manufacturers issue a warning against the possible potentiation of the action and side-effects of phenothiazine compounds by amine oxidase inhibitors. However, the potentiation of toxic actions does not appear to be a very real danger. Combinations of drugs of these two groups have been used in psychiatric practice with apparent impunity (Bailey *et al*, 1959; Ayd, 1960; Holt *et al.*, 1960; Gosline *et al.*, 1960).

Though the experience of many workers indicates that the danger of liver damage by pheniprazine is unequivocally less than with iproniazid, the fact that jaundice and severe hepatic damage can occur in patients treated with this drug should be taken into account when prescribing the drug. The combination of phenothiazines and mono-amine oxidase inhibitors involves a risk the potential of which has not yet been fully assessed.

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REFERENCES

Ayd, F. J (1960). *Psychomatics*, **1**, 37.
Bailey, S. d'A., Bucci, L., Gosline, E., Kline, N. S., Park, I. H., Rochlin, D., and Saunders, J. C. (1959). *Ann. N.Y. Acad. Sci.*, **80**, 652.
Beer, D. T., and Schaffner, F. (1959). *J. Amer. med. Ass.*, **171**, 887.
Berkowitz, D., Rotman, M., Greenstein, R. H., and Sloane, N. G. (1961). *Amer. J. dig. Dis.*, **6**, 160.
Gogerty, J. H., and Horita, A. (1959). *Fed. Proc.*, **18**, 395.
Gosline, E., Bluestone, H., and Saunders, J. C. (1960). *J. clin. exp. Psychopath.*, **21**, 220.
Greenblatt, 1 J., and Kahn, A. (1959). *Ann. N.Y. Acad. Sci.*, **80**, 947.
Holt, J. P., Wright, E. R., and Hecker, A. O. (1960). *Amer. J. Psychiat.*, **117**, 533.
Popper, H. (1958). *J. Amer. med. Ass.*, **168**, 2235.

A Case of Acute Pancreatitis with Methaemalbuminaemia

On October 1, 1960, a sample of serum from a patient known to have acute pancreatitis was noticed to be the colour of strong milkless tea. Schumm's test was positive, showing that the colour was due to methaemalbumin—a curious finding in this patient.

CASE HISTORY

An obese woman of 28 developed intermittent abdominal pain and nausea on September 24, 1960, and by September 28 it had become constant and intense. She was then admitted to hospital, distressed and shocked, with a pulse rate of 120 and a mass of tenderness in the epigastrium; there were no bowel sounds. A diagnosis of acute pancreatitis was made, this being confirmed by a serum amylase of over 3,000 units.

Next day she became slightly jaundiced, and the serum bilirubin was 7.5 mg./100 ml.; on September 30 her temperature and pulse rose, and the blood sugar was found to be 560 mg./100 ml. On October 1 she complained of thirst, and Chvostek's and Trousseau's signs were positive, with a serum calcium of 6.3 mg./100 ml. and low electrolyte levels. At this stage the bronze-coloured serum was noticed, and in spite of supportive treatment her condition deteriorated rapidly and she died in hyperpyrexia on October 2.

At the post-mortem examination the most striking finding was the extent of the fat-necrosis, which was present in the greater omentum, posterior abdominal wall fat, and anterior abdominal wall peritoneum, and also in the parietal pleura, where the intercostal fat was necrotic. About 0.5 l. of haemorrhagic fluid was present in both pleural cavities, and a further 2 l. in the peritoneal cavity.

COMMENT

Methaemalbumin (Fairley, 1941) is formed when haemoglobin is liberated into the plasma in quantities greater than those needed to saturate the haptoglobins, and its presence implies intravascular haemolysis. It occurs in conditions such as blackwater fever, *Clostridium welchii* septicaemia, drug haemolytic anaemias, and pernicious anaemia.

If in this case the presence of methaemalbumin were ascribed to intravascular haemolysis the most likely haemolytic agent would be trypsin. This appears in the circulation only when the quantity released is enough to neutralize the antitryptic agents of the blood (Powers *et al.*, 1955) and therefore only in the severest cases. Rush and Cliffton (1952) showed that in induced pancreatitis in dogs, and also after injection of trypsin, treatment with a proteolytic inhibitor was followed by recovery from shock, and they suggested that proteolysis was due to trypsin itself and to the activation of serum plasminogen to plasmin, which is also a proteolytic enzyme. These may cause haemolysis by attacking the protein leaflets of the erythrocyte membrane, and this can be shown to happen *in vitro*; combination of the resulting haemoglobin breakdown products with albumin can then take place to form methaemalbumin.

An alternative source for the necessary haem is the 2.5 l. of haemorrhagic fluid in the abdominal cavity, for not only can solutes be absorbed from the peritoneal cavity into the circulation but even actual red cells, as is the case in intraperitoneal transfusion. These presumably pass through the stomata described by Florey (1927); transfused erythrocytes can be seen in the dog, and traced by means of radioactive iron as they pass through the anterior mediastinal lymphatics (Halm *et al.*, 1944, quoted by Waite *et al.*, 1956). Chromium studies have also been used to demonstrate the transfused cells in the circulation.

It is surprising that no report similar to this can be found, as haemorrhagic fluid is often present in acute pancreatitis and is a common post-mortem finding. Probably recognizable quantities of methaemalbumin may be formed only in the presence of a large amount of haemorrhagic exudate, and its presence is probably a vaguely quantitative estimate of the volume of the exudate. It may therefore indicate a bad prognosis.

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REFERENCES

Fairley, N. H. (1941). *Quart. J. Med.*, **10**, 95.
Florey, H. (1927). *Brit. J. exp. Path.*, **8**, 479.
Powers, S. R., jun., Brown, H. H., and Stein, A. (1955). *Ann. Surg.*, **124**, 690.
Rush, B., jun., and Cliffton, E. E. (1952). *Surgery*, **31**, 349.
Waite, M. E., Colucci, D. D., and Glaser, J. (1956). *Amer. J. Dis. Child.*, **91**, 561.