It is generally recognized that splenectomy, at least temporarily, increases susceptibility to bacterial infections (Lowdon *et al.*, 1966) and in the present small series several infections occurred after splenectomy. The findings in a large number of splenectomies in Hong Kong were similar (Cook *et al.*, 1963). The two patients who died suddenly in their villages some months after operation may have succumbed to overwhelming bacterial infections or to malaria. No clinical observations could be made during their fatal illnesses. Splenectomy alters the effective state of immunity to malaria, and in latent infections in experimental animals parasitaemia induced by splenectomy may persist for life (Garnham, 1963). Fatal *Plasmodium falciparum* malaria has been demonstrated after splenectomy in a previously immune chimpanzee (Rodhain and Jadin, 1964). *P. vivax* parasitaemia occurred in the postoperative period in one patient and some months after operation in two. Chloroquine tablets were given to each patient, with instructions that they be taken if fever occurred. Therefore little information concerning the hazards of malaria after splenectomy is provided. However, for cases from areas with endemic malaria and sporadic medical surveillance, splenectomy should probably be restricted to patients with disabling symptoms. Most persons with tropical splenomegaly are not greatly disabled despite their very large spleens and considerable anaemia.

The microscopical appearances of the liver biopsy specimens confirmed the absence of hepatic cirrhosis (Pitney *et al.*, 1967), so that the results of operation might be expected to be better than those of Chaudhuri *et al.* (1956) and Cook *et al.* (1963), where many patients had cirrhosis. This latter study also showed much less mortality after splenectomy in those patients without cirrhosis. Hepatomegaly is usually found in tropical splenomegaly and, as jaundice, wasting, and even oedema may occur, it is difficult to exclude cirrhosis other than by liver biopsy.

Summary

The results of splenectomy are described in 15 patients with tropical splenomegaly in New Guinea. Splenectomy was performed only in those patients with disabling symptoms due to anaemia or the size and weight of the spleen. There was one operative death due to haemorrhage, and two other patients died suddenly some months after splenectomy.

The main cause of anaemia in this disorder was an expanded plasma volume, which was reduced after splenectomy. Increased red cell destruction was also corrected by splenectomy. There was a sustained rise in haemoglobin values, and the surviving patients have remained well over a short follow-up period. It is suggested that, until more information is available concerning the natural history of the disorder and the long-term results of splenectomy in regions where malaria is endemic and suppressive therapy difficult to administer, splenectomy should be reserved for patients with disabling symptoms.

I wish to thank Professor W. R. Pitney, of the University of New South Wales, for helpful advice and criticism; Dr. A. R. Shepherd, who performed the splenectomies; the Wellcome Trust for the provision of equipment; and Dr. R. F. R. Scragg, Director of Public Health, Territory of Papua and New Guinea, for making available facilities for this work.

References

Atkinson, M., and Sherlock, S. (1954). *Lancet*, **1**, 1325.
Bowdler, A. J. (1963). *Ann. intern. Med.*, **58**, 664.
Chaudhuri, R. N., Saha, T. K., Basu, S. P., Mukherjee, A. M., and Chaudhuri, M. N. R. (1956). *Indian J. med. Res.*, **44**, 305.
Cook, J., McFadzean, A. J. S., and Todd, D. (1963). *Brit. med. J.*, **2**, 337.
Crawford, H., and de Gruchy, G. C. (1958). *Med. J. Aust.*, **1**, 657.
Dacie, J. V., and Lewis, S. M. (1963). *Practical Haematology*, 3rd ed. London.
Garnham, P. C. C. (1963). In *Immunity to Protozoa*, edited by P. C. C. Garnham, A. E. Pierce, and I. Roitt. London.
Hamilton, P. J. S., *et al.* (1965). *E. Afr. med. J.*, **42**, 196.
Lowdon, A. G. R., Stewart, R. H. M., and Walker, W. (1966). *Brit. med. J.*, **1**, 446.
McFadzean, A. J. S., Todd, D., and Tsang, K. C. (1958). *Blood*, **13**, 524.
Pitney, W. R., Pryor, D. S., and Tait-Smith, A. (1967). *J. Path. Bact.* In press.
Prankerd, T. A. J. (1963). *Brit. med. J.*, **2**, 517.
Pryor, D. S. (1967a). *Quart. J. Med.*, **36**, 321.
—— (1967b). Ibid., **36**, 337.
Rodhain, J., and Jadin, J. (1964). *Ann. Soc. belge Méd. trop.*, **44**, 531.
Veall, N., and Vetter, H. (1958). *Radioisotope Techniques in Clinical Research and Diagnosis*. London.
Weinstein, V. F. (1964). *Lancet*, **2**, 218.

