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THE EFFECT OF SPINAL CORD INJURY ON THE MOTOR FUNCTION OF THE HUMAN COLON, AND THE ASSESSMENT OF THE INFLUENCE OF IMPLANTED RADIOFREQUENCY STIMULATING ELECTRODES.

Norman Rodger Binnie

Thesis submitted for the degree of
Doctor of Medicine (M.D.)
University of Edinburgh
1989
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I dedicate this thesis to my wife Ann.
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### Abbreviations and Units of Measurement

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<tr>
<th>Abbreviation</th>
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<tbody>
<tr>
<td>ARA</td>
<td>Ano-rectal angle (degrees)</td>
</tr>
<tr>
<td>cm</td>
<td>Centimetres</td>
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<tr>
<td>CP</td>
<td>EAS cough reflex pressure (cm H2O)</td>
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<tr>
<td>DGN</td>
<td>Dorsal genital nerve</td>
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<tr>
<td>EAS</td>
<td>External anal sphincter</td>
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<td>EMG</td>
<td>Electromyography</td>
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<tr>
<td>H2O</td>
<td>Water</td>
</tr>
<tr>
<td>HPZ</td>
<td>High pressure zone (cm)</td>
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<tr>
<td>hr</td>
<td>Hour</td>
</tr>
<tr>
<td>Hz</td>
<td>Hertz (cycles per second)</td>
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<tr>
<td>IAS</td>
<td>Internal anal sphincter</td>
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<tr>
<td>MRP</td>
<td>Maximum resting pressure (cm H2O)</td>
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<tr>
<td>mA</td>
<td>Milliamperes</td>
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<tr>
<td>MI</td>
<td>Motility index</td>
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<tr>
<td>ml</td>
<td>Millilitres</td>
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<tr>
<td>mm</td>
<td>Millimetres</td>
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<tr>
<td>ms</td>
<td>Milliseconds</td>
</tr>
<tr>
<td>MTP</td>
<td>Rectal pressure at maximum tolerable volume (cm H2O)</td>
</tr>
<tr>
<td>MTV</td>
<td>Maximum tolerable rectal volume (ml H2O)</td>
</tr>
<tr>
<td>MUPD</td>
<td>Motor unit potential duration (ms)</td>
</tr>
<tr>
<td>MVC</td>
<td>Maximum voluntary contraction pressure (cm H2O)</td>
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<tr>
<td>n</td>
<td>Number</td>
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<tr>
<td>Symbol</td>
<td>Description</td>
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<tr>
<td>P</td>
<td>Probability</td>
</tr>
<tr>
<td>P</td>
<td>Pressure (cm H2O)</td>
</tr>
<tr>
<td>PAR</td>
<td>Pudendo-anal reflex latency (ms)</td>
</tr>
<tr>
<td>PR</td>
<td>Puborectalis</td>
</tr>
<tr>
<td>r</td>
<td>Correlation coefficient</td>
</tr>
<tr>
<td>RC</td>
<td>Rectal compliance</td>
</tr>
<tr>
<td>RSR</td>
<td>Rectosphincteric reflex</td>
</tr>
<tr>
<td>s</td>
<td>Seconds</td>
</tr>
<tr>
<td>S234</td>
<td>Sacral spinal segments 2, 3, and 4.</td>
</tr>
<tr>
<td>SD</td>
<td>Standard deviation</td>
</tr>
<tr>
<td>SEM</td>
<td>Standard error of the mean</td>
</tr>
<tr>
<td>SP</td>
<td>EAS maximum voluntary squeeze pressure (cm H2O)</td>
</tr>
<tr>
<td>SSEP</td>
<td>Somatosensory evoked potential</td>
</tr>
<tr>
<td>STP</td>
<td>Rectal pressure at sensory threshold volume (cm H2O)</td>
</tr>
<tr>
<td>STV</td>
<td>Rectal volume of sensory threshold (ml H2O)</td>
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<td>V</td>
<td>Volts</td>
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<td>Vol</td>
<td>Volume (ml)</td>
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<td>μV</td>
<td>Microvolts</td>
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Spinal cord injury and pelvic nerve damage result in dysfunction of the distal colon, pelvic floor and external anal sphincter. Manometric, electrophysiological and radiographic techniques are used to assess these problems. An implanted anterior sacral nerve root stimulator is known to control bladder function for selected paraplegic subjects and offers the opportunity of studying colonic and anal sphincter function during the same electrical stimulation in man. At the outset it was desirable to assess the baseline characteristics of the colonic dysfunction in paraplegics by determination of bowel frequency, stool water content and intestinal transit time plus ano-rectal manometry and electrophysiological studies of the pelvic floor. Constipation becomes the dominant colonic problem associated with chronic spinal cord injury. The opportunity was then taken to examine the electrical stimulation of the left colon and rectum in man.

Analogous studies were then done with instrumental means of correcting faecal incontinence and constipation in man. For faecal incontinence an externally applied stimulator was devised and for intractable constipation due to non-relaxing pelvic floor muscles, a biofeedback approach was adopted. The measurement of the external anal sphincter EMG was facilitated by a new anal plug surface electrode.
ABSTRACT

The control of bowel function in spinal cord injury is the principal problem examined in this thesis. After a preliminary review of the relevant anatomy and physiology, the accepted methods for the study of distal bowel function and of the pelvic floor are reviewed.

A new anal plug electrode with longitudinal alignment of the electrode plates has been designed for surface acquisition of external anal sphincter EMG activity. This electrode is compared to an old electrode with circumferential electrode plates and is found to be significantly more sensitive in detecting external anal sphincter EMG activity during standard electrophysiological investigations such as the pudendo-anal reflex latency test. The new electrode is also compared to invasive fine wire electrodes and shows a significant direct correlation at rest, squeeze and strain activities thus validating use of the new electrode in the detection of anismus.

A study of paraplegics with and without neurogenic bowel stimulation by the Brindley stimulator compared with controls confirms a slow transit type of constipation with prolonged colonic transit time, reduced frequency of defaecation and reduced faecal water content. The Brindley S234 anterior sacral nerve root
stimulator reduces the colonic transit time and increases the frequency of defaecation but is paradoxically associated with a further decrease in faecal water content. Stimulation of the S234 anterior roots results in increased peak wave height and colonic motility index above basal activity in all areas from the transverse colon to the rectum. The area most responsive to stimulation is the splenic flexure and S3 anterior root stimulation produces the greatest motility response in the colon with a distal gradient while the rectal pressure is maximally increased by S4 anterior root stimulation. The position of the pelvic floor and pressure generated by the external anal sphincter are maximally elevated by S4 anterior root stimulation. The maximum pressure produced by the external anal sphincter during S4 stimulation shows significant direct correlation with the duration of the implant and is in keeping with hypertrophy of the sphincter. The drug cisapride reduces the oro-anal gastrointestinal transit time in paraplegic subjects by significantly reducing the colonic transit time. The drug also increases the frequency of defaecation and intestinal mixing of transit markers.

A non-invasive dorsal genital nerve stimulator was then designed to activate the external anal sphincter for the control of continence via a train of stimulations of the pudendo-anal reflex. The stimulator is shown to produce a significant rise in external anal sphincter pressure and EMG activity. An eight week course of
repeated stimulation with this device is shown to return continence to a group of women with neurogenic faecal incontinence who have an intact pudendo-anal reflex.

Finally the problem of intractable constipation with inappropriate contraction of the external anal sphincter during straining at defaecation, anismus, is shown to be amenable to correction by a course of electrical biofeedback training using a portable EMG device with a new type of anal-plug electrode.

In a final discussion, neural control is seen as a means whereby it is possible to manipulate neurogenic and reflex distal bowel activity in paraplegic man and in others with incontinence and severe chronic constipation.
CHAPTER 1.1

INTRODUCTION
The association between spinal cord injury and disruption of bowel and bladder function has been recognised for many years. The Edwin Smith Surgical Papyrus, written by an unknown Egyptian physician about 5,000 years ago, gives a clear description of the cardinal symptoms of a complete lesion of the cervical cord following dislocation or fracture of the spine; paralysis of all four limbs, complete sensory loss and loss of bladder control (Hughes 1988). In 400 BC Hippocrates described chronic paraplegia with constipation, dysuria and pressure sores. The majority of these spinal injury subjects had a short life expectancy of two or three years as a result of sepsis from infection of the bladder and kidneys and also pressure sores (Guttmann 1973). The management and quality of life of paraplegics has improved with a better understanding of the functional results of spinal cord injury and the introduction of methods to assist respiration, bladder and bowel function plus protection of the skin (Nakajima et al 1989). The parasympathetic nerve supply to the bladder and distal bowel plus the somatic efferent supply to the striated pelvic floor, urethral and external anal sphincter are transmitted via the same sacral nerve roots S2, 3 and 4. After spinal shock, reflex activity returns in the cord below the lesion and may facilitate reflex bladder emptying. Although the technique of clean intermittent self catheterisation has revolutionised bladder management in paraplegia (Maynard 1987) there are continuing efforts to achieve good bladder function without catheters. Functional electrical stimulation is the application of a controlled electrical stimulus.
to the intact peripheral or intramuscular nerve in an attempt to replace upper motor nerve control which may be lost due to spinal injury or other lesions (Allin 1986). Brindley introduced an implantable anterior sacral nerve root stimulator to give a degree of control over both bladder emptying and continence of urine for selected paraplegic subjects (Brindley 1982). Electrical stimulation of the S234 nerve roots, though intended for bladder function, could also have effects on the distal colon, rectum and pelvic floor. A group of paraplegic subjects with Brindley implants provided a unique opportunity to study the distal bowel, pelvic floor and external anal sphincter in man under the influence of electrical stimulation. A specific interest in the effects of the Brindley sacral nerve root stimulator on colorectal and pelvic floor function formed the basis for this thesis.

Investigating the large bowel function of other paraplegic subjects prompted the study of a novel pharmacological agent, cisapride, which stimulates colonic motility possibly by the selective release of acetylcholine at the myenteric plexus. Manipulation of neural control of the distal bowel has in turn led to the possibility of extending these means of bowel control in other ways, for example as an alternative to 'internal' neural stimulation, an 'external' device has been devised acting on the principle of utilisation of a specific distal spinal reflex, the pudendo-anal reflex. During these studies using conus medullaris stimulation via S234 afferents, recordings were done on the effector side of the reflex arc revealing stimulation of the
external anal sphincter and pelvic floor. It became important to measure the electrical activity of the external anal sphincter repeatedly and for this purpose a new surface non-invasive anal plug electrode was devised and compared and contrasted with an earlier version as well as with direct invasive ‘micro-wire’ contact electromyography. This anal canal surface electrode was also used in the estimation of anismus and in attempts to influence it by biofeedback techniques. Anismus was diagnosed as an obstruction to defaecation initiated by a non-relaxing pelvic floor and causing a type of obstruction with constipation often with an empty rectum.
CHAPTER I.2

IMPLICATIONS OF ANATOMY
The gastrointestinal tract may be described in terms of the foregut, midgut and hindgut. The hindgut extends distally from the middle of the transverse colon. The rectum commences at the level of the third sacral vertebral body and extends to the junction with the anal canal at the ano-rectal angle. The term anal canal was first used by Symington in 1889. The mucous membrane of the hindgut is lined by columnar epithelium down to the anal canal where stratified squamous epithelium is encountered (Stroud 1896). Between the distal portion of the columns of Morgagni is lined by non hairy stratified squamous epithelium and the last 1 to 1.5cm of the anal canal is completely lined by squamous epithelium (Goligher 1955).

The smooth muscle of the transverse, descending and sigmoid colon has a uniform inner circular layer and the outer longitudinal layer is concentrated into three band like portions called taeniae. The colon appears saccular, possibly due to an elongation of the muscle bundles of the circular muscle coat (Christensen 1981) or because the taeniae are shorter than the colon to which they are attached (Ellis 1981), causing the colonic wall to bulge out in the spaces between the taeniae. At the ano-rectal junction the internal smooth muscle layer thickens to form the internal anal sphincter and the posterior band of outer longitudinal rectal smooth muscle splits to pass anterior and posterior to the striated external anal sphincter. The anterior layer is the more substantial and forms the intersphincteric plane between the external and internal anal sphincters (Wilde 1949).
The external anal sphincter and levator ani muscle complex form the ano-rectal striated sphincter mechanism. The external anal sphincter appears in the embryo at eight weeks and seems to have a common developmental origin with the adjacent levator ani (Hamilton and Mossman 1972, Nobles 1984). The distribution of muscle in cases of anal agenesis would however suggest that the levator ani and external sphincter arise separately (Stephens and Durham-Smith 1971 and 1984). The levator ani (Thomson 1891) has the components of the puborectalis, pubococcygeus, iliococcygeus and fourthly the ischiococcygeus which is usually only rudimentary. There is some debate regarding the puborectalis muscle as to whether it should be classed as a pelvicaudal muscle or as part of the sphincter cloacae which has become tethered anteriorly. The balance of evidence to date including histology, electron microscopy, enzyme histochemical analysis and electrophysiology all support the sphincter cloacae source for puborectalis, thus separating it from the rest of levator ani (Wendell-Smith 1967, Beersiek et al 1979, Critchley et al 1980). The external anal sphincter has superficial and deep components with the superficial part extending to the subcutaneous region while the deep part fuses with the puborectalis muscle. As would be expected in muscles in a state of tonic contraction the external anal sphincter and puborectalis muscles contain a predominance of type I fibres which have a larger diameter than the type II fibres in these muscles (Beersiek et al 1979, Wunderlich and Swash 1983). In this respect these muscles differ from other striated skeletal muscles in the body.
The hindgut receives its arterial blood supply from the inferior mesenteric artery which continues directly as the superior rectal artery to supply the rectum and proximal two thirds of the anal canal where it anastomoses with the inferior rectal artery, a branch of the internal pudendal artery (Ayoub 1978). In the submucosa of the anal canal there is cavernous vascular tissue which is rich in arteriovenous anastomoses. These vascular sinuses are now recognised as a normal feature (Thomson 1975).

