Gut, 1976, **17**, 306-312

Influence of autonomic nerves on the internal anal sphincter in man

B. FRENCKNER AND T. IHRE

From the Departments of Clinical Physiology and Surgery, Serafimer Hospital, S-112 83 Stockholm, Sweden

SUMMARY The internal anal sphincter receives its parasympathetic nerve supply from the sacral outflow and its sympathetic supply from the thoracicolumbar outflow of the spinal cord. In order to investigate the influence of the tonic discharge of these nerves, eight healthy subjects receiving high spinal anaesthesia (T 6-T 12) and five receiving low spinal anaesthesia (L 5-S 1) were examined. Continuous recordings of anal pressure and electromyographic activity from the external sphincter were obtained during rest and during expansion of the ampulla recti by means of an air-filled balloon. The results were compared with those obtained in an earlier study from 10 subjects with a bilateral pudendal block which paralysed the striated sphincter muscles without affecting the autonomic nerve supply to the internal sphincter. Anal pressure at rest decreased significantly more with high spinal anaesthesia (32 $\pm$ 3·2 mm Hg) than with low (11 $\pm$ 7·1 mm Hg) or with pudendal block (10 $\pm$ 3·9 mm Hg) and the relaxations of the internal sphincter induced by rectal distension were somewhat smaller with high spinal anaesthesia. However, the remaining anal pressure at maximal relaxation, induced by a substantial rectal distension, was essentially the same with the three forms of anaesthesia. It is concluded that, at rest, there is a tonic excitatory sympathetic discharge to the internal anal sphincter in man. However, this seems to be without excitatory effect when the sphincter is relaxed after a substantial rectal distension. Furthermore, the results indicate that at rest there is no tonic parasympathetic discharge affecting the sphincter tone.

The anal canal is normally kept closed by the anal sphincter muscles and the pressure which can be recorded in the anal canal is higher than in the rectum and sigmoid colon (Hill *et al.*, 1960; Duthie and Bennett, 1963). It is mainly generated by the internal anal sphincter (Duthie and Watts, 1965; Frenckner and v. Euler, 1975) but the striated sphincter muscles—namely, the external sphincter and the puborectalis muscle—also display a continuous tonic electromyographic activity (Floyd and Walls, 1953). During moderate rectal distension, anal pressure falls because of a transient relaxation of the internal anal sphincter (Gowers, 1877; Denny-Brown and Robertson, 1935). This is accompanied by a reflex contraction of the striated sphincter muscles (Gaston, 1948; Goligher and Hughes, 1951), a response that has been termed the 'inflation reflex' (Ihre, 1974). Further rectal distension elicits a constant relaxation of the internal sphincter, so that anal pressure fails to return to its original level (Gaston, 1948; Schuster *et al.*, 1963; Kerremans, 1969).

The relaxation of the internal sphincter is mediated *via* local nerve pathways in the gut wall, probably involving purinergic nerves (see review by Burnstock 1972) as the nerve transmittor seems to be neither adrenergic nor cholinergic (Rayner, 1971; Garrett and Howard, 1972). However, the internal sphincter is also under complex nervous control of the extrinsic innervation from the sympathetic thoracicolumbar outflow and the parasympathetic sacral outflow. According to the classical theory of reciprocal innervation, one would expect the sympathetic nerves to be excitatory and the parasympathetic inhibitory to the sphincter. The internal sphincter's contractile response to noradrenaline in *in vitro* studies (Parks *et al.*, 1966; Friedmann, 1968) supports this but the sphincter is reported to relax after stimulation of the hypogastric nerves in man (Shepherd and Wright, 1968). However, in the cat, the opposite reaction was obtained by stimulation of the hypogastric nerves and section of the nerves

Received for publication 19 Janurary 1976

caused a reduced tone of the internal sphincter (Garrett *et al.*, 1974).

The present investigation was performed in order further to examine the influence of the tonic discharge of autonomic nerves on the internal sphincter in man. Patients were investigated who were receiving low and high spinal anaesthesia respectively before surgery. The activity of the internal sphincter was assessed quantitatively by manometric recordings of anal pressure and the results were compared with those obtained from the same subjects without anaesthesia as well as those obtained in an earlier study, in which the striated sphincter muscles were paralysed by pudendal block (Frenckner and v. Euler, 1975).