Joint Haemorrhage in Haemophilia: Is Full Advantage Taken of Plasma Therapy?

ANITA M. ALI,* M.B., B.S.; R. H. GANDY,*† M.B., B.S.; MARGARET I. BRITTEN,* B.SC.
KATHARINE M. DORMANDY,* M.B., M.R.C.P., M.C.PATH.

Brit. med. J., 1967, 3, 828–831

Recurrent bleeds into joints and muscles are among the most typical and frequent of the haemorrhages sustained by the severely affected haemophiliac (Wilkinson *et al.*, 1961; Ramgren, 1962; Biggs and Matthews, 1966; Stuart *et al.*, 1966; and many others). Without immediate and competent treatment these bleeds are potentially crippling, leading to various degrees of functional disability (Jordan, 1965a); their frequency makes adequate schooling practically impossible (Britten *et al.*, 1966), thereby jeopardizing the boy's future career.

Despite the fact that plasma replacement therapy, given as soon as possible after the onset of bleeding, has for some years been the accepted treatment for musculoskeletal haemorrhage (Biggs, 1964; van Creveld, 1964; Hardisty and Ingram, 1965; Salzman and Britten, 1965), it was apparent that many haemophiliacs in the Metropolitan Regional Hospital Board Areas were receiving inadequate treatment or none at all. A survey was therefore undertaken to ascertain the extent to which this treatment was in fact being used in that area. An attempt was also made to correlate the outcome of bleeds with the therapy that had been received. This paper describes our findings during 1965 and 1966.

* Department of Haematology, the Royal Free Hospital (North-western Branch), London N.W.3.
† Present address: Department of Pathology, the Combined Cambridge Hospitals, Tennis Court Road, Cambridge.

Materials and Methods

The patients and their parents who took part in this investigation were already in touch with us through our survey into the educational problems of haemophiliacs. A total of 210 patients under the age of 30 ("boys"), registered at many different hospitals and all suffering haemarthroses, agreed to co-operate.

The boys were usually visited at home, with one or both parents present. A detailed medical history, with particular reference to the musculoskeletal system, was taken and the patient's joints were examined. The range of each joint was assessed on the active movements which the boy could accomplish without difficulty or pain. The degree of muscle-wasting was noted.

Haemophilia and Christmas disease were considered together. The patients were divided into groups according to age and the treatment received.

The age groups chosen were 0 to 5 years, 5 to 11, 12 to school-leaving, and school-leaving to 30. (School-leaving age was used in preference to a definite age, because the hazards of the schoolboy differ from those of the young adult.)

The treatment groups chosen were "no plasma" and "plasma." Boys were assigned to the no plasma group if they had never or only rarely received replacement therapy, and to the plasma group if they regularly or frequently received it. Their treatment was regarded as prompt if they received a rapid infusion of fresh-frozen plasma or its equivalent within 12 hours of the onset of a joint or muscle haemorrhage.

A system of grading, based on that of DePalma and Cotler (1956), was used for all joints into which there had been a haemorrhage. The assessment was clinical, as no radiological examinations were made. Thus:

Grade 1—joints into which there had been one or more bleeds but which showed no residual decrease in the range of movement and therefore no deformities which could be clinically observed.

Grade 2—joints showing slightly reduced range of movement but no appreciable joint dysfunction.

Grade 3—joints in which the reduced range of mobility interfered with function.

Grade 4—joints showing more advanced changes than in grade 3—that is, severe deformities.

Results

Of the 210 boys 196 sustained numerous episodes of joint haemorrhage; 131 were found to have deformities. Haemorrhage had occurred into 999 joints, of which 415 were deformed. Of the affected joints, 38% had sustained first bleeds before the boys had reached the age of 4, and 60% had done so by the age of 6.

A quarter of the boys under the age of 5, nearly half of the 5–11 group, and the majority of the older boys had deformities. Table I shows not only how early haemophilic deformities begin but how rapidly their severity increases during the years of growth. Several of the boys in the school-leaving to 30-year group had spent a period of their childhood confined to wheelchairs because of severe knee deformities. All of these had had orthopaedic treatment and were able to walk again.

