THE PRESENT POSITION OF GASTRIC HYPOTHERMIA

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Introduction

In 1958 gastric cooling for massive gastric haemorrhage and subsequently in 1961 gastric freezing as a definitive treatment for peptic ulcer was introduced by Wangensteen. The initial enthusiastic acceptance of the new technique gave way to a more cautious attitude as the problems and complications associated with the procedure became known.

The author has had the opportunity of observing and taking part in the techniques employed by Wangensteen and his co-workers and in the experimental work currently being done at the University of Minnesota. The purpose of this paper is to make an assessment of the present position of gastric hypothermia in the light of this experience.

Gastric Freezing

Gastric freezing has been criticised from two aspects. On the one hand various complications of the procedure have been reported. It seems also that the effects are short-lived and the technique does not produce a permanent cure.

Complications

(a) Errors in technique

White et al. (1964) mention rupture of the oesophagus and stomach as a hazard but agree that such occurrences are due to faulty technique and should not happen if adequate care is taken.

They also mention rupture of the balloon with leakage of alcohol into the stomach and acute alcoholic poisoning as a possible hazard. This risk has now been eliminated by using a silicone fluid as the coolant.

(b) E.C.G. Changes.

Karacadag and Klotz (1964) reported E.C.G. changes associated with gastric freezing. An abnormal electrocardiograph is a constant finding during the freezing procedure. In many cases the abnormal tracing may persist up to 24 hours following the gastric freeze. All investigators report these abnormal E.C.G. records as transient and none have persisted beyond 24 hours.

(c) Melaena

This is a not infrequent complication. Wangensteen (1963) reported an incidence of 5 per cent in a series of 841 cases. There was no associated mortality.

McFarland (1968) found melaena in 19 of 184 patients. In 6 of these the haemorrhage was sufficiently severe to warrant blood transfusion.
(d) Post-freeze Gastric Ulcer

The occurrence of gastric ulcer after freezing is probably the most serious objection to the technique. At the University of Minnesota post-freeze gastric ulcer was acknowledged as a problem. In a series of 949 episodes of gastric freezing there were 27 gastric ulcers with two perforations. However, there was no mortality.

The causes of post-freeze ulcer include excessively low mucosal temperatures, over-distension of the balloon and a "cold spot" created by a jet of coolant from a terminal vent in the inflow tube. The creation of lateral vents in the inflow tube after sealing its lower end has reduced the incidence of gastric ulcer.

An additional precaution against the occurrence of post-freeze ulcer is the use of low molecular weight destran to prevent sludging in the gastric vessels (Goodale, 1964).

Post-freeze ulcer has been reported by Lisker (1964) who reported a death from a post-freeze perforated ulcer which failed to heal following repair of the perforation. The failure to heal was apparently due to necrosis of the stomach wall.

(e) Necrosis of the Stomach Wall

This serious complication may be due to pressure necrosis from over-distension of the balloon. Excessively low temperatures may also be a factor especially in association with a high degree of acidity. The body and fundus of the stomach are most susceptible because of their great distensibility (Marx and Kalig, 1964).

The control of balloon temperature can be difficult. Most workers appear to rely on inflow and outflow temperatures. At the University of Minnesota balloon-mucosal temperatures are continuously monitored from a number of thermocouples on the balloon surface. This gives a high degree of temperature control as downward drifts can be corrected immediately.

Results of Gastric Freezing

Symptomatic Relief

All authors agree that there is immediate relief of symptoms following a gastric freeze. Wangensteen found 90 per cent of patients asymptomatic and 90 per cent with x-ray evidence of healing of the ulcer in the early post-freeze period.

McFarland (1968) found immediate relief of symptoms in 74.7 per cent of patients.

White et al. (1964) reviewed 150 cases and found relief of symptoms in them all. However, there was pre-freeze evidence of duodenal ulceration in only 66 per cent of these cases.

The measurement of results by using subjective criteria such as symptomatology is always open to question and the only way round the difficulty is the use of the double-blind trial. Such trials have been done (McIlrath and Hallenbeck, 1964) and their results indicated a genuine reaction following gastric freezing with reduction of acid secretion and relief of symptoms.