Methods

SUBJECTS

The study was undertaken on 13 adult subjects, who received spinal anaesthesia before surgical treatment (mostly hernias, varicose veins, or genital operations). Eight of them (mean age 49 years, range 21-77 years) received a high spinal anaesthesia extending to a level between T 6 and T 12 and five (mean age 51 years, range 23-73 years) received low spinal anaesthesia (L 5-S 1). The level of the anaesthesia was measured with the aid of dermatomes and their sensibility to temperature and touch. None had any history of anorectal disorders (apart from two with minor internal haemorrhoids), persistent diarrhoea or constipation. They were all informed of the nature, purpose, and possible risks of the study before giving their consent to participate.

PROCEDURE

Each subject was first examined immediately after the operation when the spinal anaesthesia was still complete. The study was then repeated a few days later. Before the operation the patients had received 10-15 mg diazepam (Valium, Roche) and this was also given before the second examination. No other drugs were given.

During the examinations, the subjects were placed in the left supine position with the hips flexed 90°. The right leg was earthed *via* a plate electrode attached to the skin. A latex balloon was then introduced through the anus and placed in the rectum 6-8 cm from the anal verge. This balloon, which measured about 3 × 1·5 cm when empty, was connected to a thin polyethylene tube about 75 cm long with an internal diameter of 2·0 mm and external of 2·2 mm. Great care was taken not to dilate the anal canal more than necessary. After this, an instrument with a water-filled balloon (Fig. 1) was introduced into the anal canal. This device consists of a metal cylinder fitted with a latex balloon and has a hole down its centre, through which the polyethylene tube to the rectal balloon passed. Both balloons were connected to electric pressure transducers (EMT 34, Siemens-Elema), in the case of the rectal balloon *via* a three-way stopcock and a 50 ml glass syringe, so that air could be inflated into the balloon. The signals from the transducers were amplified (EMT 31, Siemens-Elema) and recorded (Mingograph 42 B, Elema). The anal balloon was then placed where the highest pressure (maximal anal pressure) could be recorded. A concentric needle EMG electrode, 21 mm long, was inserted into the external sphincter via the skin 0·5-1 cm directly to the left of the anus. This was connected *via* an amplifier (Grass, model P9A) to the recorder. The signal was also recorded after integration (time constant 0·3 s; Integrator EMT 43 B, Siemens-Elema).

Before the rectal balloon was filled with air, the subject was instructed to tell the investigator as soon as he experienced a slight feeling of filling in the rectum and then, as filling proceeded, when the feeling became so pronounced that he would normally have gone to the lavatory; this will be referred to as a substantial filling of the rectum. After these instructions, the balloon was inflated with air, starting with 20 ml, then 30 ml, and then portions of 50 ml, with an interval of 30 s between each portion. It took approximately 0·5-1·0 s to fill each portion. Filling was stopped and the balloon evacuated when the subject was unable to tolerate any further expansion of the rectum, when the balloon was spontaneously defaecated, or when it had been filled to capacity (500 ml).

After each experiment, when the rectal balloon had been removed from the subject, it was inflated with air in the surrounding air to see what pressure the balloon itself generated at different volumes.

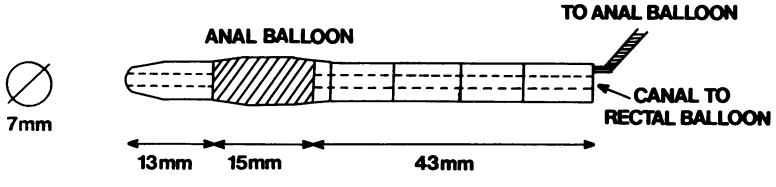

Fig 1 *Diagrammatic drawing of the device for measuring pressure in the anal canal. It consists of a metal cylinder fitted with a latex balloon.*

These pressures were then subtracted from those measured with the balloon in the rectum, in order to obtain corrected values for the pressure which the rectum exerted on the balloon (Ihre, 1974). The term rectal pressure below refers to these corrected values.

STATISTICS

Standard statistical methods have been employed using the paired *t* test when applicable. Analysis of variance has been used to compare differences in anal pressure induced by high spinal anaesthesia, low spinal anaesthesia, and pudendal block respectively. When a significant difference was obtained, the means of the three groups were compared using Scheffe's test (see Snedecor and Cochran, 1967). Data in the text and figures are given as mean ± SE (standard error of the mean).