Varying degrees of muscle-wasting, especially in the thigh and calf, were seen in most of the boys. Where there was gross quadriceps-wasting the boys appeared to have knee-bleeds with ever-increasing frequency unless the knee had been protected by a calliper or other appliance. Muscle haemorrhages accounted for five equinus deformities resulting from calf bleeds, one claw-hand after a forearm bleed, and one fixed flexion deformity of the left elbow after a haemorrhage into the biceps.

Though temporary immobilization, preferably by means of splints, is an important aid to recovery from haemarthroses and muscle bleeds, as is graded physiotherapy (France and Wolf, 1965), many of the boys had never had a joint splinted nor had they been taught suitable exercises to maintain the muscles controlling the affected joint.

TABLE I.—*Deformed Joints. Percentage of Lesions of Grades 2, 3, and 4 in Each Age Group.*

Deformities	Age Groups			
	0–4	5–11	12–SL*	SL–30
Percentage grade 2 ..	80	58	48	52
" " 3 ..	20	27	32	28
" " 4 ..	0	15	20	20

* SL=School-leaving age.
N.B.—Grade 1 lesions are omitted because they are not deformities.

Plasma Replacement Therapy

Extent of Use

Table II shows the division of the boys into age and treatment groups. Seventy-one boys did not attend hospital for haemarthroses; 29 others went to hospital but were not infused, and 16 had had plasma only once or twice in their lives. Three boys had inhibitors to factor VIII; the histories of the remaining 16 classified as "miscellaneous" were vague.

TABLE II.—*Treatment Received by the Boys Up To the Time of the Survey*

Treatment	Age Groups				Total
	0–4	5–11	12–SL*	SL–30	
Plasma { Prompt ..	7	6	12	4	29 } 75
Plasma { Delayed	7	7	25	7	46 }
No plasma ..	12	48	30	26	116 } 135
Miscellaneous† ..	0	3	15	1	19 }
Total ..	26	64	82	38	210

*SL = School-leaving age.
† "Miscellaneous" includes boys known to have had some plasma therapy but for whom no detailed history could be obtained and 3 with inhibitors to factor VIII.

Of the 210 boys, only 75 had received plasma with any regularity; the treatment of 46 of these had been delayed, leaving only 29 who appeared to be receiving prompt and regular therapy.

Reasons for Delayed Treatment.—(a) Delay on the part of the patient or his parents in seeking hospital treatment: 55 boys did not go to hospital within 12 hours of the onset of a haemarthrosis; half of these waited several days or even longer. (*b*) *Delay in obtaining admission to hospital*: about 26% of the boys contacted their general practitioners before going to hospital. Sometimes this was because their hospitals insisted on a doctor's letter each time; others relied on him to arrange transport. Further delays occurred on arrival at many hospitals, where there were no short cuts through the time-consuming process of admission and a haemarthrosis was not regarded as an emergency. (*c*) *Delay in hospital before an infusion was given*: 16 boys were not treated on admission; of these, 14 waited several days.

Short-term Effects of Plasma Therapy

In addition to the rapid relief of pain and the faster reduction of swelling, plasma treatment considerably shortened the time "out of action." Thirty-nine boys gave estimates of the periods of time they were incapacitated by haemarthroses treated with plasma and those not so treated. A further 44, who did not receive plasma for joint bleeds, estimated the time missed with each episode. Of those treated, 72% were active in less than a week, and none were laid up for more than two weeks, whereas 52% of the untreated boys were out of action for two to four weeks and 7% for over four weeks. Only boys with very minor haemarthroses recovered untreated in less than a week.

Long-term Effects of Plasma Therapy

Fourteen boys, all under the age of 8, had received plasma treatment throughout their lives. Among their 40 affected joints there were only three deformed ones, two of which were in the same child. Both these boys had had treatment only after delays of two days. Two of the deformities were grade 2 and the third grade 3.

A detailed comparison was made between the numbers of new deformities which had developed in treated and untreated

boys between the ages of 5 and school-leaving; this was the period during which the maximum number of deformities had been found to start. Our findings were:

Untreated boys: 1 deformity to every 7 joints with haemorrhage.
Treated boys: 1 deformity to every 39 joints with haemorrhage.

