vena cava, and fluids containing high-calorie dextrose, amino-acid, and electrolyte were given. His general condition improved during the next two weeks and he was able to walk around the ward with a portable drip apparatus.

Eighteen days after admission he complained of the onset of sharp right-sided chest pain, worse on inspiration; nine hours later his condition deteriorated and he died unexpectedly.

Necropsy Findings.—The only macroscopic finding of note was dilatation of the first 18 in. (46 cm.) of small intestine. The remainder of the gastro-intestinal tract, the spleen, and other abdominal organs showed no macroscopic abnormality. The histological findings proved to be diagnostic. Sections from jejunum, heart, and lungs are reproduced. Fig. 1 A and B contrast abnormal and normal small bowel, both preparations being from the patient. In the affected section (Fig. 1 A) it can be seen that the muscle layers have been largely replaced by fibrous tissue. Fig. 2 shows lung and pleura with complete obliteration of normal lung architecture by fibrous tissue containing some cystic spaces lined by bronchial epithelium. Fig. 3 shows diffuse myocardial fibrosis. The vessels are normal. Sections from the other organs showed no significant abnormality.

FIG. 1.—Histological sections showing (A) an area of normal small bowel and (B) an abnormal area of small bowel. ($\times 75$.)

DISCUSSION

Generalized scleroderma, as emphasized by Prowse (1951), may cause fatal fulminating disease of the bowel, heart, or lungs. Skin involvement has almost universally been reported in published series. The only two reports of visceral scleroderma without skin changes found in the literature are those of Abrams *et al.* (1954) and of Miller *et al.* (1959), and neither paper emphasizes the rarity of this presentation.

There is general agreement that abnormalities of the gastro-intestinal tract occur in approximately 75% of cases of visceral scleroderma (Orabona and Albano, 1958). The characteristic x-ray changes are dilatation of the small bowel, especially of the second and third parts of the duodenum and the jejunum. There is also marked delay in the passage of barium (Pugh, 1948). Histologically, the small intestine shows thickening of the submucosa, and fibrosis and atrophy of the muscularis (Beerman, 1948; Bevans, 1945; Boyd *et al.*, 1954).

Electrocardiographic abnormalities reported include conduction defects, T-wave abnormalities, and ventricular extrasystoles (Weiss *et al.*, 1943). Histologically, the myocardium shows fibrotic change; arterial lesions of the myocardium have also been reported (Rottenberg *et al.*, 1959). Pulmonary fibrosis has often been described, and may lead to reduced vital capacity.

In the present case the combination of excessive vomiting, cardiac irregularities, and duodenal ileus

FIG. 2.—Histological section of lung and pleura. ($\times 75$.)

FIG. 3.—Histological section of myocardium. ($\times 20$.)

should perhaps have led to the diagnosis of visceral scleroderma. The patient's age, however, was unusual, most of the reported cases occurring in patients aged 40 to 60.

I thank Dr. Geoffrey Steed for permission to publish this report, and Dr. J. D. N. Nabarro for his help and encouragement in writing it. The barium-meal examination was carried out by Dr. C. G. Whiteside, radiology department, Middlesex Hospital, and the histological sections were prepared by Dr. John Arthur, Bland-Sutton Institute of Pathology, Middlesex Hospital.

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REFERENCES

- Abrams, H. L., Carnes, W. H., and Eaton, J. (1954). *Arch. intern. Med.*, **94**, 61.
 Beerman, H. (1948). *Amer. J. med. Sci.*, **216**, 458.
 Bevans, M. (1945). *Amer. J. Path.*, **21**, 25.
 Boyd, J. A., Patrick, S. I., and Reeves, R. J. (1954). *Arch. intern. Med.*, **94**, 248.
 Miller, R. D., Fowler, W. S., and Helmholtz, F. H., jun. (1959). *Proc. Mayo Clin.*, **34**, 66.
 Orabona, M. L., and Albano, O. (1958). *Acta med. scand.*, **160**, Suppl. 333.
 Prowse, C. B. (1951). *Lancet*, **1**, 989.
 Pugh, D. G. (1948). *Amer. J. med. Sci.*, **216**, 571.
 Rottenberg, E. N., Slocumb, C. H., and Edwards, J. E. (1959). *Proc. Mayo Clin.*, **34**, 77.
 Weiss, S., Stead, E. A., jun., Warren, J. V., and Bailey, O. T. (1943). *Arch. intern. Med.*, **71**, 749.