Results

SUBJECTIVE PERCEPTIONS AND RECTAL PRESSURE

Without spinal anaesthesia, the level of rectal filling at which the 13 subjects reported a slight feeling averaged 36 ml (range 20-50). The feeling was reported as substantial at an average of 212 ml (range 150-300) and the maximal tolerable volume averaged 363 ml (range 250-500). With low spinal anaesthesia, four of the five subjects reported a slight feeling at an average rectal volume of 350 ml (range 300-450); however, this was not a rectal sensation, but rather a feeling of abdominal discomfort. The fifth subject had a diffuse feeling of filling with 20 ml inflated into the rectal balloon; however, he claimed this was not a rectal sensation and during filling to 350 ml, when the balloon was defecated autonomously, he did not experience an urge to defecate. With high spinal anaesthesia, none of the eight subjects had any feeling of the rectal balloon while this was being inflated.

Rectal pressure at different volumes was not altered by either high or low spinal anaesthesia. Slight filling corresponded to a rectal pressure of 10-15 mm Hg and a substantial filling to approximately 25-45 mm Hg.

The rectal balloon was defecated autonomously in four of the five subjects with low spinal anaesthesia (average rectal volume 400 ml, range 350-500) and in four of the eight with high anaesthesia (average rectal volume 388 ml, range 250-500). This did not happen in any of the subjects when examined without anaesthesia. However, in only two of the eight subjects, who defecated the balloon, did this occur at volumes below the maximal tolerable volume without anaesthesia.

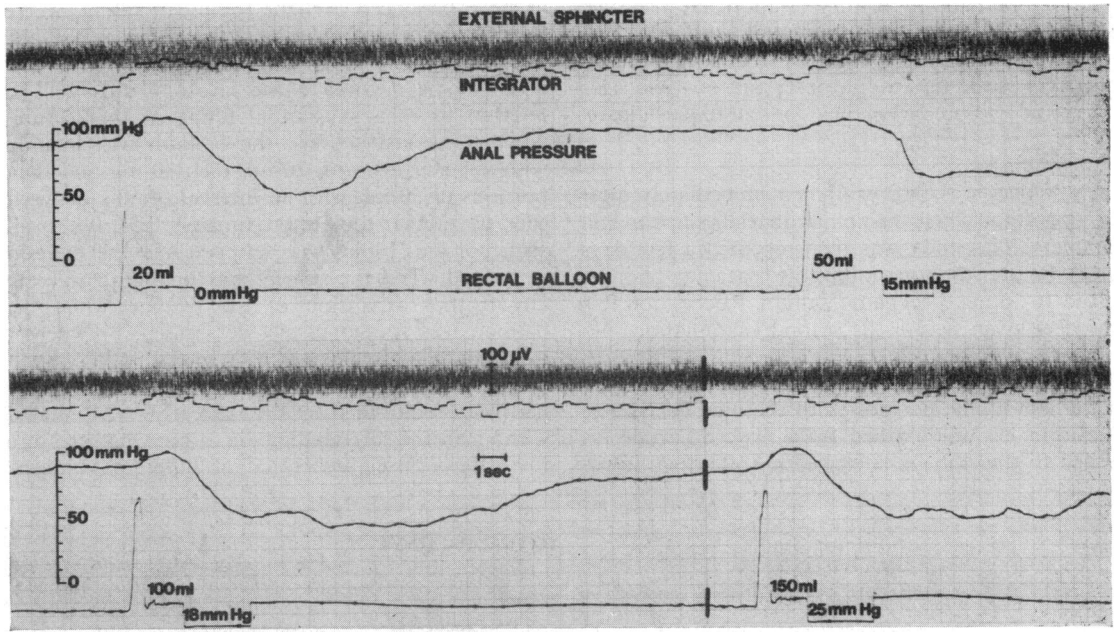

Fig. 2 *Recording of anal pressure and electromyographic activity of the external sphincter during stepwise filling of the rectum without spinal anaesthesia. Each filling of the rectum elicits an inflation reflex and a relaxation of anal pressure.*

ACTIVITY OF EXTERNAL SPHINCTER

All subjects displayed a clear-cut tonic (Fig. 2) as well as voluntary activity of the external sphincter when examined without anaesthesia. During distension of the rectum, inflation reflexes were registered in 11 subjects with 20 ml in the rectal balloon. In the other subjects, the rectal volumes required were 50 ml and 100 ml respectively. In two cases, the electrical activity in the striated muscles disappeared entirely, at rectal volumes of 150 ml and 350 ml respectively.