The intramural nerve structures of the hindgut constitute subserous, myenteric (Auerbach’s plexus), submucosal (Meissner’s plexus) and mucous plexuses (Christensen 1981). This contrasts with the more proximal caecum and appendix where the ganglion cells are arranged in an irregular pattern within the muscle layers (Emery and Underwood 1970). Abundant neurons have been described in the submucous plexus in the colon and particularly in the rectum (Duthie 1960). The myenteric plexus of the rectum continues on into the internal anal sphincter with the layer of circular smooth muscle.

Sympathetic preganglionic nerve fibres emerge in the ventral roots of all the thoracic and upper two lumbar spinal nerves as white rami communicantes. Some relay in adjacent ganglia while others pass upwards or downwards in the sympathetic trunk before relaying in other ganglia. Post ganglionic fibres in the grey rami communicantes from level T4 to L2 supplying the viscera and vessels in the abdomen may therefore be of considerable length.

The parasympathetic system has a cranial outflow in the vagus
nerve to the foregut and midgut while the sacral preganglionic outflow in the nerves S2, 3 and 4 supplies the hindgut. The sacral fibres leave the spinal nerves as the pelvic splanchnic nerves (nervi erigentes) and pass directly to the inferior hypogastric (pelvic) plexus. Some fibres synapse here but most pass through to synapse in microscopic ganglia in or near the viscus they innervate. Thus in contrast to the sympathetic system, the parasympathetic system has long preganglionic and short postganglionic fibres.

The somatic nerve supply to the anorectal region is also carried in the sacral nerve roots of S2, 3 and 4. The primary sensory neurons are located in the dorsal spinal nerve root ganglia. The peripheral processes of these cells run in the spinal nerves to terminate in specialised or free nerve endings (Duthie 1960). The impulses are carried into the spinal cord by the central processes of the ganglionic cells. The majority of these fibres ascend to reach the postcentral gyrus of the parietal lobe or enter the cerebellum after further synapsing with secondary and tertiary sensory neurons en route.

The corticospinal fibres from the precentral gyrus of the frontal lobes descend to synapse with the anterior cornual (horn) cells. The axons of these motor cells form the final common pathway to all voluntary muscle in the anorectal region (Onuf 1901, Schroder 1980 and 1981). The anterior roots of the 2nd, 3rd and 4th sacral spinal nerves descend in the "cauda equina" before exiting the spinal canal through the sacral foramina to join the lumbosacral
trunk. The pudendal nerve provides the principle innervation to the perineal muscles (Wilson 1973, Lawson 1981). The nerve passes out of the pelvis through the greater sciatic notch, crosses the ischial spine and enters the pelvis and perineum through the lesser sciatic notch. In the perineum it passes forwards to end by dividing into the perineal nerve and the dorsal genital nerve. The perineal nerve divides almost at once and the deep branch supplies the sphincter urethrae and other muscles of the anterior perineum. The superficial branch of the perineal nerve supplies the posterior skin of the scrotum. The dorsal genital nerve traverses the deep perineal pouch, pierces the perineal membrane and then penetrates the suspensory ligament of the penis or clitoris to supply the dorsal aspect of that structure. The inferior rectal nerve arises in the posterior pudendal canal and passes medially across the roof of the ischiorectal fossa to reach the levator ani and external anal sphincter. The majority of the motor supply to the levator ani complex is from direct pelvic nerve branches from above the pelvic floor while the supply to external anal sphincter remains from below the pelvic floor via the pudendal nerves (Percy et al 1981).
CHAPTER 1.3

IMPLICATIONS OF PHYSIOLOGY
The colon has evolved to serve three main functions in man, the extraction of water and electrolytes from the contents of the lumen, the abundant growth of micro-organisms and the formation of and controlled passage of faeces (Christensen 1981).

The mucosa of the colon actively transports sodium out of the lumen and water is reabsorbed by osmosis to leave the water content of stool at 70 - 75% with 150ml of water in the faeces per day (Cummings 1975). Maintenance of the sodium pump, a normal mucosa and adequate exposure of contents to the mucosa are essential to avoid excessive fluid loss. Prolonged exposure of the stool to the mucosa will result in dry inspissated stools.

Colonic bacterial flora become established early in life and the bacteria form about 30% of the total solids in adult faeces (Ganong 1975). A variety of bacteria release the gases hydrogen, methane and carbon dioxide in appreciable volumes during fermentation reactions (Levitt 1970 and 1971, Levitt and Bond 1981). Fermentable substances which are incompletely absorbed in the small bowel and are delivered to the colonic bacteria act as substrates (Levitt and Bond 1970). The volume and composition of human intestinal gas can be determined by a washout technique (Levitt 1971). The gas is eliminated from the gut by convection as flatus, bacterial or mucosal consumption and diffusion from the lumen into the blood. In the lungs the gas is cleared efficiently from the blood to the expired air in a single pass (Levitt 1981). The sampling of breath hydrogen (Welsh 1981) can be used as a sensitive detector of carbohydrate malabsorption (Metz 1976) and

The movement of colonic contents can be considered in two phases, a) the passage from the caecum to the rectum and b) evacuation of the rectum. The use of xrays and contrast medium to study the otherwise inaccessible intestines was explored in animal models before being applied in man (Cannon 1902). Three types of colonic movement have been identified by radiographic observations; retrograde propulsion (Elliot 1904), segmental non-propulsive movement (Ritchie 1968) and mass movements (Hertz 1907). Retrograde propulsion from the transverse colon to the caecum retards the forward movement of contents allowing for fermentation in the caecum and also the absorption of salt and water (Cohen 1983). Segmental movements achieve mixing and may thus facilitate the absorption of water (Ritchie 1971). Mass movements occur only a few times per day and result in the movement of a long faecal bolus over a long colonic segment (Holdstock et al 1970). Colonic electromyographic recordings reveal oscillating spike potentials which migrate at 4 cm/sec which is more rapid than the mass movement seen radiologically (William 1967) or the 1 cm/sec high pressure wave propagation detected manometrically in normal volunteers accompanied by the urge to defaecate (Narducci et al 1987). Direct correlation of the electrical patterns of activity with movements colonic content is not yet possible although the
Electrical characteristics identified suggest that the functional role of the slow wave activity is to retard movement (Taylor et al. 1975, Sarna et al. 1980) while the migrating spike bursts represent periods of release of the muscle from tonic inhibition maintained by intramural inhibitory nerves (Schang and Devroede 1983). Electromyography of the colon and rectum has been developed considerably since the original electroenterograms at the beginning of this century. There are two main components of colonic electrical rhythm, the basic electrical rhythm or "slow waves" (Taylor 1975) and the "spike bursts" (Christensen 1974). The colonic electrical activity may be studied in vitro (Chambers 1981) but the majority of studies concentrate on in vivo studies in health and in functional disorders (Szursewski 1981, Bueno et al. 1980, Daniel 1975, Frexinous and Fioramonti 1984). Evidence is now accumulating on the control of this colonic electrical activity (Sarna et al. 1980, Huizinga et al. 1986) and a degree of heterogeneity in the colon (Sandle et al. 1986). The effect of stimulation by pentagastrin 6ug/kg/hr intravenously or neostigmine 0.5mg intramuscularly or two bisacodyl suppositories has shown increased myoelectrical activity of the rectosigmoid (Taylor et al. 1974). Neostigmine and bisacodyl increased the incidence of the fast rhythm of 6 – 9Hz. Pentagastrin increased the incidence of the slow rhythm 2.5 – 4Hz but had no effect on the fast rhythm incidence. The effect of eating and gastrointestinal hormones such as gastrin I or the octapeptide of cholecystokinin has been investigated (Snape et al. 1978). It was shown that high calorie
meals (1000 calorie) increased spike and motor activity but did not alter slow wave activity while gastrin I infusion also increased spike and motor activity. Abnormal colonic myeloelectrical activity such as increased slow wave (<3Hz) activity at rest has been associated with the irritable bowel syndrome (Snape et al 1977, Taylor et al 1978, Edmonds 1970) and in chronic idiopathic constipation (Shoular and Keighley 1986). The effects of the extrinsic nerves to the colon are that the pelvic parasympathetic is stimulatory while the splanchnic sympathetic is inhibitory (Devroede and Lamarche 1974, Wingate 1938). This is reflected in the inhibitory effects of sectioning of the parasympathetic nerves of the colon (Scott 1969) and in the colonic response to autonomic drugs in general with cholinergic drugs causing activation and adrenergic agents being inhibitory (Burks 1981). Histamine may cause either response (Bennett 1966). Movement of colonic content occurs along a pressure gradient in the lumen. When the intestine is distended by a bolus the intrinsic myenteric nerve plexus facilitates inhibition of distal contractile activity with a small increase in proximal pressure to move the bolus down the gradient to the relaxed segment (Wood 1989). The manometric response of the colon to distension has been studied in diverticular disease (Smith 1981), in irritable bowel syndrome (Ritchie 1973, Varma and Smith 1985), in ulcerative colitis (Farthing 1978), in multiple sclerosis (Halden 1982), in spinal cord injury (Meshkinpour 1983). The effect of food on colonic motility has long been recognised as the gastro-colic reflex (Cannon 1902, Hertz 1913). During this
post-prandial response the colonic transit is accelerated with an increase in electrical and motor activity in the colon (Snape 1978). The absence of this post-prandial stimulation of the sigmoid colon motility in thoracic spinal cord injury subjects suggests a spinally mediated neural transmission of this response (Menardo 1984, Glick 1986) although other authors favour a hormonal mediation of the response (Snape 1978). Bowel function and dysfunction can be variously affected by drugs (Godding 1984) and intestinal motility can be facilitated or inhibited (Bennett 1966, Burks 1981, Farrar 1982). Clinically the treatment of ileus by the use of sympathetic blocking agents has been investigated (Catchpole 1969, Catchpole 1972) as has the treatment of prolonged ileus after spinal cord injury with metoclopramide (Miller 1981) and the treatment of post operative atony with cisapride (VerLinden et al 1987).

Prior to final expulsion, the colonic contents enter the rectum where continence is the normal state (Gaston 1947). The proctometrogram, rectal distension with a water filled balloon, gives reproducible measurements of rectal viscoelastic properties, capacity and compliance (Arhan et al 1976, Varma 1986) while the mathematical prediction of the capacity of an ileal pouch reservoir in restorative proctocolectomy concludes that the "W" pouch gives the greatest volume for any given length of ileum (Thomson et al 1987). Pelvic floor activity and sphincter pressures can be measured objectively by ano-rectal manometry. This is now recognised as essential in the assessment of ano-rectal physiology.
and in detecting abnormalities of function (Henry et al 1985, Snooks and Swash 1986). Ano-rectal manometry can also be used to assess the function of the anal sphincters after ano-rectal surgery such as sphincter saving resections of the rectum (Goligher 1951), colo-anal anastomosis (Lane and Parks 1977, Varma and Smith 1986) and surgical treatment for faecal incontinence (Browning and Parks 1983, Browning and Motson 1983, Keighley and Fielding 1983, Browning et al 1984). In the normal resting state there is a zone of high pressure in the anal canal which is maintained by the tonic activity of both the smooth muscle internal anal sphincter and the striated external anal sphincter (Taverner and Smiddy 1959). The puborectalis is also in a state of tonic contraction, maintaining an acute angle between the rectum and anal canal (Hardcastle and Parks 1970). This angle forms a flap valve mechanism which prevents the rectal contents having direct access to the anal canal (Parks, Porter and Hardcastle 1966). The external anal sphincter provides about 15% of the maximum resting pressure in the anal canal (Frenckner and Von Euler 1975) and there is an increase in pressure related to upright posture and also in response to sudden rises in intra-abdominal pressure such as when coughing (Parks et al 1962). The pubo-rectalis and the external anal sphincter contain one or more muscle spindles but the factors responsible for maintaining this continuous activity have not yet been determined (Winckler 1958, Swash 1982). The acute phase of spinal cord injury affects the reflex activity of the external anal sphincter by reducing the
response pressure (Melzak and Porter 1964). The resting pressure in the anal canal is also reduced in the acute phase (Meunier and Mollard 1977) but not in chronic paraplegia (Denny-Brown and Robertson 1935, Wheatley 1977). The receptors responsible for awareness of rectal distension probably do not reside in the rectum but are located in the pelvic floor muscles. Stretch receptors have been identified in the levator ani (Winkler 1958). The differentiation of the nature of rectal contents may be a function of intrarectal pressure (Goligher 1951) but may also depend on the contents entering the distal anal canal to make contact with sensory receptors there (Duthie 1963). Following anterior resection of the rectum with colo-anal anastomosis subjects can regain perception of "rectal" sensation (Lane and Parks 1977). However some subjects after colo-anal sleeve anastomosis have incontinence due to loss of precise perception, being unable to distinguish between flatus and faeces (Goligher 1951). The internal anal sphincter undergoes rhythmical changes in resting pressure reflected in the slow (10 - 20/min) and ultra slow (<3/min) waves demonstrated by Bouvier and Gonella (Bouvier and Gonella 1981). The recto-sphincteric relaxation reflex results in relaxation of the internal anal sphincter when the rectum is distended (Gowers 1877). Continuous rectal distension initially causes sphincter relaxation but the muscle gradually returns to its resting tone despite continuous rectal distension. The reflex is independent of higher nerve centres and a normal reflex is obtained in cases of thoracic cord transection (Glick 1986), cauda equina lesions
(Gowers 1877) and hypogastric nerve resections (Denny-Brown and Robertson 1935). Although dependent on intramural neural activation (Lubowski 1987) the reflex can be modulated by sacral cord activity (Meunier and Mollard 1977). The reflex is absent if the myenteric plexus is incomplete as in Hirschsprung’s disease (Lawson and Nixon 1967). A 50% reduction in resting anal pressure has been recorded after internal anal sphincter myotomy (Bennett and Duthie 1964) when measured with open ended tubes. Pathological reduction in ano-rectal manometric resting pressures are recorded in adult neurogenic faecal incontinence (Parks 1975, Read et al 1979, Keighley and Fielding 1983, Snooks and Swash 1986). There is also reduced anal sphincter resting pressure in traumatic anal incontinence (Christiansen and Pedersen 1987), rectal intussusception (Frenckner and Ihre 1976) and faecal incontinence following some cases of anal dilatation (Macintyre and Balfour 1972, Snooks et al 1984). A less dramatic reduction in pressure is seen in subjects with pruritis ani (Allan et al 1987). There is however increased anal canal resting pressure in cases of fissure-in-ano (Gough and Lewis 1983, Weaver et al 1987). At times of defaecation although the internal anal sphincter may be relaxed the rectal contents cannot be expelled unless the puborectalis and external anal sphincter are also relaxed (Preston and Lennard-Jones 1985). Inappropriate rises in anal sphincter pressure when straining at stool are found in subjects with anismus, the spastic pelvic floor syndrome (Preston and Lennard-Jones 1985, Read et al 1986, Meunier 1986, Bleijenberg and Kuypers 1987, Martelli et al
1987) and also in subjects with solitary rectal ulcer syndrome (Womack et al 1987). The act of defaecation involves straining to raise intra-abdominal pressure while at the same time inhibiting the puborectalis and external anal sphincter to allow widening of the ano-rectal angle and passage of stool through the anal canal. Following defaecation there is a rapid return to the normal resting sphincter pressures. The balloon xray-proctogram technique can give objective measurement of rectal position and capacity (Preston 1984), while the ano-rectal angle and flap valve theory of ano-rectal continence is assessed by a similar balloon technique (Bartolo 1986). Except at the time of defaecation when the striated sphincters are inhibited, there are continence protecting reflexes which cause contraction of these muscles in response to sudden rapid rises in intra-abdominal pressure such as coughing or sneezing. There is a similar transient response in the external anal sphincter to sudden rectal distension (Melzak and Porter 1964). A further reflex mechanism is active during micturition, the vesico-anal reflex, which allows the internal anal sphincter activity to be increased while the external anal sphincter is inhibited with the external urethral sphincter. This reflex therefore provides for anal continence during the act of micturition (Salducci 1982).