Acute Left Ventricular Failure after Successful Surgical Treatment of Bleeding Gastric Ulcer

This paper describes the case of a patient with oligæmic shock due to hæmorrhage who, immediately after arrest of the hæmorrhage, developed left heart failure with pulmonary oedema. The main purpose is to demonstrate that such a new development can take place with dramatic suddenness and that the treatment should be altered diametrically to correspond.

CASE REPORT

A married woman aged 76 was admitted to hospital on February 8, 1961, with a history of having vomited a quantity of bright red blood after experiencing a sudden sharp pain in the upper part of her abdomen. She vomited blood repeatedly and started passing fresh blood per rectum. Her past history revealed that she had suffered "a heart attack" around Christmas, 1960, but otherwise had always been fit and active.

On examination the patient was a thin, pale woman, mentally very active. The pulse was 96 a minute, regular, and of good volume; blood-pressure 160/50. There was evidence of mitral stenosis and aortic regurgitation: not in failure clinically. An E.C.G. showed left ventricular hypertrophy with strain. There was some tenderness in the epigastrium. Dark red blood was passed per rectum. On admission the hæmoglobin was 55%.

On account of the history and the evidence of free aortic regurgitation, conservative treatment was decided upon. During the next 18 hours 12 pints (6.8 litres) of blood were given, but repeated hæmatemeses and melaena were of such extent that she barely held her ground. At this stage it was decided to deal with the problem surgically.

She was taken to the theatre on her bed, with transfusions set up in both arms. Digoxin 0.25 mg. was given before she went to the theatre. On arrival in the anaesthetic room she vomited bright red blood and passed blood and clots per rectum. Rapid induction was performed on the table, and almost immediately she suffered a severe collapse, the carotid pulses being impalpable. The abdomen was quickly

opened through an upper midline incision. The stomach was found to be full of blood clot. A high partial gastrectomy was performed in just under 30 minutes, and at the end of operation her condition appeared to be satisfactory in the circumstances. During the operation a total of 1 pint (570 ml.) of blood was transfused. Examination of the specimen revealed an eroded artery in a subacute gastric ulcer. A disturbing factor noted at the conclusion of the operation was the degree of jugular venous distension. A few minutes later, just before her return to the ward, cyanosis developed. Frothy, pinkish fluid appeared at the corners of her mouth and the chest became full of moist sounds. B.P. at the time was 190/60. Oral suction was performed and then a bronchoscope was passed and suction of bronchi continued by this route for 20 minutes—fluid forming as quickly as it was removed.

In view of this acute left and right ventricular failure, aminophylline 0.25 g. was given intravenously and both transfusions were taken down. Esmarch bandages were applied on the suggestion of Dr. K. D. Keele to all four limbs to impede the venous return, thus performing a physiological venesection. At the end of 20 minutes the bronchoscope was removed and suction continued via the oropharynx. After 45 minutes from the onset of symptoms there was a noticeable improvement in the patient's condition. This continued steadily over the next 30 minutes, during which time the necessity for suction became less and the jugular venous engorgement disappeared. The total amount of bronchial aspirations was 2½ pints (1,420 ml.) of frothy blood-stained fluid. The patient was then returned to the ward and placed in an oxygen tent. The limb bandages were released and reapplied at intervals of 30 minutes over the next four hours, until they were finally taken off one at a time, the effect on jugular venous pressure being noted.

The patient made a satisfactory recovery. It is interesting to note that on the first post-operative day her hæmoglobin was 87% and hæmatocrit reading 47; transfusion, therefore, had not been excessive. Her subsequent post-operative progress has been uneventful.

COMMENT

This case raises a number of interesting points: (1) It illustrates a remarkable and dramatic situation where successful surgical treatment of severe hæmatemesis in a patient with a gross cardiac lesion precipitated an equally serious medical emergency. (2) It demonstrates the swift change from exsanguination to left ventricular failure requiring opposite modes of treatment. (3) It also shows the success of combining bronchial aspiration and medical venesection in the emergency treatment of acute left ventricular failure. (4) With increasing boldness of surgical approach in the treatment of hæmatemesis in the elderly, particularly those with cardiac lesions, this type of case may not be uncommon.

We thank Dr. K. D. Keele, under whose care this patient was originally admitted, and Mr. O. D. Morris, who performed the operation, for permission to publish this case.

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A scholarship in memory of the late Mr. S. I. Levy has been established by his widow at the Medical School of the Hebrew University of Jerusalem. Mr. Levy was for many years surgeon to the East Ham Hospital and to the Westminster Hospital, and for about 30 years senior surgeon to the London Jewish Hospital. The scholarship will be granted annually to a graduate of the Hebrew University specializing in surgery.