During spinal anaesthesia no electrical activity could be recorded from the external sphincter (Fig. 3).

ANAL PRESSURE

Maximal anal pressure at rest remained on a stable level in all subjects during as well as without anaesthesia. It averaged 40 ± 6·6 mm Hg among the patients with high spinal anaesthesia, compared with 72 ± 6·7 mm Hg among the same patients without anaesthesia, the average decrease of anal pressure being 32 ± 3·2 mm Hg. With low spinal anaesthesia maximal anal pressure decreased from 74 ± 4·3 mm Hg to 62 ± 10·9 mm Hg, the average decrease being 11 ± 7·1 mm Hg. In an earlier study (Frenckner and v. Euler, 1975) the average decrease of maximal anal pressure induced by bilateral pudendal block was 10 ± 3·9 mm Hg (Fig. 4). Thus, the three types of anaesthesia all decreased anal pressure; the decrease induced by high spinal anaesthesia was significantly ($P < 0.01$) greater than that induced by low spinal anaesthesia or pudendal block. The latter two did not differ between each other.

When the ampulla recti was filled with the first 20 ml of air, relaxations of anal pressure were recorded in all subjects but one when examined without anaesthesia (Fig. 2). Upon further rectal distension, relaxations were seen in all 13 subjects. The same applied with anaesthesia (Fig. 3), except that one subject with high spinal anaesthesia showed no relaxation of anal pressure at any rectal volume; this subject had a maximal anal pressure at rest during anaesthesia of only 10 mm Hg. In general, the relaxation became deeper and lasted longer as filling of the rectal balloon proceeded.

The manner in which the relaxations have been measured is indicated in Fig. 3. With high spinal anaesthesia, they were on average essentially the same as without anaesthesia. With low spinal anaesthesia, however, they were greater than without, but the difference did not attain statistical significance at any rectal volume. The remaining