We obtained the above figures in the following way: All boys in the no-plasma group who sustained recurrent haemarthroses in more than one joint were considered, as were all similarly affected boys in the plasma group, provided that they had been receiving treatment for at least three years.

The average age of the 26 treated boys was 11.1 years (range 5–17), and they had been receiving treatment for an average of 4.5 years (range 3–7). Only four deformities had developed in the 156 affected joints. Three of these had occurred in boys who had received delayed infusions. The fourth, a grade 2 knee lesion, appeared to have been promptly treated for each haemarthrosis, but this joint had sustained many haemorrhages in a short period. The average age of the 50 untreated boys was 11.3 years (range 5–17). Records were made of all deformities that had developed in these boys in the 4.5 years before their examination for the survey. Thirty-six deformities had started in the 251 affected joints.

Haemarthroses occurred to approximately the same extent in the two groups both before and during the period studied; before that time the incidence of deformity had also been similar.

Thirty-three boys said that on one or more occasions aspiration had been used in the treatment of massive haemorrhage into a knee or ankle. In some cases joints had unfortunately been aspirated without plasma cover. Where plasma had been given in conjunction with aspiration some boys had felt great relief, but for others this procedure had been unsuccessful and painful.

Nearly half of the boys were not registered at a haemophilia centre, though there were 10 in the area investigated. Many of those who were registered had attended only for diagnosis and were treated at other hospitals or at home. One severely crippled boy of 20 had never been to any hospital for a haemarthrosis.

Discussion

Many authors have reported a big reduction in the incidence of deformity in patients receiving plasma replacement therapy (Biggs, 1964; Poulain, 1964; Ahlberg, 1965; Jordan, 1965b; Brinkhous *et al.*, 1966). As the results of our survey were similar, it was disturbing to find how few were the boys receiving prompt, or indeed any, plasma treatment for joint and muscle haemorrhages.

Since many of the affected joints examined had sustained first bleeds before the boys had even reached the age of 4, it is clear that the best therapy must be available from earliest childhood if permanent deformity is to be prevented. This applies equally in cases of muscle haemorrhages, as the deformities which follow these are usually severely disabling—none of those seen in the survey had been promptly treated.

Effective treatment involves the early administration of active therapeutic material. Day or night the patient or his parents must themselves be able to telephone the hospital to say he is coming for an infusion. As plasma infusions are given rapidly (Matthews, 1966) treatment can often be on an outpatient basis, thus causing minimal interruption to the boy's life. Evans (1967) has commented that most of the haemophilic boys at the Lord Mayor Treloar College, Froyle, Alton, Hampshire, where there are more than 30 over the age of 11, are absent from school with a haemarthrosis for hours instead of weeks now that they are receiving prompt plasma therapy.

At the time of the survey much of the frozen plasma issued to hospitals in the south-east of England was deficient in factor VIII, and therefore useless for the control of haemophilic haemorrhage (Bennett *et al.*, 1967). Because their own tests had shown the poor quality of this plasma, several haemophilia centres were making their own therapeutic materials (personal communications from R. M. Hardisty, 1966; C. A. Holman, 1966; and G. I. C. Ingram, 1966). All the 29 boys thought to be having prompt and effective treatment attended one of those centres. It seems likely that the use of inactive plasma, as well as delayed administration, was responsible for the lack of success of the therapy received by some of the boys.

Because haemophilia is a rare disorder, and its management is highly specialized, it is desirable that patients should be treated only at hospitals where every facility is always available. Many patients have found it worth while to go to a centre for treatment even when a considerable journey was involved. Where it would be impracticable for a boy to attend a haemophilia centre for routine treatment, his local hospital should co-operate closely with the centre where he is registered, so that advice can be easily obtained at any time (Dalrymple-Champneys *et al.*, 1967).

The majority of doctors have little chance to gain experience in the treatment of haemophilia. Many did not seem to appreciate the seriousness of joint and muscle bleeds, and were unaware of the great advances made for the treatment of these. Discouraged by the results of the conservative care given by many hospitals, general practitioners often merely advised bed-rest. It was evident during the survey that much needless suffering and crippling was occurring because doctors, parents, and patients did not know where to seek help.