Electromyography (EMG) in various forms is used for investigating neuro-muscular disorders especially where the nerve supply of muscle is damaged (Swash 1985). EMG recordings depend on muscle cell membrane electrical potential changes which can be detected by
electrodes. After suitable filtering and amplification these electrical signals can be displayed on an oscilloscope, recorded on paper or undergo further electronic manipulation and analysis. The electromyographic activity of human muscle was first recorded with a string galvanometer (Piper 1908). However it was not until the advent of concentric needle electrodes that the technique was brought into clinical use (Adrian and Bronck 1929). Muscle fibre membrane is depolarized at the motor end plate by acetylcholine released from the presynaptic motor nerve by the nerve impulse. The muscle action potential passes along the muscle fibre at 3 m/s and causes actin myosin contraction. This contraction plus the repolarization of the membrane after the contraction is energy dependent. The term "motor unit" refers to the anterior horn cell, its axon and axonal branches, end plates and muscle fibres innervated by this cell. EMG recordings may be acquired using surface electrodes, concentric needle or single fibre electrodes. Parameters in the study of EMG action potentials are amplitude, duration, number of phases and firing rate which give information about innervation and functional state of individual motor units of the muscle. Alternatively quantitative measurements of electrical activity can be used as a functional test of muscle activity (Lenman et al 1983). Factors such as contact area and muscle fatigue affect surface EMG recordings (Bigland-Ritchie 1979). A variety of electrophysiological tests are currently available for the investigation of neurogenic disorders of the pelvic floor and sphincter mechanisms (Swash 1985, Percy et al 1981). EMG has been
invaluable in the investigation of ano rectal incontinence (Parks 1975, Snooks and Swash 1986). The persistence of reflex EMG activity of the external anal sphincter in spinal injured man has been confirmed (Melzak and Porter 1964). The neural control of internal anal sphincter function has also been investigated using EMG (Lubowski 1987). In cases of abnormal function, electromyography has helped define a group of young women who have inappropriate contraction of the anal sphincters when straining to defaecate which then results in severe constipation (Read et al 1986). There are often EMG abnormalities to be found in the anal sphincters of subjects with the solitary rectal ulcer syndrome (Womack et al 1987). An EMG study of the abdominal muscles in tetraplegic patients has shown decreased activity (Goldman et al 1986). Simultaneous recording of EMG and mechanical activity from postural muscles in paraplegic patients has shown that unexpected beneficial effects may be obtained by delivering stimuli to a diseased nervous system (Harris and Walsh 1981). They were able to show that by thumping both thighs with his fists a paraplegic male was able to produce reflex extensor spasm of the knees. Using this technique he could stand with the aid of a stick or a little help from another person. The EMG properties of skeletal muscle have been shown to change following spinal cord injury as the muscle adapts to its altered nerve supply (Lieber 1986). A similar pattern of change in muscle EMG is noted after myelotomy in paraplegic patients (Scelsi et al 1986).

Closely related to electromyography is the field of nerve
conduction studies. This offers a means of assessing nerve conduction velocities and locating the site of any delay (Brindley 1981, Kiff and Swash 1984). After spinal cord injury the level of lesion can be defined (Gruninger and Ricker 1981, Lehmkuhl et al 1986) as can the presence of syringomyelia (Di Benedetto and Rossier 1977). Slowed pudendal nerve conduction is seen in neurogenic faecal incontinence (Kiff and Swash 1984) while other authors have shown a delay in the pudendo-anal reflex arc in similar subjects (Varma and Smith 1985).

A better understanding of these colo-recto-anal systems will aid in the wider understanding of normal physiological function and may allow categorization of abnormalities in such a way as to help towards their appropriate management.
CHAPTER 1.4

GENERAL METHODS
The accepted methods for studying colonic and ano-rectal function can be broadly grouped into categories concerning intestinal transit time, colo-ano-rectal manometry, electrophysiology and radiological studies. These motor and sensory investigations have been shown to be valid when performed by different examiners in different centres (Rogers et al 1989). Most of the materials used in this thesis are available commercially and the sources are given in Appendix 1. In other cases the item was either made locally or adapted to specification locally.

1. Transit Time

Gastrointestinal transit time (Smith 1980) can be divided into Oro-Caecal Transit Time and Oro-Anal Transit Time. Subtraction of the former from the latter allowed calculation of the time in the colon or Colonic Transit Time.

Oro-Caecal Transit Time

The method used was that of Bond et al as it provides a simple, safe, non-invasive means of studying small bowel transit in man. It is easily repeated and does not involve ionising radiation (Bond et al 1975). Other available methods such as using radio-telemetry capsules (Bloom 1965) and isotopes (Read 1980) are less simple and less easy to repeat, particularly in compromised spinal cord injured subjects. Orally administered nondigestible carbohydrate lactulose is fermented in the caecum and within ten minutes of reaching the caecum the product gas hydrogen appears in the exhaled
breath (Bond 1975). The hydrogen concentration is measured in parts per million (ppm) on a GMI breath hydrogen analyser (Appendix 1). Results are given numerically on a visual display and as a permanent record on heat sensitive paper. The analyser has a low range 0 - 50 ppm and a high range of 0 - 250 ppm. The analyser has to be zeroed at the beginning of each session and then calibrated using gas samples of standard hydrogen concentrations of 50 ppm and then either 93 ppm or 96 ppm. The analyser has to be permanently connected to the mains electricity and switched on, in order to maintain the stability of the electro-cell. There is an internal rechargeable battery which will last for up to 24 hours, however on reconnection to the mains a further 24 hours are required for the cell to restabilise. The manufacturers claim an accuracy of between 2 and 5 ppm provided that all instructions are adhered to and that the analyser is serviced regularly every four to six months by their engineer. The subject is prepared by fasting overnight and a baseline end expiratory 20 ml breath sample is taken prior to breakfast. The breath sample is collected in a standard 20 ml disposable plastic syringe which is attached to a Y connector. The Y connector has a mouth piece and a 3 way tap attached to the other two ends respectively. The subject blows into the mouth piece with the 3 way tap open to the Y connector and the atmosphere. The subject indicates the end of expiration and the 3 way tap is closed off to the Y connector leaving the syringe still open to the Y connector. At this point the subject continues to blow and the syringe plunger is withdrawn to fill the syringe with
end expiratory breath before the syringe is disconnected from the Y connector with its 3 way tap closed off. The lactulose, 10g in 15 ml water, is given with a standardised breakfast of cereal with milk followed by tea or coffee plus buttered toast with marmalade (500 calories). The patient does not eat or drink and smoking cigarettes is forbidden thereafter until the test is completed.

The end expiratory breath samples are repeated every 15 minutes until the rise in Hydrogen ppm due to the arrival of the lactulose in the caecum is recorded. At each breath sampling time, two 20 ml samples marked 1 and 2 were taken and run through the analyser in the order collected. After the rise in breath hydrogen due to the lactulose reaching the caecum was recorded, sampling was continued for a further one hour to confirm that this was not a spurious rise in breath hydrogen.

Oro-Anal Transit Time

The method used for this measurement was that of Hinton, involving a single ingestion of fifty, 2mm cube, radio-opaque pellets with a standard meal and recording the passage of these marker pellets per rectum (Hinton 1969). This method avoids exposure of the subject to Xray radiation, is acceptable to patients and is easily repeated. Various other methods are available for measuring whole gut transit time such as using radio-telemetry capsules (Bloom 1965) and isotope markers (Read 1980). Using the Hinton method the stools are collected from the time of ingestion of transit markers and are stored in a sealed
radiolucent plastic container at -20 degrees centigrade before being Xrayed in the container. The content of transit markers is revealed on the Xray film. This process is continued until at least 80% of the pellets have been recorded. The time from ingestion to the 80% recovery point is taken as the oro anal transit time. In the case of paraplegic subjects with spinal cord lesions above T3 the stools were collected by an attendant while subjects with more caudal spinal cord lesions could on the whole collect their own stool specimens.

2. Ano-rectal Manometry

The intraluminal pressure in the colon, rectum and anal sphincters can be recorded using such devices as perfused open ended tubes (Phillips 1965) or closed balloon systems attached to pressure transducers (Varma 1986, Hancock 1976). More recently solid state micro pressure transducers have become available (Varma 1984).

Anal Sphincter Pressure Profile

The method used was that described by Varma and Smith which assesses the anal canal pressure in cm of water pressure by using a micro balloon water filled system (Varma and Smith 1984). The subject is placed in the left lateral position and the microballoon is inserted into the anal canal until it lies 6cm from the anal margin. The balloon is then withdrawn in steps of 1cm and the pressure recorded for 30sec at each stage. The recording shows the
pressures from the rectum through the combined length of the internal and external anal sphincters at 1cm intervals and reveals the High Pressure Zone (HPZ) or effective Anal canal Sphincter Length (ASL). The Maximum Resting Pressure (MRP) in the anal canal is recorded and is normally at 1 or 2cm from the anal margin where the internal and external anal sphincters overlap. The point of MRP is used to determine the Maximum Voluntary "squeeze" Contraction pressure (MVC) of the external anal sphincter if the subject has voluntary control of the external sphincter. The Cough Reflex contraction Pressure (CRP) of the external sphincter in response to coughing is also recorded at the MRP point in the anal canal.

The Procto Sphincteric Reflex

This reflex is tested by placing the anal sphincter pressure balloon in the anal canal at the point of maximum resting pressure while a second balloon within the rectum is distended with 50 ml of air. The reflex is deemed to be intact if there is a reflex fall in the internal anal sphincter pressure of at least 10% of the maximum resting pressure.

The Proctometrogram

The method used for this measurement was that described by Varma and Smith which involves instilling water at 67ml/min into a highly compliant intra-rectal balloon while recording the pressure within this rectal balloon using a micro balloon pressure transducer (Varma and Smith 1986). The subject is placed in the
left lateral position and digital rectal examination is performed to ensure an empty rectum. The proctometrogram balloon is then held at the anal level to permit zeroing of the pressure transducer before being inserted into the rectum. The basal intrarectal pressure is now recorded before and instillation of water into the rectal balloon begins. The intra-rectal pressure is recorded continuously on heat sensitive paper by a chart recorder with a minute time marker. The time marker on the tracing gives an indication of the volume being instilled. The Volume of Sensation (VOS) is taken as that volume when the rectal contents are first perceived. The Volume of Continuous Sensation (VCS) is when there is continuing urge to defaecate but not yet at maximum tolerable rectal volume. The maximum rectal volume or capacity (MRV) is taken at the point when the subject cannot tolerate any more distension or the balloon is expelled spontaneously or there is evidence of autonomic dysreflexia whichever appears first. The volume of water instilled can be estimated from the time taken by the pump and the continuous infusion rate and is measured exactly in a measuring cylinder by syphoning off the rectal balloon at the end of the procedure.