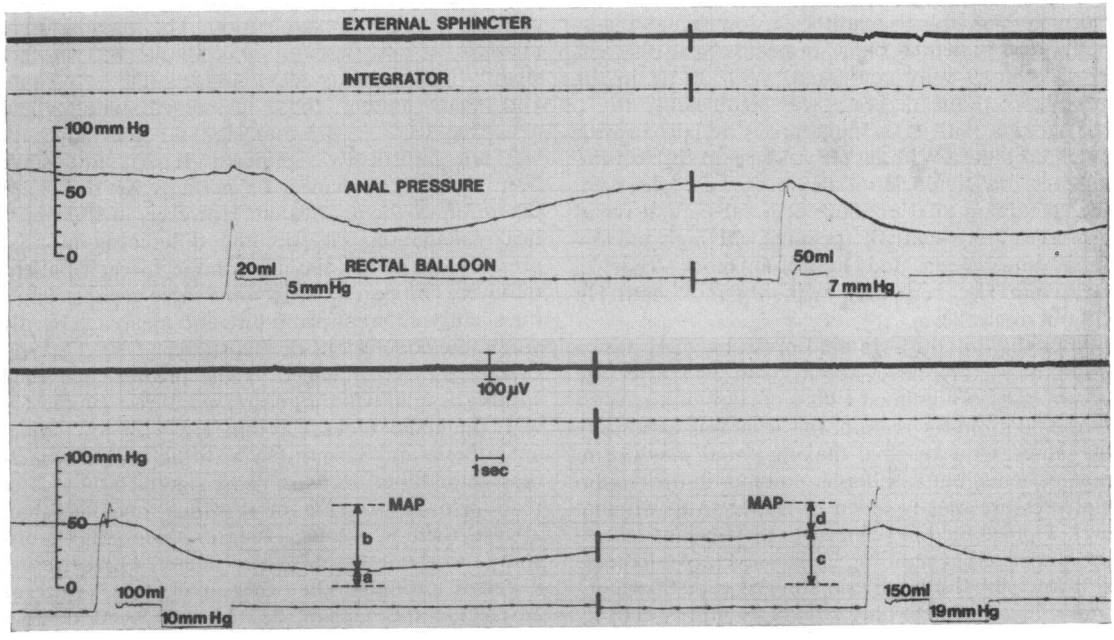

Fig. 3 *Recording of anal pressure and electromyographic activity during stepwise filling of the rectum in the same subject as in Fig. 2, now with high spinal anaesthesia (T 11). There is a diagrammatic representation of relaxation and constant relaxation: a = remaining anal pressure at maximal relaxation, b = relaxation, c = remaining anal pressure at constant relaxation, and d = constant relaxation (MAP indicates maximal anal pressure).*

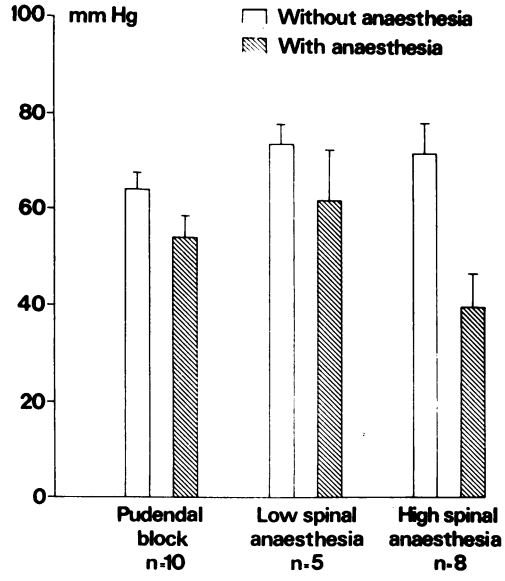

Fig. 4 *Maximal anal pressure at rest with and without anaesthesia. High spinal anaesthesia decreases anal pressure significantly more than low spinal anaesthesia or pudendal block.*

anal pressure at maximal relaxation (indicated by *a* in Fig. 3) was lower with spinal anaesthesia than without at all rectal volumes and the decreases induced by high spinal anaesthesia, low spinal anaesthesia, and pudendal block respectively at different rectal volumes were compared. With 20 ml in the ampulla recti the decrease was significantly ($P < 0.05$) greater with high spinal anaesthesia than with pudendal block. With larger volumes in the rectum, however, no significant differences were observed. The remaining anal pressure at a substantial rectal distension was essentially the same with high and low spinal anaesthesia and pudendal block—that is, 10-20 mm Hg, compared with about 35 mm Hg without anaesthesia.

After the initial fall in anal pressure, there was a return to a stable level about 20 to 30 s after the rectum had been inflated with a portion of air. With 20 and 50 ml in the rectum, this level was essentially the same as the original maximal anal pressure in most patients, but with larger volumes in the rectum anal pressure mostly failed to return to its original level, a phenomenon referred to as constant relaxation (indicated by *d* in Fig. 3). However, two patients with low spinal anaesthesia showed a distinct constant relaxation with only 20 and 50 ml respectively in the rectum. On average, the constant relaxation was greater with either low or high spinal anaesthesia than without, though the difference was not statistically significant at any rectal volume.

Discussion

The internal anal sphincter receives its parasympathetic nerve supply from the sacral outflow and its sympathetic supply from the thoracicolumbar outflow of the spinal cord. A low spinal anaesthesia therefore deprives the sphincter of its parasympathetic innervation and a high spinal anaesthesia also of its sympathetic nerve supply. In addition, the striated muscles of the pelvic floor, including the external sphincter, are paralysed. The results of this study were compared with those obtained in an earlier investigation (Frenckner and v. Euler, 1975), in which the striated sphincter muscles were paralysed by a bilateral pudendal block, without affecting the autonomic innervation to the internal sphincter. Consequently, any differences in anal sphincter function during low spinal anaesthesia compared with pudendal block must be an effect of the deprivation of the tonic parasympathetic discharge to the internal sphincter, while the differences between patients with high and low spinal anaesthesia respectively must be caused by the deprivation of tonic sympathetic discharge.