This country has been well to the fore, both technically and clinically, in research into the problems of haemophilia, as was indicated in a leading article in the *British Medical Journal* (1966). It would indeed be sad if Great Britain were to lag behind in the treatment of the most frequent complications of the disorder. For practical reasons we had to limit the area studied, and have therefore made no mention of the position in other parts of the country, though we are well aware that a number of coagulation units are providing excellent care for their haemophilic patients. Now that cryoprecipitate factor VIII concentrate is becoming more readily available it should be easier to organize the provision of good therapy, and on a wider scale, than it was before 1966. It is essential that knowledge of the advantages of this treatment, and of where it can be obtained, should be more widespread among both doctors and patients.

Summary

The state of the joints of 210 haemophilic boys was clinically investigated in south-east England during 1965–6. Particular attention was paid to the treatment that had been received.

Prompt infusions of active plasma preparations had reduced (*a*) the duration of incapacity from haemarthroses by over half, and (*b*) the incidence of new deformities by four-fifths.

Without plasma therapy joint damage begins at an early age—one-quarter of the boys under 5 years had deformed joints. Overall, 131 boys had deformities involving 415 joints.

Only one-third of the 210 boys had received any plasma therapy; only one-seventh had had prompt treatment.

Some of the reasons why so few boys had had effective treatment were: (*a*) many doctors and parents were unaware of the benefits of plasma therapy for haemarthroses or of the need for prompt infusions; (*b*) few hospitals had arranged for patients' transport and speedy admission—few had issued instructions or given advice; and (*c*) many haemophiliacs attended hospitals which had no specialized coagulation units.

We wish to thank the many consultants, general practitioners, school medical officers, and school principals who have made this survey possible. We are especially grateful to Dr. Rosemary Biggs for her help in planning the survey and in interpreting the results. The co-operation of patients and their parents has been unlimited.

Financial support was given by the Medical Research Council and the Haemophilia Society.

Our preliminary findings were reported by K. M. D. to the Institute of Haemophilia during the 10th World Congress of the International Society for the Rehabilitation of the Disabled, Wiesbaden, September 1966.

REFERENCES

Ahlberg, A. (1965). *Acta orthop. scand.*, Suppl. No. 77, p. 74.
Bennett, E., Dormandy, K. M., Churchill, W. G. L., Coward, A. R., Smith, M., and Cleghorn, T. E. (1967). *Brit. med. J.*, **2**, 88.
Biggs, R. (1964). *Proc. roy. Soc. Med.*, **57**, 1171.
——and Matthews, J. M. (1966). *Treatment of Haemophilia and Other Coagulation Disorders*, edited by R. Biggs and R. G. Macfarlane, p. 129. Oxford.
Brit. med. J., 1966, **2**, 1608.
Brinkhous, K. M., Swanton, M., Webster, W. P., and Roberts, H. R. (1966). *Proceedings of Congress of the World Federation of Hemophilia*, Australia. In press.
Britten, M. I., Spooner, R. J. D., Dormandy, K. M., and Biggs, R. (1966). *Brit. med. J.*, **2**, 224.
Dalrymple-Champneys, W., Hunter, J. R., and Polton, K. R. (1967). Ibid., **2**, 440.
DePalma, A. F., and Cotler, J. (1956). *Clin. Orthop.*, **8**, 163.
Evans, E. S. (1967). *Proceedings of British Orthopaedic Association Clinical Meeting*, Spring, 1967, p. 54.
France, W. G., and Wolf, P. (1965). *J. Bone Jt Surg.*, **47B**, 247.
Hardisty, R. M., and Ingram, G. I. C. (1965). *Bleeding Disorders: Investigation and Management*, p. 78. Oxford.
Jordan, H. H. (1965a). *Acta orthop. belg.*, **31**, 640.
—— (1965b). *Proceedings of Institutes on Hemophilia* (National Hemophilia Foundation, N.Y.), p. 22.
Matthews, J. M. (1966). *Treatment of Haemophilia and other Coagulation Disorders*, edited by R. Biggs and R. G. Macfarlane, p. 70. Oxford.
Poulain, P. (1964). *Réadaptation*, No. 108, p. 27.
Ramgren, O. (1962). *Acta med. scand.*, **171**, 237.
Salzman, E. W., and Britten, A. (1965). *Hemorrhage and Thrombosis*, p. 38. Boston and London.
Stuart, J., Davies, S. H., Cumming, R. A., Girdwood, R. H., and Darg, A. (1966). *Brit. med. J.*, **2**, 1624.
van Creveld, S. (1964). *Nederlandse Hemofiliestichting*, p. 2. Utrecht.
Wilkinson, J. F., Nour-Eldin, F., Israëls, M. C. G., and Barrett, K. E. (1961). *Lancet*, **2**, 947.