Colonic Motility Index

The motility of the recto-sigmoid region was measured using the method described by Connell (Connell et al. 1963). Subjects were prepared by avoiding laxatives for two days and were fasted for at least two hours before the study began. They were placed in the
left lateral position in quiet surroundings for the study. A water
filled system was used with three 5ml balloons in series 5cm apart
and attached to separate external force transducers by fine bore
non-compliant plastic tubing. A permanent paper record was obtained
from the multichannel recorder (Appendix 1). A rigid sigmoidoscope
was passed to beyond the recto-sigmoid junction with minimal air
insufflation before the recording balloons and tubing were passed
through the sigmoidoscope and left in position at 25, 20 and 15cm
from the anal margin. The three pressure recorders thus placed
could record simultaneously and records could be made during
periods of rest, post prandially, after intravenous injections of
stimulatory drugs and after anterior sacral nerve root stimulation.
Measurements which could be made included Percentage Activity (PA)
which was the period during the recording time, usually 30 min,
when the colonic pressure was above 10cm of water pressure and did
not coincide with respiration or movement. The colonic pressure
wave Peak Pressures (PP) could be measured and were recorded. The
colic Motility Index (MI) was derived from the product of the
average height of the waves and the activity period in which they
were produced. This latter calculation was simplified by the use of
a micro-computer. On occasions the direction of the pressure
waves in relation to the transducers could be determined on
recordings made with higher speed paper tracing. Varma 1986, 

3. Electrophysiology
Pudendo-anal reflex

The pudendo-anal reflex arc incorporates the dorsal genital nerve, the S234 components of the spinal cord and the efferent nerves to the pelvic floor and external anal sphincter. The integrity and latency of the reflex arc can be assessed using the method which was developed in this laboratory by Varma and Smith (Varma and Smith 1986). The subject lies in the left lateral position on an insulated examination couch. Using the Medelec MS92a apparatus (Appendix 1) a ground electrode is wrapped around the subject's right thigh and an anal plug electrode is coated with electrode gel and placed in the anal canal. The dorsal genital nerve is stimulated at the base of the penis or clitoris using saline soaked felt electrodes and the response in the external anal sphincter is recorded via an anal plug surface electrode. At least one hundred impulses are applied and the response signals are digitized and averaged to eliminate noise from the final composite signal. The latency from stimulus to response is measured and termed the Pudendo-Anal Reflex Latency (PARL).

Motor Unit Potential Duration

External anal sphincter Motor Unit Potential Durations (MUPD) were recorded using the method described by Bartolo et al (Bartolo et al 1983). The subject is placed in the left lateral position and a ground electrode placed around the uppermost thigh before a DISA concentric needle electrode (Appendix 1) is inserted without
anaesthetic into the external anal sphincter. The EMG monitor was setup with a display time base of 10 ms/cm, a gain of 100uV and filter settings of 20Hz - 10kHz. The trigger and delay facility of the Medelec MS92a EMG apparatus enabled individual motor unit potentials to be identified and their stability assessed. At least 100 consecutive action potentials were recorded on one channel before being repeated on the second channel and then superimposed to confirm that it was the same motor unit on each recording. The action potential duration was now measured from the first deflection from the baseline until the return to the baseline. The mean duration of twenty action potentials was taken as the Mean Motor Unit Potential Duration of the external anal sphincter and can be used as index of neuropathy (Buchtal and Pinelli 1953). The number of phases of each action potential was assessed as the number of phases between crossings of the baseline a polyphasic unit contained more than four phases.

Single Fibre Density

The external anal sphincter single fibre density was measured by the EMG technique described by Stalberg and Thiele (Stalberg and Thiele 1975). This involved the use of a single fibre EMG needle electrode (Appendix 1) with a leading-off surface of 25um diameter and uptake radius of about 270um. The Medelec MS92a EMG apparatus was set up to receive with a time base of 10ms/cm and a gain of 100uv with amplifier filter settings of 500Hz - 10kHz. Using the trigger and delay function of the EMG recorder the action
potentials that were derived from muscle fibres belonging to the same motor unit could be identified and counted. The mean number of single muscle fibre potentials recorded in twenty different positions within the external anal sphincter circumference was taken as the single fibre density of the external anal sphincter. The needle was inserted into the four quadrants and five readings taken at each insertion. The normal fibre density for the external anal sphincter is 1.5 +/- 0.16 (Neill and Swash 1980).

4. Xray Studies.

Ano-rectal Angle and Pelvic Floor Position.

These radiological studies were performed using a modification of the defaecography technique described by Mahieu, Pringot and Bodart in 1984. The spinal cord injury subjects were studied in the left lateral position which is accepted as not being ideal for studying the pelvic floor although measurements of the descent of the perineum was defined originally by measuring radiologically the drop in anorectal junction on straining with subjects in the left lateral position with Barium soaked gauze to outline the rectum and anal canal (Parks, Porter and Hardcastle, 1966). Hardcastle and Parks 1970 defined the position of the anorectal junction < 1.8 cm below the pubococcygeal line, although the pelvic floor was again not stressed in these studies in the left lateral position. Although the resting position may vary with the patient sitting on a commode the descent of the ano-rectal
junction on straining should not exceed 2cm (Mahieu, Pringot and Bodart 1984a and 1984 b). Balloon proctography was developed as a static examination, needing only minimal equipment and being readily tolerated by patients. Advantages are that the anorectal angle and level of the pelvic floor are shown clearly and are easily measured. Disadvantages are the lack of a recording of the movements of the balloon during defaecation and the possibility that the balloon may behave in an unphysiological manner hiding minor abnormalities. Magnification factor of about 1.3 and no allowance has been made for the figures calculated here. In the xray studies done for this thesis static xray films were taken in the left lateral position using balloon proctography. The rectal capacity or volume was first established by the reproducible continuous infusion proctometrogram technique (Varma and Smith 1986). The rectal balloon was then emptied before being refilled to 25% of rectal capacity with an xray contrast fluid (Hypaque). A marker was place over the pubic bone and also at the anal opening to ease the later marking of the xray films. The measurements and calculations performed on the xray films included:

1 The anorectal angle as the angle between the luminal axis of the anal canal a line drawn along the lower border of the distal rectum. It can be measured at rest and during straining with normal values being 94° +/- 1.5° at rest and 137 +/- 1.5° during straining (Mahieu, Pringot, Bodart 1984a and 1984b).

2 The pelvic floor position. At rest this is the vertical distance between the pubococygeal line and the anorectal junction. It must
be measured from a fixed point on the puborectalis line. The normal pelvic floor must not descend more than 2 cm during defaecation (Mahieu, Pringle, Bodart 1984a and 1984b).
CHAPTER I.5

A NEW ANAL PLUG ELECTRODE FOR DETECTING EMG ACTIVITY IN THE

EXTERNAL ANAL SPHINCTER
Summary

A new anal plug electrode has been developed in which the two electrode plates were orientated in the long axis of the anal canal and were thus separated along the length of the circular external anal sphincter muscle fibres. This design principle was hoped to give improved external anal sphincter EMG recording during ano-rectal electrophysiological investigations when compared to the older anal plug electrode design with circular electrode plates lying parallel to the sphincter muscle fibres. The new surface electrode was compared to invasive fine wire stainless steel electrodes and showed significant direct correlation for resting EMG ($p < 0.01$), squeeze EMG ($p < 0.001$) and strain EMG ($p < 0.01$) in a group of eight patients. In another group of patients the new electrode was compared to an old surface electrode and showed a significant increase in the amplitude in the EMG signals recorded for rest, squeeze and strain ($p < 0.01$). During pudendo-anal reflex latency testing the incoming signal amplifier sensitivity was able to be significantly reduced ($p < 0.01$) from $12.4\mu V \pm 5.3\mu V$ to $134.3\mu V \pm 51.8\mu V$ while continuing to record clear EMG responses with the new electrode. In a further 12 cases where the pudendo-anal signal was either absent or not detected with the old standard electrode, the new electrode was able to detect a low amplitude response in 11. Inappropriate contraction of the external anal sphincter when straining at stool, anismus, was detected using the new electrode in 52 patients investigated for intractable constipation but in 12 of them the EMG abnormality was not
detected when they were tested with the old electrode. This new electrode facilitates surface acquisition of EMG activity in the external anal sphincter and enables better recognition of functional abnormalities such as anismus.

Introduction

Muscle tissue is electrically anisotropic with the longitudinal conductivity being five to fifteen times larger than in the transverse direction. Bipolar surface electrodes for recording striated muscle EMG are therefore usually placed along the length of the muscle fibres (Lindstrom and Petersen 1983). The external anal sphincter is composed of striated muscle with the fibres being arranged in an annular form (Thomson 1899). The commercially available anal plug electrodes e.g. Disa 13K78/79 (Disa, Electronics, Bristol), have two circular electrode plates which are circumferential on the anal plug and thus lie parallel to the external anal sphincter muscle fibres. The new anal plug electrode (Unived 1987) has the two electrode plates placed along the longitudinal axis of the plastic plug and equally separated on the circumference. The electrode plates are thus separated along the length of the external anal sphincter fibres (figs. I.5.1 and I.5.2). Anorectal electrophysiological investigations include i) the pudendoanal reflex latency test (Varma et al 1986) and ii) the integrated EMG activity of the external anal sphincter at rest, maximum voluntary squeeze and during straining at stool (Binnie et al 1988). The pudendoanal reflex assesses the integrity of the arc
from the dorsal genital nerve, to the S234 component of the sacral cord and the efferent pudendal nerve to the external anal sphincter. The normal response to straining at stool is relaxation of the pelvic floor and external anal sphincter with a reduction in the EMG activity. Anismus or inappropriate contraction of the external sphincter can be recognised by detecting a rise in external anal sphincter EMG activity when straining at stool. The aim of this study was to evaluate the new anal plug electrode by comparing the results of the EMG studies described above when performed using the new anal plug electrode and the conventional one as well as comparing the new anal plug electrode with fine wire stainless steel electrodes inserted directly into the muscle.

Patients

The first group were involved in the control study of comparing the new surface electrode to the invasive fine stainless steel wire electrodes. The group was composed of eight consecutive subjects attending the ano-rectal laboratory. There were three males and five females with a mean age of 52yrs (range 19 - 83yrs). Further details of presenting complaints etc are given in Table I.5.1.

The second group of subjects involved in this study were also seen at the laboratory for investigation of a variety of anorectal dysfunctions and gave informed consent for the procedures involved. There were 117 in total with 94 females and 23 males. Patient details are given in table I.5.2.
### Control Study of Wire and Surface Electrodes

<table>
<thead>
<tr>
<th>Age</th>
<th>Sex</th>
<th>Presenting Complaint</th>
<th>Duration of Presenting Complaint</th>
</tr>
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<tbody>
<tr>
<td>47</td>
<td>F</td>
<td>Constipation</td>
<td>2 years</td>
</tr>
<tr>
<td>19</td>
<td>F</td>
<td>Constipation</td>
<td>1 &quot;</td>
</tr>
<tr>
<td>44</td>
<td>M</td>
<td>Faecal Incontinence</td>
<td>1 &quot;</td>
</tr>
<tr>
<td>36</td>
<td>F</td>
<td>Constipation</td>
<td>4 &quot;</td>
</tr>
<tr>
<td>49</td>
<td>F</td>
<td>Faecal Incontinence</td>
<td>7 &quot;</td>
</tr>
<tr>
<td>65</td>
<td>M</td>
<td>Faecal Incontinence</td>
<td>2 &quot;</td>
</tr>
<tr>
<td>72</td>
<td>M</td>
<td>Pre-op. Colectomy</td>
<td>4 &quot;</td>
</tr>
<tr>
<td>83</td>
<td>F</td>
<td>Faecal Incontinence</td>
<td>16 &quot;</td>
</tr>
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TABLE 1.5.2
Subjects in study comparing old and new anal plug electrodes.

<table>
<thead>
<tr>
<th>Subject Group</th>
<th>No.</th>
<th>Sex</th>
<th>Age</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pudendo-anal reflex (PAR) present with old electrode</td>
<td>23</td>
<td>M</td>
<td>36.1 +/- 6.2yrs</td>
</tr>
<tr>
<td></td>
<td>94</td>
<td>F</td>
<td>55.4 +/- 9.3yrs</td>
</tr>
<tr>
<td>PAR absent with old electrode</td>
<td>14</td>
<td>F</td>
<td>66.5 +/- 8.6yrs</td>
</tr>
<tr>
<td>Chronic constipation (with anismus diagnosed with new electrode)</td>
<td>14</td>
<td>M</td>
<td>32.4 +/- 5.7yrs</td>
</tr>
<tr>
<td></td>
<td>38</td>
<td>F</td>
<td>48.2 +/- 11.5yrs</td>
</tr>
</tbody>
</table>
Materials

Electrode

The fine wire stainless steel electrodes (Basmajian and Stecko 1963, Womack et al. 1985) were placed in a hypodermic needle with the ends protruding past the end of the needle. The wires were insulated with a teflon coating and approximately 0.25cm of the tips were bared and hooked over in order to be able to retain position in the muscle on withdrawal of the needle (fig. I.5.3).

The electrode referred to as the old electrode is the Disa 13K78/79 (Disa, Electronics, Bristol) as shown in fig. I.5.2.

The new anal plug electrode has two 0.25cm x 2cm stainless steel electrode plates placed in parallel on the longitudinal axis of the neck of the plastic anal plug separated equally by 1cm on the circumference. The electrode plates are thus separated in the direction of the external anal sphincter fibres (fig. I.5.1) (Appendix 1). The plug is constructed out of solid PVC and is machined to the appropriate shape. The overall length is 7cm with the bulbous distal end 1.5cm in diameter and the neck 0.5cm in diameter. These dimensions are similar to other available anal plug electrodes with the exception that the other anal plug electrodes have two circular electrode plates which are circumferential on the neck of the anal plug and thus lie parallel to the external anal sphincter fibres (fig. I.5.2).
FIGURE I.5.1
Alignment of electrode plates in old and new anal plug electrodes.