When comparing results obtained with pudendal block and spinal anaesthesia, it may be argued that the 10 subjects examined with pudendal block were younger as a group (mean age 26 years), were not premedicated and had had their anal canal slightly dilated at a digital examination. The maximal anal pressure at rest without anaesthesia was in fact slightly lower among these subjects (64 ± 3.5 mm Hg) than among those examined with spinal anaesthesia (73 ± 4.3 mm Hg), but the difference was not statistically significant. It may thus have been caused by chance, or perhaps by the slight dilatation of the anal canal. However, in the statistical calculations, it was the differences in anal pressure caused by the anaesthesia in each patient that were compared between the three groups. Thus, the slightly different procedure and mean age hardly vitiate the conclusions of this study.

Anal pressure, defined as the pressure recorded within the anal balloon, decreased at rest 10 mm Hg with pudendal block, 11 mm Hg with low spinal anaesthesia and 32 mm Hg with high spinal anaesthesia, the latter decrease being significantly greater than the other two (Fig. 4). It is thus concluded that, at rest, there is a tonic excitatory sympathetic discharge to the internal anal sphincter. Furthermore, it seems probable that this involves a direct α-adrenergic mechanism, as this has been demonstrated in the cat (Garrett *et al.*, 1974; Garrett and Howard, 1975) and as the human internal sphincter contains α-excitatory receptors (Parks, Fishlock, Cameron, and May, 1969). The present results also

indicate that at rest there is no tonic parasympathetic discharge affecting the tone of the internal sphincter.

During rectal filling, the well-documented relaxation of the internal sphincter was observed at the same rectal volumes as reported earlier (Schuster *et al.*, 1963; Kerremans, 1969). This is normally counteracted to some extent by the reflex contraction, inflation reflex, of the striated sphincter muscles, as the relaxations of anal pressure were significantly greater with pudendal block than without (Frenckner and v. Euler, 1975). Similar differences, though not statistically significant, were seen with low spinal anaesthesia, thus suggesting that the parasympathetic supply to the internal sphincter does not affect its relaxation. With high spinal anaesthesia, however, the relaxations were on average essentially as great as without anaesthesia, thus somewhat smaller than with low spinal anaesthesia or pudendal block. This is probably due to the tone of the internal sphincter being already reduced at rest.

The remaining anal pressure at maximal relaxation at a substantial distension of the rectum was essentially the same with high and low spinal anaesthesia and with pudendal block—10 to 20 mm Hg, compared with about 35 mm Hg without anaesthesia. Consequently, the sympathetic nerve supply, which at rest—according to the conclusion above—is excitatory to the internal sphincter, seems to be without effect when the muscle is relaxed by a substantial rectal distension.

The present study confirms earlier reports that anal pressure does not return to the original level after a substantial distension of the rectum. This constant relaxation of the internal sphincter is counteracted to some extent by the striated sphincter muscles, as it was more pronounced with pudendal block than without. As the same difference was found with spinal anaesthesia, although without statistical significance, it seems that the constant relaxation of the internal sphincter is normally not affected by autonomic nerve impulses.

In an earlier study, patients with transverse lesions of the spinal cord (from C 6 to L 1) were examined (Frenckner, 1975). Maximal anal pressure at rest was essentially the same as in healthy subjects. It was therefore concluded that the function of the internal sphincter normally is independent of cerebral activity. In the patients with high spinal lesions, the cerebral sympathetic connections to the spinal cord were completely interrupted, and in the patients with low lesions partly interrupted. Consequently, the tonic excitatory sympathetic discharge to the internal sphincter, demonstrated in the present investigation, must at least partly be generated from the spinal cord independently of higher cerebral connections. That spinal centres have such influence on the sympathetic activity to other effector organs is well known (for example, Thomas and Baldwin, 1968; Folkow and Neil, 1971).

Low spinal anaesthesia abolished the normal rectal sensibility, in keeping with earlier studies (Goligher and Hughes, 1951). However, a feeling of abdominal discomfort was elicited by vigorous rectal distension and it is concluded that this sensation is mediated *via* sympathetic nerves. It resembles the 'colonic sensation' described by Goligher and Hughes (1951), which was evoked by distension of a balloon in the sigmoid colon and was likewise found to be mediated *via* sympathetic nerves. High spinal anaesthesia abolished all sensibility, as expected.