Trial of Gastric Cooling for Haematemesis*

J. B. McFARLAND,† M.D., CH.M., F.R.C.S.; J. G. GOW,‡ M.D., CH.M., F.R.C.S.

Brit. med. J., 1967, **3**, 831–834

Gastric cooling as a method of controlling upper gastrointestinal haemorrhage was initially developed by Wangensteen *et al.* (1958). The temperature and the rate of flow of coolant which they employed produced an intragastric temperature of the order of 15° C. They suggested that this caused: (1) reduced gastric blood flow; (2) depressed acid/pepsin secretion and activity; and (3) decreased gastric motility.

In an extended clinical series they reported on the treatment of 75 patients by gastric cooling, with control of haemorrhage in 57 (76%) (Nicoloff *et al.*, 1962). This result is comparable to the figures analysed by Crampton *et al.* (1964), who added a further 123 reported cases and noted an overall control of haemorrhage in 72.6%. Unfortunately, cessation of haemorrhage cannot necessarily be equated with patients' survival, and in one of the large series included in this analysis (Wangensteen *et al.*, 1963) haemorrhage was controlled in 87% of cases but the hospital mortality was 57%.

Usually a commercial refrigerator formed the basis of the apparatus for gastric cooling. A refrigerated alcohol/water solution was pumped through an intragastric balloon. Mechanical complications with such apparatus have been described by Wangensteen (1962), Nicoloff *et al.* (1962), Richman and Anthony (1963), and Miller *et al.* (1963). Because of these complications a simplified water-circulating apparatus was used in this study.

Haematemesis is an alarming symptom of a serious situation. There is a definite place for a non-operative technique that would assist in its early management and allow time for an exact diagnosis to be made later. We here compare a trial of early gastric cooling with conventional methods of managing massive gastrointestinal haemorrhage.

Subjects, Materials, and Methods

The patients treated had been admitted urgently to a general hospital after a haematemesis or melaena. The condition was thought to be serious enough for the subject to be included in the series if one or more of the following features were present: (1) haematemesis in excess of 1,000 ml., observed by a competent medical witness; (2) fall in systolic blood pressure to 100 mm. Hg. or below for one hour or longer; and (3) acute fall in haemoglobin to below 10.2 g./100 ml. or haematocrit to below 30%.

The patient was admitted to a special unit in a surgical ward, and after he had been assigned to an age group (under or over 50 years) a random selection was made for treatment to include gastric cooling or to be by conventional methods.

The policy of the conservative management is indicated by the following points:

(1) Blood was replaced intravenously at a rate and to an amount indicated by the patient's condition. The blood pressure, pulse rate, haemoglobin, and haematocrit were used as guides, though less importance was given to the latter two in the later stages.

(2) Intravenous dextrose/saline was given slowly (500 ml. six-hourly), usually to keep an intravenous drip infusion open.

(3) A nasogastric tube was *not* used during conservative management and was introduced only if operation (or gastric cooling) was planned.

(4) The patient was encouraged to take milk-and-water orally during conservative management (and immediately after a period of gastric cooling).

(5) Sedation, usually by intramuscular injection, was used as indicated. The drug used was decided for each patient individually: morphine, Omnopon, and a combination of chlorpromazine 25 mg. with promethazine 25 mg. were most commonly given.

(6) Antibiotics, usually by injection, were given when specifically indicated or when cover appeared particularly valuable.

Half the patients in the series were managed conservatively. For the other half gastric cooling was used in addition.

A few days subsequent to this treatment a barium-meal or Gastrografin *x*-ray examination was carried out.

Gastric Cooling Apparatus

The cooling unit in this study was the Cryocycle hypothermia system (Fig. 1). This consisted, in principle, of a pump which circulated water at a rate of about 2 litres a minute in a closed system through copper pipes in an ice-bath, and by tubes to the intragastric balloon. A double balloon of extremely thin (condom) rubber was used with a thermistor

* From a paper read at the Annual Meeting of the British Society of Gastroenterology held in London on 3–5 November 1966.
† Senior Lecturer in Surgery, University of Liverpool.
‡ Consultant Surgeon, Liverpool Regional Hospital Board.