External Anal Sphincter

Old Electrode

E.M.G.

External Anal Sphincter

New Electrode

E.M.G.
FIGURE 1.5.2

Photograph of old and new anal plug electrodes.
FIGURE I.5.3
Photograph of teflon coated fine wire stainless steel electrodes.
Methods

Integrated EMG

The subject lies in the left lateral position with a ground electrode on the right thigh. The autoclaved sterilized fine wire stainless steel electrodes with the tips hooked (fig. I.5.3) are placed into the external anal sphincter midline posteriorly by insertion with a hypodermic needle to a depth of 1.5cm. The needle is then withdrawn leaving the electrodes in place in the external anal sphincter muscle. The electrode wires are connected to an isolated EMG integrator (Ormed 4880, MX216) and the recorder is calibrated to read the required EMG range (0 - 50 uV). After several minutes to allow relaxation to return after insertional EMG activity the "Resting EMG" activity in the external anal sphincter was recorded. The subject was then asked to contract the external sphincter and the "Squeeze EMG" was recorded. The subject was finally asked to strain as if at stool and the "Strain EMG" was recorded (fig. IV.1.4). This was repeated twice to confirm the recordings. The wire electrodes were now withdrawn and the anal plug electrode was inserted to the anal canal before the investigations were repeated. In the other subjects the investigations were carried out with the old electrode and then the new electrode without prior use of the fine wire stainless steel electrodes.
FIGURE I.5.4

External anal sphincter EMG with wire and new anal plug electrode.
Pudendoanal Reflex Latency

The PAR polysynaptic reflex arc incorporates the sensory dorsal genital nerve, the S234 spinal cord and the efferent pudendal nerve to the external anal sphincter (fig. 1.5.5). The subjects lie on their left side on the examination couch and a ground electrode is placed around the subject's right thigh. The anal plug electrode is coated with electrode gel and inserted into the anal canal. The stimulating electrode is placed on the skin in the midline at the base of the penis or clitoris and a controlled stimulus applied. The stimulus is then increased to the level of sensation before being increased to submaximal tolerable level. The Medelec MS92a stimulus triggered response unit records the time delay from stimulus to response in the external anal sphincter. The digitized response is held on the oscilloscope screen. The time to onset of the digitally averaged response to at least one hundred impulses is taken as the pudendoanal reflex latency. The sensitivity of the signal amplifier is selected to the most appropriate sensitivity for the amplitude of the incoming signal. If the amplifier sensitivity is set too high the display store will be overloaded and the signal peaks will be flattened. The amplitude of the signal response in micro volts (uV) is therefore equal to the volts per division times the screen divisions and this can be measured electronically within the apparatus. Finally a paper copy of the PAR response can be printed out with all that information included.
FIGURE I.5.5

Pudendo-anal reflex arc.
Results

Integrated EMG

There was a significant direct correlation between the new electrode and the fine wire electrode for the external anal sphincter resting EMG ($r = 0.99, p < 0.01$) (fig. I.5.6), the squeeze EMG ($r = 0.99, p < 0.001$) (fig. I.5.7) and the strain EMG ($r = 0.91, p < 0.01$) (fig. I.5.8). The direct correlation in the normal situation with reduction of EMG on straining (fig.I.5.9) persisted in the abnormal state of anismus when there was inappropriate contraction of the external anal sphincter on straining at stool (fig.I.5.10).

The EMG amplitudes recorded from the external anal sphincter with the new electrode were significantly higher than those recorded with the old electrode during rest, squeeze and strain ($p < 0.01$) (fig. I.5.11).

Pudendo anal reflex

The latency of the pudendo-anal reflex response signal when obtained from the external anal sphincter with both the old and new electrodes (45.3ms +/- 2.3ms) was identical. There was however a significantly higher response amplitude when the responses were recorded with the new electrode ($P < 0.01$) (fig. I.5.12). In keeping with this the sensitivity of the signal amplifier of the EMG apparatus could be significantly reduced when using the new electrode ($p < 0.01$) (fig. I.5.13)
FIGURE I.5.6

Correlation of external anal sphincter EMG at rest recorded with wire and new anal plug electrode.

External Anal Sphincter E.M.G. at Rest

Wire Electrode (uV)

Anal Surface Electrode (uV)

$r = 0.99$

$p < 0.01$
Correlation of external anal sphincter EMG during voluntary squeeze recorded with wire and new anal plug electrode.
FIGURE 1.5.8
Correlation of external anal sphincter EMG during straining as if at stool recorded with wire and new anal plug electrode.

External Anal Sphincter E.M.G. Strain

Wire electrode (uV)

Anal surface electrode (uV)

\[ r = 0.91 \]

\[ p < 0.01 \]
FIGURE 1.5.9 (Copy of Fig. 1.5.4)
Normal EMG at rest, squeeze and strain with fine wire and new anal plug electrodes.

ANAL SPHINCTER INTEGRATED ELECTROMYOGRAPHY

[Graph showing EMG activity with labels for rest, squeeze, and strain]
FIGURE I.5.10

EMG at rest, squeeze and strain in a subject with anismus or inappropriate contraction of the external anal sphincter during straining at stool recorded with fine wire and new anal plug electrodes.

ANAL SPHINCTER INTEGRATED ELECTROMYOGRAPHY

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FIGURE I.5.11

Comparison of mean EMG amplitude recorded during rest, squeeze and strain with old and new anal plug electrodes.
FIGURE I.5.12
Mean pudendo-anal reflex response amplitude with old and new anal plug electrodes.
EMG recorder incoming signal amplifier sensitivity with old and new anal plug electrodes.
Discussion

External anal sphincter EMG activity can be recorded using various methods such as surface EOG electrodes at the anal skin (Molander and Frenckner 1983) an anal plug surface electrode (Varma, Smith and McInnes 1986), a concentric needle electrode (Bartolo et al 1983), a single fibre needle electrode (Neill and Swash 1980) and fine stainless steel micro-wire electrodes (Womack et al 1987), depending on the particular EMG information being examined for.

The familiar hourglass anal plug electrode shape was originally made by Hopkinson by placing wet plaster of Paris in a finger piece of a rubber glove and inserting this into the anal canal (Hopkinson and Lightwood 1966). The subject then contracted the external anal sphincter maximally and allowed the plaster of Paris to set in this shape. The plug shape was then copied and made out of perspex. Two parallel circumferential electrodes 1cm apart were placed at either end of the neck of the electrode at the presumed site for stimulation of the "recto-pubalis" muscle. This electrode was designed as an alternative to Caldwell's implantable electrical stimulator for faecal incontinence (Caldwell 1963). However the circular external anal sphincter and loop of puborectalis muscle at the anorectal junction are arranged such that the longitudinal line of their muscle fibres lie in the same circle or loop shape respectively. As mentioned already muscle tissue is electrically anisotropic with the longitudinal conductivity being five to fifteen times larger than in the
transverse direction (Lindstrom and Petersen 1983). Stimulating electrodes should therefore be placed longitudinally with respect to muscle (Wynn Parry 1953). This is at variance with the design of the Hopkinson electrode. The same electrode design and shape as the Hopkinson electrode has been kept and used for more modern anal electro-stimulation devices used in the treatment of female urinary incontinence (Eriksen et al 1987). The same design is true for anal plug electrodes used for surface recording of the EMG activity of the external anal sphincter (Disa 13K78/79). Due to the increased longitudinal conductivity of muscle it is accepted that bipolar surface electrodes for recording striated muscle EMG are usually placed in the direction of the muscle fibres (Lindstrom and Petersen 1983). Each electrode is connected to either side of a balanced amplifier while a third electrode connects the patient to ground (Lenman and Ritchie 1983). The new anal plug described with its electrode plate alignment fullfills these criteria.

Electrophysiological investigation of the pelvic floor can provide detailed information on the pelvic floor muscle motor unit potential duration and single fibre density. Similarly the pudendal nerve terminal motor latency and cauda equina conduction velocity can be determined (Henry and Swash 1985). However these are specialised investigations, are variously invasive and can be uncomfortable for the subjects involved. The pudendoanal reflex latency investigation is well tolerated, is much less invasive and is a useful anorectal EMG screening test (Varma et al 1986). This
study shows that the new anal plug electrode with the critical alignment of the electrode plates significantly improves the detection of the external anal sphincter EMG signal response on eliciting the pudendoanal reflex. This electrode therefore allows for detection of weak EMG signals which would otherwise be declared absent.

When healthy skeletal muscle is completely relaxed there is usually no EMG activity (Lenman and Ritchie 1983). The pelvic floor external anal sphincter and urethral sphincter are unusual in that they have a continuous tonic EMG activity at rest (Parks et al 1962) provided that the reflex arc is intact (Bishop 1959, Gunterberg et al 1976). The detection of changes in EMG activity in the pelvic floor can be recorded with a concentric needle electrode (Bartolo et al 1983). This manner may also be used to detect anismus (Preston and Lennard-Jones 1985). However the insertion of needle electrodes into the external anal sphincter induces a degree of insertional EMG activity and may also induce inappropriate voluntary EMG activity due to anal pain or discomfort when straining as if at stool. The use of fine wire electrodes which are not felt by the patient once in place are therefore to be regarded as the "gold standard" for diagnosing inappropriate contraction of the external anal sphincter during dynamic studies while straining at stool (Womack et al 1987). It was thought that the presence of the new anal plug electrode within the anal canal might induce non-physiological responses in the external anal canal but the significant direct correlation with the fine wire electrode
at rest (p < 0.01), during squeeze (p < 0.001) and strain (p < 0.01) shows that this is not so with the new anal plug electrode. In particular where there were inappropriate increases in the EMG during straining as detected with the new anal plug electrode these were reflected in a similar EMG response detected with the fine wire electrode.

While one would not disagree with the specialised accuracy of the fine wire electrodes it has to be remembered that the wires require the use of a hypodermic needle for their insertion and the use of needles in the anal region is poorly tolerated even by the most compliant patients. The use of needle and wire electrodes would therefore appear to be directed towards the diagnostic field. On the other hand ano-rectal EMG biofeedback treatment requires repeat EMG measurements to be performed throughout the day over a period of time and any form of needle insertions would be both impractical and inappropriate. The new anal plug electrode would however be ideal in the situation of EMG Biofeedback (Section IV.1) as it is well tolerated by subjects, is easily cleansed and is made of robust material.
CHAPTER II.1

ASSESSMENT OF BOWEL FUNCTION IN CHRONIC SPINAL CORD INJURY IN MAN
Summary

A group of ten subjects with complete spinal cord transection were studied and found to have significant constipation as judged by oro-anal transit time \( (p < 0.01) \), faecal water content \( (p < 0.05) \) and frequency of defaecation \( (p < 0.01) \) when compared to a group of ten non-spinal cord injured subjects. A separate group of paraplegics each with a Brindley S234 anterior sacral nerve root stimulator implanted were also studied. They showed a similar degree of significant constipation when compared to controls but showed a significant increase in frequency of defaecation \( (p < 0.01) \) when compared to the spinal cord injury group although the faecal water content was significantly less \( (p < 0.05) \). The Brindley stimulator group also showed a more rapid oro-anal transit time than the spinal cord injury group but this did not reach statistical significance. Spinal cord injury is thus associated with significant constipation which appears to be favourably influenced in some respects by the Brindley S234 anterior nerve root stimulator.

Introduction

The motility of the gastrointestinal tract has been shown to be clearly affected by lesions of the spinal cord since the demonstration of the inhibitory action of the sympathetic system on peristalsis in animal studies in the rabbit \( (\text{Lister 1858}) \). Bayliss and Starling studied the large intestine between the ileo-caecal valve and the attachment of the levator ani in dogs and
rabbits after "destruction of the spinal cord below the tenth dorsal vertebra or by extirpation of the inferior mesenteric ganglia combined with section of the pelvic nerves" (Bayliss and Starling 1900). They concluded that the pelvic parasympathetic nerve supply was stimulatory to the colon while the sympathetic supply was purely inhibitory and that the parasympathetic supply to the distal colon was more important in achieving emptying than the intrinsic nerve activity. Other groups reported similar findings (Elliot and Barclay-Smith 1904). The clinical investigation of colonic and ano-rectal function in spinal cord injured man has seen many advances since the early studies on the automatic action of the internal anal sphincter by Gowers (Gowers 1887). There is a continuing need for objective measurements to aid clinicians when bowel function is disturbed by neurological injury. Modern colo-rectal investigations are aimed at identifying both anatomical and functional abnormalities and are used in the assessment of methods of treatment of these dysfunctions (Henry and Swash 1985). Constipation is often apparent after spinal cord injury and is usually of a chronic intractable form (Glick 1984). The aims of this study were to assess the degree of constipation in a group of spinal cord injured subjects as measured by the oro-anal transit time, faecal water content and frequency of defaecation and compare the results to those in a group of non-spinal injured control subjects. A third group of subjects each with a Brindley S234 anterior sacral nerve root stimulator implanted (Brindley 1982) were similarly studied to assess the influence of the
Brindley stimulator on the same constipation measurements.

Patients

The three groups of subjects involved in this study (table II.1.1, and II.1.2) were:

a) A group of ten healthy volunteers comprising eight males and two females with no history of gastrointestinal disease or spinal cord injury. Their mean age was 29.1 years with a range 22 - 38 years. This group acted as the control group.

b) The second group of ten subjects contained nine males and one female all with complete spinal cord injury ranging from level C4 to T10. The mean age was 34.1 years with a range from 20 - 45 years and the levels of spinal cord injury ranged from C4 to T10. The mean time since spinal cord injury was 8.1 years (range 1 - 20 years).

c) The third group consisted of seven spinal cord injured subjects with implanted sacral anterior nerve root "Brindley" stimulators. There were six males and one female in this group with a mean age of 36.3 years ranging from 20 - 50 years. The levels of spinal cord injury ranged from C5 to T3 with a mean time since injury of 7.4 years (range 1 - 21 years) and mean time since stimulator implant of 2.6 years (range 1 - 5 years).
TABLE II.1.1

Subjects involved in transit time studies, controls and spinal cord injury group without a Brindley stimulator.