References

Burnstock, G. (1972). Purinergic nerves. *Pharmacological Review*, **24**, 509-581.

Denny-Brown, D., and Robertson, E. G. (1935). An investigation of the nervous control of defaecation. *Brain*, **58**, 256-310.

Duthie, H. L., and Bennett, R. C. (1963). The relation of sensation in the anal canal to the functional anal sphincter; a possible factor in anal continence. *Gut*, **4**, 179-182.

Duthie, H. L., and Watts, J. M. (1965). Contribution of the external anal sphincter to the pressure zone in the anal canal. *Gut*, **6**, 64-68.

Floyd, W. F., and Walls, E. W. (1953). Electromyography of the sphincter ani externus in man. *Journal of Physiology*, **122**, 599-609.

Folkow, B., and Neil, E. (1971). *Circulation*. Oxford University Press: London.

Frenckner, B. (1975). Function of the anal sphincters in spinal man. *Gut*, **16**, 638-644.

Frenckner, B., and v. Euler, Chr. (1975). Influence of pudendal block on the function of the anal sphincters. *Gut*, **16**, 482-489.

Friedmann, C. A. (1968). The action of nicotine and catecholamines on the human internal anal sphincter. *American Journal of Digestive Diseases*, **13**, 428-431.

Garrett, J. R., and Howard, E. R. (1972). Effects of rectal distension on the internal anal sphincter of cats. *Journal of Physiology*, **222**, 85-86P.

Garrett, J. R., and Howard, E. R. (1975). Neutral control of the internal anal sphincter of cats after chemical sympathectomy with 6-hydroxydopamine. *Journal of Physiology*, **247**, 25-27P.

Garrett, J. R., Howard, E. R., and Jones, W. (1974). The internal anal sphincter in the cat: a study of nervous mechanisms affecting tone and reflex activity. *Journal of Physiology*, **243**, 153-166.

Gaston, E. A. (1948). The physiology of fecal continence. *Surgery, Gynecology, and Obstetrics*, **87**, 280-290.

Goligher, J. C., and Hughes, E. S. R. (1951). Sensibility of the rectum and colon. Its role in the mechanism of anal continence. *Lancet*, **1**, 543-548.

Gowers, W. R. (1877). The automatic action of the sphincter ani. *Proceedings of the Royal Society of London*, **26**, 77-84.

Hill, J. R., Kelley, M. L., Schlegel, J. F., and Code, C. F. (1960). Pressure profile of the rectum and anus of healthy persons. *Diseases of the Colon Rectum*, **3**, 203-209.

Ihre, T. (1974). Studies on anal function in continent and in-

continent patients. *Scandinavian Journal of Gastroenterology*, Suppl. 25.
Kerremans, R. (1969). *Morphological* and *Physiological Aspects of Anal Continence and Defaecation*. Arscia Uitgaven: Brussels.
Parks, A. G., Fishlock, D. J., Cameron, J. D. H., and May, H. (1966). Catecholamine release in the lower gastrointestinal tract. *Gut*, **7**, 104.
Parks, A. G., Fishlock, D. J., Cameron, J. D. H., and May, H. (1969). Preliminary investigation of the pharmacology of the human internal anal sphincter. *Gut*, **10**, 674-677.
Rayner, V. (1971). Observations on the functional internal anal sphincter of the vervet monkey. *Journal of Physiology*, **213**, 27-28P.
Schuster, M. M., Hendrix, T. R., and Mendeloff, A. I. (1963). The internal anal sphincter response: manometric studies on its normal physiology, neural pathways, and alteration in bowel disorders. *Journal of Clinical Investigation*, **42**, 196-207.
Shepherd, J. J., and Wright, P. G. (1968). The response of the internal anal sphincter in man to stimulation of the presacral nerve. *American Journal of Digestive Diseases*, **13**, 421-427.
Snedecor, G. L., and Cochran, W. G. (1967). *Statistical Methods*. Sixth ed. Iowa State University Press: Ames, Iowa.
Thomas, i. E., and Baldwin, M. V. (1968). Electrical activity of gastric musculature. In *Handbook of Physiology*, pp. 1937-1968. American Physiological Society: Washington, D.C.

The March 1976 Issue

THE MARCH 1976 ISSUE CONTAINS THE FOLLOWING PAPERS