Groups Involved in Transit Time Studies

<table>
<thead>
<tr>
<th>Controls:</th>
<th>Spinal Cord Injury:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>Sex</td>
</tr>
<tr>
<td>30</td>
<td>M</td>
</tr>
<tr>
<td>24</td>
<td>F</td>
</tr>
<tr>
<td>25</td>
<td>M</td>
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<td>25</td>
<td>M</td>
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<td>M</td>
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<td>34</td>
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<td>M</td>
</tr>
<tr>
<td>29</td>
<td>M</td>
</tr>
<tr>
<td>38</td>
<td>F</td>
</tr>
<tr>
<td>31</td>
<td>M</td>
</tr>
</tbody>
</table>
TABLE II.1.2
Subjects involved in transit time studies, group with Brindley stimulator.

Patients with Brindley Sacral Stimulator

<table>
<thead>
<tr>
<th>No.</th>
<th>Age</th>
<th>Sex</th>
<th>Level of Cord Injury</th>
<th>Duration of Injury (yrs.)</th>
<th>Duration of Implant (yrs.)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>37</td>
<td>M</td>
<td>T3</td>
<td>21</td>
<td>5</td>
</tr>
<tr>
<td>2</td>
<td>40</td>
<td>M</td>
<td>C7 +</td>
<td>16</td>
<td>4</td>
</tr>
<tr>
<td>3</td>
<td>50</td>
<td>M</td>
<td>C7</td>
<td>3</td>
<td>2.5</td>
</tr>
<tr>
<td>4</td>
<td>32</td>
<td>M</td>
<td>C6</td>
<td>4</td>
<td>2</td>
</tr>
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<td>C6</td>
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</tr>
<tr>
<td>7</td>
<td>28</td>
<td>F</td>
<td>T3</td>
<td>3</td>
<td>1</td>
</tr>
</tbody>
</table>

+= Incomplete lesion
* = Required an external urethral sphincterectomy
Methods

Subjects entering this study took no laxatives for at least two days prior to the study and refrained from aperients throughout the study period. They were fasted for eight hours before commencing the transit studies and after completion of the oro-caecal transit time normal diet was continued thereafter.

Oro-Caecal Transit Time

The method used was that of Bond et al as it provides a simple, safe, non-invasive means of studying small bowel transit in man, is easily repeated and does not involve ionising radiation (Bond et al 1975). Other available methods such as using radio-telemetry capsules (Bloom 1968) and isotopes (Read 1980) are less simple and less easy to repeat, particularly in compromised spinal cord injured subjects. The subjects were prepared for oro-caecal transit time estimation by an eight hour overnight fast before a baseline end expiratory 20 ml breath sample was taken prior to breakfast. Ten grams of lactulose in 15ml water was given with a standardised breakfast of cereal with milk followed by tea or coffee plus toast with preserve (500 calories). The orally administered nondigestible carbohydrate lactulose is fermented in the caecum and within eight minutes of reaching the caecum the product gas hydrogen appears in the exhaled breath (Bond 1975). The patient did not eat or drink and smoking was not permitted thereafter until the test was completed. The end expiratory breath samples were repeated every 15 minutes until the rise in breath hydrogen due to the arrival of
the lactulose in the caecum was recorded.

Oro-Anal Transit Time

The method used for this measurement was that of Hinton which involves a single ingestion of fifty 2mm cube radio-opaque pellets (Hinton 1969). This method also avoids the exposure of the subject to radiation, is non-invasive and is easily repeated. The stools were collected from the time of transit marker ingestion and were stored in a sealed container with the time and date recorded. They were then Xrayed to reveal the contained transit markers. This process was continued until at least 80% of the pellets had been recorded as having been passed. The time from ingestion to the 80% recovery point was taken as the oro-anal transit time. In the paraplegic subjects the stool collection was by manual evacuation plus the occasional spontaneous rectal evacuation. A rectal digital examination was performed each day after breakfast and any rectal residue was collected into the stool specimen containers. The subjects with lesions at or below T3 could perform self examinations while those subjects with spinal cord lesions at or above C7 required the assistance of an attendant.

Colonic Transit Time

This was derived by subtracting the oro-caecal time from the oro-anal transit time.
Faecal Water Content

The faecal water content was derived by weighing the stool specimen before and after freeze drying using the technique as described by Eastwood (Eastwood et al 1984).

Frequency of Defaecation

The frequency of defaecation was noted from the time of ingestion of the transit markers until completion of the oro-anal transit time.

Statistical Analysis

Statistical calculations were performed using the Wilcoxon rank test for paired and unpaired data where appropriate.

Results

Oro-caecal transit time

There was no significant difference between the oro-caecal times for the controls 2.95hrs +/- SEM 0.15hrs and the spinal cord injury group 3.4hrs +/- SEM 0.34hrs (p > 0.05) or between the controls and the Brindley stimulator group 3.4hrs +/- SEM 0.34hrs (p > 0.05). Similarly there was no significant difference between the spinal cord injury group and the Brindley stimulator group (p > 0.05).
Oro-Anal Transit Time

The oro-anal transit time (fig. II.1.1) in the controls was 68.7hrs +/- SEM 7.6hrs and was significantly shorter than in the spinal cord injury group which was 187.3hrs +/- SEM 27.7hrs (p < 0.01).

The control oro-anal transit time was also significantly shorter than that in the Brindley implant group 135.6hrs +/- SEM 7.5hrs (p < 0.01) (fig. II.1.1).

The oro-anal transit time in the Brindley stimulator group was shorter than in the spinal cord injury group but not significantly so (p > 0.05).
FIGURE II.1.1

Oro-anal transit time of controls, spinal cord injury and Brindley stimulator group.
Faecal Water Content

There was a significant difference between the faecal water content (fig. II.1.2) of the controls which was 69.7% +/- SEM 0.7% and the spinal cord injury group which was 63.9% +/- SEM 1.6% (p < 0.05) and also between the controls and the Brindley stimulator group which was 58.1% +/- SEM 0.9% (p < 0.01) (fig. II.1.2). The faecal water content in the spinal cord injury group was significantly higher than in the Brindley stimulator group (p < 0.05) (fig. II.1.3).
FIGURE II.1.2

Faecal water content of controls, spinal cord injury and Brindley stimulator group.
Faecal water content of spinal cord injury and Brindley stimulator group.

FIGURE II.1.3 (data from Fig. II.1.2)
Frequency of Defaecation

The frequency of defaecation (fig. II.1.4) or stools per day was significantly different between the controls 1.12/day +/- SEM 0.14/day and the spinal cord injury group 0.37/day +/- SEM 0.07/day (p < 0.01) and between the spinal cord injury group and the Brindley implant group 0.78/day +/- SEM 0.08/day (p < 0.01) (fig. II.1.5). There was however no significant difference between the frequency of defaecation in the controls and the Brindley implant group (p > 0.05).
FIGURE II.1.4
Frequency of defaecation of controls, spinal cord injury and Brindley stimulator group.
FIGURE II.1.5
Frequency of defaecation of spinal cord injury and Brindley stimulator group.
Discussion

Chronic constipation has been recognised since the time of Hippocrates in 400 BC to be one of several major problems complicating spinal cord injury (Guttmann 1973). Alterations in gastrointestinal motility after spinal cord transection have been shown by experimental studies in animals (Bayliss and Starling 1900, Elliot and Barclay-Smith 1904, Garry 1933). Studies on human subjects with low spinal cord or sacral lesions by Denny-Brown and Robertson concluded that the mechanism controlling defaecation was mediated through the sacral spinal segments and its peripheral nerves (Denny-Brown and Robertson 1935) which is in agreement with later works (Roman and Gonella 1981, Delbro et al 1984). Early colonic motility studies in spinal cord injured man showed a rapid rise in pressure during slow filling of the colon with water in subjects with a high lesion and a slow pressure rise in those with a low or cauda equina lesion (White et al 1940). These findings were however not in full agreement with the later findings of Connell who found reduced activity in the pelvic colon at rest in subjects with a high cord lesion and increased activity at rest in those with low lesions (Connell et al 1963). Reduced colonic compliance has been detected in subjects with thoracic spinal cord injury (Meshkinpour 1983) while absence of the postprandial colonic myoelectrical and motor activity in the colon was demonstrated in similar patients by Glick et al (Glick et al 1984). The absence of the postprandial response is thought to be due to lack of parasympathetic neural continuity to the left colon and rectum via
the sacral outflow in the 2nd, 3rd and 4th anterior sacral nerve roots (Glick et al 1982). In keeping with this theory, left colonic and rectal stasis has been shown to be one of the main causes of delay in gastrointestinal transit time following spinal cord injury (Menardo et al 1984). Other causes of spinal lesion such as multiple sclerosis have been associated with abnormal colonometrograms (Haldeman et al 1982, Glick et al 1982). Weber has suggested that the pons may be involved in the supra-spinal control of colorectal activity which can thus be affected by brain stem lesions (Weber et al 1985).

As the foregut parasympathetic innervation is via the vagus nerve which is not compromised in spinal cord injury it might be expected that the oro-caecal transit times in spinal cord injured subjects would not be significantly different to non-spinal injured controls. The results in this study showed no significant difference between controls and the spinal cord injury group (p > 0.05) or between controls and the Brindley stimulator group (p > 0.05). The Brindley stimulator acts on the anterior roots of the sacral nerves S234 and thus affects only the sacral parasympathetic outflow which does not directly influence the foregut. There was no difference between the oro-caecal transit times in the spinal cord injury group and the Brindley stimulator group (p > 0.05).

The presence of constipation with slow transit in spinal cord injury subjects is now accepted (Cornell et al 1963, Watson 1981, Miller et al 1981, Meshkinpour 1983, Glick et al 1984, Menardo et al 1984, Binnie et al 1988). In this study there was a significant
prolongation of oro-anal transit time between controls and both the spinal cord injury group (p < 0.01) and the Brindley stimulator group (p < 0.01). The oro-caecal times were normal thus leaving the delay in oro-anal transit time to be accounted for by prolonged colonic transit time. The colonic delay has been recorded in the left colon and rectum (Menardo et al 1984). The Brindley stimulator activates the sacral parasympathetic outflow and might be expected to influence the left colon and rectum. The mean colonic transit time in the Brindley stimulator group was 132.2 hrs +/- SEM 7.4 hrs while the mean colonic transit time in the spinal injury group was 183.9 hrs +/- SEM 27.6 hrs. This difference was however not statistically significant (p > 0.05).

There was a significant decrease in the frequency of defaecation between the spinal cord injury group and the controls (p < 0.01) but not between the Brindley stimulator group and the controls (p > 0.05). The frequency of defaecation was however significantly higher in the Brindley stimulator group when compared to the spinal cord injury group (p < 0.01) possibly reflecting an influence of the stimulator on the left colon and rectum. Recent work has shown that S3 anterior root stimulation is predominant in eliciting left colonic motor responses while the S2 and S4 responses are less well pronounced. The S4 anterior root also carries the somatic supply to the striated sphincters and its activation produces a simultaneous outlet obstruction due to pelvic floor contraction (Varma et al 1986).

The faecal water content was significantly less in both the
spinal cord injury group and the Brindley stimulator group when compared to controls (p < 0.01). This result may well reflect the overall colonic transit times of the respective groups as it is known that prolongation of exposure time to colonic mucosa results in dehydration of the stool [Binns, 1988]. However although the overall colonic transit time is shorter and frequency of defaecation is increased for the Brindley group compared to the spinal cord injury group the faecal water content is paradoxically less in the Brindley group (p < 0.05). One possible explanation for this lies in the colonic activity / motility patterns induced by the stimulator which may alter the segmental colonic transit times in the stimulator subjects. An area of physiological obstruction or retropulsion at the splenic flexure could result in increased time spent in the right colon or caecum whilst once the contents were beyond the splenic flexure stimulated motility could compensate for this delay and reduce the total colonic transit time while still passing a less well hydrated stool.
CHAPTER II.2

MOTOR EFFECTS OF S234 ANTERIOR NERVE ROOT STIMULATION ON THE FUNCTION OF THE LEFT COLON, RECTUM AND ANAL CANAL
Summary

The extent and nature of colonic and ano-rectal motility responses to S234 anterior nerve root stimulation was studied in seven paraplegic patients with a Brindley electromicturition sacral implant. After sequential S234 stimulation wave height activity was increased above basal from the transverse colon to the rectum. The mean motility index response to sequential stimulation was greatest at the splenic flexure. The greatest peak wave height and mean motility index response to individual anterior nerve root stimulation was to S3 which could empty the left colon by a distal motility gradient. S4 anterior root stimulation increased the intrarectal pressure and also raised the anal canal pressure to high levels.

Introduction

Brindley introduced the implantable anterior sacral nerve root stimulator for bladder control in paraplegic subjects (Brindley et al 1982). Using this device it has been possible to show by activating the second, third and fourth anterior sacral roots (S234) that these nerve roots carry the parasympathetic nerve supply in man to the bladder (Brindley et al 1982), sigmoid colon and rectum (Varma, Binnie, Smith et al 1986) and genital organs (Brindley 1981). The S234 anterior roots also carry the somatic motor supply to the striated muscles of the pelvic floor (Brindley 1977). The effects of the Brindley stimulator on bladder function have been studied with urodynamic observations by Cardozo et al
(Cardozo et al 1984). The effects on the striated anal sphincter and distal colorectal activity have recently been studied for the first time in man (Varma, Binnie, Smith et al 1986).

The present study investigates the extent and nature of the motility response to S234 anterior root stimulation throughout the left colon and ano-rectum in the area likely to be influenced by the parasympathetic supply in the S234 anterior sacral nerve roots.

Patients

Seven subjects, six male and one female, with traumatic spinal cord injury (Table II.2.1) had their left colonic and rectal motility studied after implantation of a Brindley sacral root stimulator. The studies described were carried out at a mean time of 2.6 years after implantation with a range of 1 to 5 years. The six male subjects had the implant to improve bladder emptying and eliminate urinary infection by minimizing residual volume. The one female subject had the implant primarily to aid urinary continence. All subjects gave informed consent for the ano-rectal investigations while inpatients in the spinal unit.
Table II.2.1

Spinal cord injury subjects with a Brindley stimulator implant

Place in study plus age, sex, level of spinal cord injury, time since injury and duration of implant for each subject.

<table>
<thead>
<tr>
<th>No.</th>
<th>Age</th>
<th>Sex</th>
<th>Level of Cord Injury</th>
<th>Duration of Injury (yrs.)</th>
<th>Duration of Implant (yrs.)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>37</td>
<td>M</td>
<td>T3 complete</td>
<td>21</td>
<td>5</td>
</tr>
<tr>
<td>2</td>
<td>39</td>
<td>M</td>
<td>C6 incomplete</td>
<td>17</td>
<td>4</td>
</tr>
<tr>
<td>3</td>
<td>50</td>
<td>M</td>
<td>C7 complete</td>
<td>3</td>
<td>2.5</td>
</tr>
<tr>
<td>4</td>
<td>30</td>
<td>M</td>
<td>C6 complete</td>
<td>4</td>
<td>2</td>
</tr>
<tr>
<td>5</td>
<td>46</td>
<td>M</td>
<td>C6 complete</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td>6</td>
<td>20</td>
<td>M</td>
<td>C5 complete</td>
<td>2</td>
<td>1.5</td>
</tr>
<tr>
<td>7</td>
<td>28</td>
<td>F</td>
<td>T3 complete</td>
<td>3</td>
<td>1</td>
</tr>
</tbody>
</table>
Stimulator

The sacral anterior root stimulators were implanted using the Brindley technique (fig. II.2.1) (Brindley et al 1982). All subjects except number 2 had three root implants S2, 3 and 4. Subject 2 had electrodes placed on roots S3 and S4 only. Subject 3 had some leakage of CSF from the site of emergence of the cable from the spinal dura but this problem resolved spontaneously and was avoided subsequently by the use of a grommet of fine woven polyester at the dural exit site (Brindley 1982).

The stimulator parameters of signal frequency, impulse burst duration, intensity and stimulation free time gaps were regulated for optimum bladder function by adjustments in the external control box (fig. II.2.2). The transmitter (fig. II.2.3) was correctly aligned on the skin over the passive subcutaneous receiver before stimulation. The stimulation electrical wave form was rectangular with a mean wave duration of 329us +/- SD 136us and the mean signal frequency was 29.8Hz +/- SD 9.2Hz. The signals were transmitted in bursts with a mean duration of 3s +/- SD 1.2s with a mean gap between bursts of 3.2s +/- SD 1.4s. The mean signal strength corresponded to 10.6V +/- SD 2.9V respectively. A standard period of total stimulation of 1 minute for each root level was used during the study of effects on the colon as this best approximated to the time scale normally used for electromicturition. The shorter trains of high frequency at high amplitude occasionally used to achieve final emptying of the bladder were not used by all subjects and therefore were not
included in this study. Similarly the continuous train of low
frequency, low amplitude impulses intended to maintain urinary
continence by contraction of the external urethral sphincter was
not used regularly by subject 7 and was therefore not studied on
this occasion.

The effects of sacral root stimulation on colonic motility were
studied without altering the stimulator parameters because altering
the bladder settings could result in autonomic dysreflexia as
clearly defined by Lindan et al (Lindan et al 1980). This occurs
more commonly when the posterior nerve roots are left intact in
Brindley stimulator patients. The autonomic effects which
characterize this dysreflexia are hypertension, sweating and
headache and are the response to visceral distension and afferent
stimulation. In this series subject 7 had the posterior roots of
S2, S3 and S4 severed at the time of implantation to avoid
spontaneous bladder contractions between periods of activation of
the stimulator as her main complaint was that of urinary
incontinence. Of the six male patients only number 3 had the S3
posterior roots cut leaving the others intact. The remaining males
did not have any posterior roots cut as adequate bladder function
was obtained using the stimulator without inducing autonomic
dysreflexia while leaving intact the option of reflex bladder
emptying and reflex penile erections.
FIGURE II.2.1

Photograph of operation scene at the implantation of a Brindley anterior sacral root stimulator. Laminectomy has been performed and the dura opened to enable the electrodes to be attached to the anterior nerve roots. The cable from the passive subcutaneous receiver enters more cranially to the left side of the photograph.
FIGURE II.2.2
Photograph of the stimulation parameter adjustment switches for the three nerve roots within the external control box of the Brindley stimulator.
FIGURE II.2.3
Photograph of the external transmitter for the Brindley stimulator attached to the signal control box which is 15 x 10 x 3 cm in size.
Methods

Manometry

Subjects were prepared for rectal and distal colonic manometric investigations by ensuring an empty proximal colon with the oral administration of one sachet of "Picolax" followed by one day of a liquid no residue diet. On the morning of the investigation any rectal residue was cleared manually and a repeat saline enema was given if required as spinal cord injury subjects have intractable constipation (Binnie et al 1988).

The motor activity was examined by three liquid filled systems placed in series each of which gave readings overlapping with the other from proximal to distal. Checks were done to ensure that manometric responses recorded at the upper end of each zone gave the same response as the distal end of the preceding one from the next recording system.

a) Transverse Colon and Splenic Flexure Studies

These proximal studies involved the use of a flexible fibreoptic sigmoidoscope with X-ray image intensifier control to introduce a fluid filled system with a single 5ml pressure balloon up to the transverse colon. The system was filled with aqueous radio-opaque fluid, to allow accurate identification and placement under radiological control. Basal (resting) activity was recorded 10cm proximal to the splenic flexure, followed by recording of pressure responses to sacral root stimulation. The balloon was now withdrawn by a measured 10cm distance to the splenic flexure, the
position checked radiographically and the recordings repeated. A third recording was taken from the mid-sigmoid region before the balloon was withdrawn to check comparability of the responses to those in b).

b) Sigmoid Colon Motility

A waterfilled system of three 5ml balloons (HSCl, Precision Drippings Ltd, U.K.) in series, separated by a fixed distance of 5cm and connected to separate external pressure transducers with a chart recorder was used. The balloons were placed in situ at 25, 20 and 15cm from the anal margin using a rigid sigmoidoscope. Colonic motility recordings were taken in the basal resting state and then in response to sacral root stimulation. The maximum pressure achieved in response to stimulation was recorded for each region. The distal balloon recording was used to check that there were comparable responses in the rectal zone, c).

c) Rectal Motility

In the rectum a water filled micro-balloon system with a fine bore tubing attachment to a strain gauge pressure transducer with a chart recorder was used (Varma and Smith 1986). The micro-balloon was placed inside a second larger balloon which in turn contained a volume of water which was equal to 25% of the maximum capacity of the rectum. The pressure in the rectum was recorded at rest and then measured during the response to stimulation of the anterior sacral roots.

d) Anal Canal Pressure

This was recorded at the point of maximum resting pressure in the
anal canal, previously determined by station pull through using a water filled microballoon system (Varma, Binnie, Smith et al 1986).

Measurements

The measurements were: Peak Amplitude measured as the maximum contraction recorded above the baseline and the Motility Index which was the product of the average motility amplitude response and the length of the response with each stimulus period. The basal motility index was derived by taking the basal mean amplitude of the spontaneous waves for a comparable period of time to the stimulated ‘response’ time. The mean Amplitude and Motility indices are expressed in response to S234 root stimulation both sequentially and individually. Brindley has suggested that simultaneous stimulation of all the S234 roots might be best for bowel emptying (Brindley, Polkey and Rushton 1988). However simultaneous stimulation of all these roots was not attempted because of the danger of autonomic dysreflexia (Lindan et al 1980) as previously described in patients with intact afferent nerve roots as was the case in this study. Since the Activity Time was largely determined by the duration of the electrical stimulation it was not a variable which allowed evaluation.

Results

The Mean Motility Response to Sequential Stimulation of S2, 3 and 4 Nerve Roots.

The peak amplitude of the waves at rest was the same throughout
the left colon. After sequential S2, 3 and 4 stimulation there was an increase in the average wave heights in all the zones. The smallest waves were in the transverse colon (fig. II.2.4).

The mean motility index after sequential S234 stimulation showed a rise above basal in all the zones (fig. II.2.5). The greatest response to stimulation in the colon was at the splenic flexure. This was significantly greater than in both the sigmoid and transverse colon. The level of rectal stimulation was however not significantly different to that of the splenic flexure.

The Motility Response to Individual S234 Root Stimulation.

S3 root stimulation produced the greatest peak wave height, for all the zones and the effect was significantly greater than that produced by S2 and S4 stimulation. The heights of the waves produced by S3 root stimulation were greater in the splenic flexure region than in the distal transverse colon but were not significantly different from those in the sigmoid and rectum (fig. II.2.6).

The mean motility index response was also greatest after stimulation of the S3 root, with the greatest motility effect at the splenic flexure (fig. II.2.7). S3 effects at the splenic flexure, sigmoid colon and rectum diminished distally. The S2 and S4 effects were similar for transverse colon, splenic flexure and sigmoid stimulation. The rectal motility response for the S4 root was however significantly greater.

The anal canal pressure rose from a mean of 68 cm H2O +/- 15 cm H2O before stimulation to 230 cm H2O +/- 40 cm H2O during stimulation.
FIGURE II.2.4.

Motility response to sequential S234 stimulation.
Wave mean peak amplitude.

* Splenic Flexure - Transverse Colon $p < 0.05$
FIGURE II.2.5.
Motility response to sequential S234 stimulation.
Mean motility index.
* Splenic Flexure - Transverse Colon  p < 0.01
# Splenic Flexure - Sigmoid Colon  p < 0.01.
FIGURE II.2.6.

Motility response to individual root stimulation for S2, 3 and 4.
Wave mean peak amplitude.
* S3 Response  Splenic Flexure - Transverse Colon p < 0.05.
# Combined S3 - Combined S2 or S4  p < 0.01.
FIGURE II.2.7.

Motility response to individual root stimulation for S2, 3 and 4.

Mean Motility Index.

* S3 Splenic Flexure - Transverse Colon p < 0.001.
# S3 Splenic Flexure - Rectum p < 0.02.
+ S4 Rectum - Sigmoid Colon p < 0.01.
Discussion

This study extends the preliminary findings of Varma and colleagues on the effects of the Brindley stimulator on the function of the distal sigmoid colon, rectum and pelvic floor (Varma, Binnie, Smith et al 1986). Quantitative data is presented on studies on paraplegic subjects throughout the left colon and encompasses sequential and individual nerve root stimulation. Following electrical stimulation of the parasympathetic nerve supply to the left colon and ano-rectum there was an increase in mean height of the motor waves in all the zones examined. The effect on the wave heights was significantly less in the transverse colon. All the other zones were stimulated to a similar wave height when the combined S2, S3 and S4 root effects were examined. The total motility response to sequential S234 stimulation showed that the splenic flexure and rectum were the maximally affected areas.

For individual root stimulation S3 produced the greatest change in amplitude in all areas in the left colon and rectum. The motility index after S3 stimulation changed to a gradient of activity down the left colon, maximal at the splenic flexure and lessening to the rectum suggesting that the activation of this root could have propulsive effects caudally.

Rectal motility was greatest with S4 root activation but this may reflect not only intrinsic contractions but with a transmitted effect from the contraction of the pelvic floor muscles, since rectal motility is normally less than that of the recto-sigmoid
when studied by similar methods after gastro-colic stimulation with food (Schang et al 1986). The pressure generated by the external anal sphincter and pelvic floor during S4 stimulation is in excess of the maximum voluntary contraction physiological range and could contribute to the high rectal pressure recorded.

It seems likely that the motility effects in the left colon could be the result of the direct electrical stimulation of the parasympathetic nerves to effector sites at smooth muscle cells in the left colon and rectum. However the possibility exists that the electrical stimulation might also be activating a pacemaker in the upper part of the left colon since this area seems to be consistently the site of greatest motility response. The distal gradient of motility established in these cases in the left colon should be effective in emptying faecal contents particularly if the contractions were peristaltic ones. Propagated waves were previously demonstrated in the recto-sigmoid in the earlier observations on the Brindley stimulator by Varma and his colleagues in 1986 (Varma, Binnie, Smith et al 1936). The high rectal pressures following S4 stimulation could delay emptying, particularly if they result from contractions of the pelvic floor (Brindley 1977). The striated contractions cease abruptly on withdrawal of the stimulus while smooth muscle contractions of the rectum tend to persist beyond it and could therefore result in evacuation. In recent notes for Surgeons and Physicians regarding the stimulator Brindley, Polkey and Rushton have suggested that anterior sacral root stimulation causes increased colonic activity.
and in most patients moves faecal material along to wards the rectum while simultaneous S234 stimulation might achieve evacuation (Brindley, Polkey and Rushton 1988).

The motility found in the transverse colon both in terms of wave height and motility index after the use of the stimulator to any one or all of all the roots regularly rose above basal levels at this site and also became higher than the basal level of the adjacent splenic flexure. If the rotor response in the transverse colon began with S2 root activity and it contracted before the major contractions of the splenic flexure were induced by S3 stimulation, filling of the left colon would begin. Further onward propulsion of the faecal stream would however be hindered by the greater S3-evoked contractions at the splenic flexure. Radio-isotope and transit marker studies indicate a delay in some normal subjects at the splenic flexure and rectum, suggesting that there may be a physiological barrier to propulsion in normal subjects at those sites (Holdstock et al 1970, Kirwan and Smith 1974). Furthermore isotope scintigraphic transit studies (Krevsky et al 1986) confirm that the transverse colon is an area where motility halts before progression of the faecal bolus into the left colon.

The overall implication from these studies seems to be that stimulation of S234 segments of the cord leads to motor activity throughout the entire area of the pelvic parasympathetic innervation from the transverse colon to the ano-rectum with the S3 level the most effective one in activating the left colon. However
It may be necessary to consider that some changes are also produced when neural stimulation is withdrawn, since the storage capacity of the left colon may be a function of inactivity between periods of stimulation.

It is unknown whether the activity in the sacral parasympathetic region is brought about by stimuli arising in all the roots together or whether it is arranged hierarchically from above downwards, S2 before S3 before S4 seriatim. Furthermore in this study the stimulus parameters chosen for bladder evacuation were accepted for stimulation of the same parasympathetic nerve roots leading to the bowel as it was feared that attempts to alter those used for bladder control would produce autonomic dysreflexia, especially since the posterior roots were intact. These observations were made in paraplegic subjects with the limitations of early experience with the Brindley device. Several of the patients in this series have subsequently been able to produce stimulus driven rectal evacuations after slight alteration in their stimulator settings. It is foreseeable that some patients may use the device as much for their bowel problem as for the bladder. Meshkinpour and colleagues (Meshkinpour et al 1983) found that the large bowel could be affected by an absence of the gastro-colic reflex elicited by a standard food stimulus after spinal cord injury with later excessive segmentation contractions which are non-propulsive (Glick et al 1984). The patients in this study maintained regular bowel evacuations by the use of alternate day suppositories and manual evacuation and therefore they had been
spared faecal retention effects.
CHAPTER II.3

THE EFFECTS OF S234 STIMULATION ON THE PELVIC FLOOR AND ANO-RECTAL FUNCTION; INCLUDING RADIOLOGICAL STUDIES ON THE ANO-RECTAL ANGLE
Summary

The effect of the Brindley stimulator on the pelvic floor and striated anal sphincter mechanism has been studied in seven paraplegic subjects. Standard manometric and electrophysiological methods were used to measure the anal sphincter pressures plus EMG activity while an X-ray proctogram technique was used to measure the anorectal angle and position of the pelvic floor. A similar group of paraplegic subjects without sacral stimulators acted as controls for this study. There was no difference in the maximum resting pressure in the anal canal between the stimulator group and controls but the fall in pressure in response to the rectosphincteric reflex as a percentage of the original resting pressure was significantly less when compared to controls. There was less descent of the pelvic floor at rest in the stimulator group yet there was no difference in the pudendoanal reflex latency, motor unit potential duration or resting EMG activity of the external anal sphincter between the two groups. The S4 root had the dominant effect on the pelvic floor with decreasing effect from the S3 and S2 roots on the pressure and integrated EMG generated by the external anal sphincter.

Introduction

Paraplegic subjects have no voluntary control over the activation or inhibition of the pelvic floor muscles and therefore lack voluntary control of urinary and faecal continence. The Brindley sacral root stimulator gives a degree of voluntary control over
bladder emptying and urinary continence by its activation of the parasympathetic supply to the bladder and the somatic efferents to the pelvic floor sphincters respectively (Brindley 1977). As the anorectal sphincters are innervated by the same S234 nerve roots as the urethral sphincter, similar activation might be expected when these roots are stimulated. Initial studies on the effect of this stimulator on the external anal sphincter confirm its innervation from these roots (Brindley 1982, Varma, Binnie et al 1986). This section compares manometric, electromyographic and Xray studies on the pelvic floor in two groups of paraplegic subjects one with and one without such a stimulator and assesses the effects of this electrical stimulation on the pelvic floor in the acute situation and after long term use.

Patients

Seven paraplegic patients with a Brindley stimulator implant were studied. The six males and one female were aged from 20 to 50 with cord lesions from C5 to T3 (Table II.3.1). All the subjects used the implant to achieve bladder emptying with minimal residual volume of urine, thus reducing recurrent urinary tract infections. Continence was aided by regular four hourly voiding using the device and as a precaution the males wore a condom sheath catheter. The control group comprised seven paraplegic subjects of similar age, level of spinal cord injury and time since injury but without a sacral stimulator implant (Table II.3.2). The control subjects agreed to participate in the study while in the spinal
injuries unit for non bowel orientated reasons, usually urological assessments. The two female subjects in this study were nulliparous. All subjects had adopted a regular bowel habit of manual rectal evacuation usually after suppositories by themselves or an attendant every two or three days, to avoid faecal impaction and spurious diarrhoea with faecal incontinence. The stimulator subjects did however feel that their frequency of defaecation had increased since using the device.
TABLE II.3.1

Patients with Brindley Sacral Stimulator

<table>
<thead>
<tr>
<th>No.</th>
<th>Age</th>
<th>Sex</th>
<th>Level of Cord Injury</th>
<th>Duration of Injury (yrs.)</th>
<th>Duration of Implant (yrs.)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 *</td>
<td>37</td>
<td>M</td>
<td>T3</td>
<td>21</td>
<td>5</td>
</tr>
<tr>
<td>2 *</td>
<td>40</td>
<td>M</td>
<td>C7 +</td>
<td>16</td>
<td>4</td>
</tr>
<tr>
<td>3</td>
<td>50</td>
<td>M</td>
<td>C7</td>
<td>3</td>
<td>2.5</td>
</tr>
<tr>
<td>4</td>
<td>32</td>
<td>M</td>
<td>C6</td>
<td>4</td>
<td>2</td>
</tr>
<tr>
<td>5</td>
<td>48</td>
<td>M</td>
<td>C6</td>
<td>3</td>
<td>1.5</td>
</tr>
<tr>
<td>6 *</td>
<td>20</td>
<td>M</td>
<td>C5</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>7</td>
<td>28</td>
<td>F</td>
<td>T3</td>
<td>3</td>
<td></td>
</tr>
</tbody>
</table>

+ = Incomplete lesion
* = Required an external urethral sphincterectomy
## Table II.3.2

### Patients Without Sacral Stimulator

<table>
<thead>
<tr>
<th>No.</th>
<th>Age</th>
<th>Sex</th>
<th>Level of Cord Injury</th>
<th>Duration of Injury (yrs.)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>36</td>
<td>M</td>
<td>T4</td>
<td>1</td>
</tr>
<tr>
<td>2</td>
<td>47</td>
<td>M</td>
<td>C5</td>
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<td>3</td>
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<td>C6</td>
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</tr>
<tr>
<td>7</td>
<td>27</td>
<td>F</td>
<td>T1</td>
<td>2</td>
</tr>
</tbody>
</table>
Stimulator

The stimulator type and method of implantation was as described by Brindley (Brindley 1982). All subjects except number 2 had three root implants S2-4. Subject 2 had electrodes placed on roots S3 and 4 only. Subject 7 had the posterior roots of S3 and S4 severed at the time of implantation to avoid any spontaneous reflex initiation of bladder emptying post operatively. The stimulator parameters of frequency, duration and intensity were regulated and set for optimum bladder or external urethral sphincter function respectively. The stimulation was given in bursts of 2 - 10 secs at a low frequency of about 10 Hz to initiate bladder emptying and bursts high frequency of up to 40 Hz used to achieve final emptying of the bladder. Continuous low amplitude S4 stimulation was intended to maintain urinary continence but this was discontinued after a short period in subject number 7. There were technical difficulties with the stimulator in this female subject and it may not have been used in the same way as in the other subjects during the study period. At this point in time the patient has now stopped using the stimulator for micturition and has returned to using a catheter pending readmission to the unit for recalibration of the stimulator settings.

Methods

The subjects were prepared by ensuring that the rectum was empty by digital examination and manual evacuation as required before
proceeding with the investigations.

Manometry

Anal Sphincter Pressure

The anal sphincter pressure and length of sphincter high pressure zone high were measured using a water filled micro-balloon system with a 1 cm station pullthrough technique. The maximum resting pressure was recorded and the distance from the anal skin margin at which this was measured was noted. The sphincter pressure was measured at this same position in the anal canal during activation of the stimulator and recorded as the maximum stimulated pressure.

Recto-sphincteric Reflex

The microballoon was placed at the site of anal canal maximum resting pressure and a second balloon of 2 x 1 cm was rapidly inflated with 50 ml of air in the rectum at 10cm from the anal margin. The reflex reduction in anal canal pressure was recorded as the rectosphincteric reflex amplitude.

Proctometrogram

The rectal capacity and intrarectal pressure at maximum volume were established using the continuous infusion proctometrogram technique (Varma and Smith 1986). This allowed calculation of the rectal compliance.
Electromyography

Pudendoanal Reflex

The pudendoanal reflex latency was recorded from the external anal sphincter with an anal plug electrode after stimulation of the dorsal genital nerve (Varma et al 1986). A minimum of 100 stimulations were given at a frequency of 1 Hz and impulse intensity 120 V. The response signal was recorded with a time base of 10 ms/cm, gain 10uV/cm and filter settings of 20Hz - 10kHz. The digitally averaged result was recorded as the pudendoanal reflex latency. The duration and amplitude of the EMG response was also measured by the recording device (Medelec MS92a, Woking, Surrey, U.K.).

Motor Unit Potential Duration

The mean motor unit potential duration of the external anal sphincter was taken as the mean of four quadrant MUPD estimations around the circumference of the external sphincter. The concentric needle EMG electrode (13149, DISA, Copenhagen) was inserted to a depth of 1.5 - 2 cm into the external sphincter and using the trigger delay facility on the Medelec MS92a single motor units were identified. At least 100 discharges were recorded from each motor unit and the digitally averaged result was taken as a motor unit potential duration. The mean of all four was taken as the MUPD representative of the whole muscle.
Integrated EMG Activity

An anal plug electrode (Chapter I.5) was placed in the anal canal and a record of the spontaneous or basal integrated EMG at rest was made with the EMG signal integrator (Electromed 4880/MX216). Thereafter the EMG activity during root stimulation was recorded.

Radiology

Anorectal Angle and Pelvic Floor Position

The proctometrogram anorectal catheter and balloon system was filled to 100 ml with a radio-opaque fluid. With the subject in the left lateral position and after correct lateral alignment using fluoroscopy an Xray proctogram film was taken at rest before a second film was taken during S4 root stimulation. The Xray films were now studied and the anorectal angle plus the pelvic floor position were calculated. The anorectal angle was at the intersection of a line drawn along the luminal axis of the anal canal and a second line drawn along the distal border of the rectum. The pelvic floor position was calculated as the distance of this angle point along a line at right angles to the pubo-coccygeal line (fig. II.3.1).
FIGURE II.3.1
Diagramatic representation of the method of calculating the ano-rectal angle and the position of this angle point in relation to the pubo-cocygeal line.
Results

Manometry

The anal sphincter high pressure zone and maximum resting pressure in the anal canal were not significantly different in the stimulator group than in the control group (Table II.3.3). The maximum anal canal pressures during stimulation of S2 (102cmH2O +/- SEM 26.3cmH2O), S3 (200cmH2O +/- SEM 14.4cmH2O), and S4 (248cmH2O +/- SEM 21.2) anterior roots (fig. II.3.2) show that the response is significantly higher with S4 root stimulation (p < 0.01). There was a significant correlation between the maximum external anal sphincter contraction pressure in response to S4 stimulation and the duration of the implant (r = 0.70, p < 0.05) (fig. II.3.3).

The rectosphincteric reflex was present in all subjects and controls. The reflex reduction in pressure of anal canal maximum resting pressure induced by the rectosphincteric reflex was not significantly different between Brindley subjects and controls. However, there was a significant difference in the amount of pressure reduction as a percentage of original anal sphincter maximum resting pressure for Brindley subjects (78% +/- SEM 1.8%) and spinal injury controls (87.3% +/- SEM 2.3%) (p < 0.05) (fig. II.3.4).

The mean maximum rectal capacity to the point of balloon expulsion and rectal compliance and were not significantly different between stimulator and control groups respectively (Table II.3.3) although one had a megarectum capacity (870ml).
### TABLE II.3.3

Results of ano-rectal manometric investigations in Brindley group and spinal cord injury controls.

#### Manometric Results

<table>
<thead>
<tr>
<th>Investigation</th>
<th>Brindley Group</th>
<th>Controls</th>
</tr>
</thead>
<tbody>
<tr>
<td>HPZ</td>
<td>2.7cm +/- 0.2cm</td>
<td>2.4cm +/- 0.2cm</td>
</tr>
<tr>
<td>MRP</td>
<td>68.3cm +/- 6.5cm H2O</td>
<td>53.6cm +/- 4.3cm H2O</td>
</tr>
<tr>
<td>MPstim</td>
<td>248.7cm +/- 21.3cm H2O</td>
<td></td>
</tr>
<tr>
<td>RSR</td>
<td>53.3cm +/- 5.6cm H2O</td>
<td>41.7cm +/- 4.6cm H2O</td>
</tr>
<tr>
<td>RSR%</td>
<td>78% +/- 1.8%</td>
<td>87.3% +/- 2.3%</td>
</tr>
<tr>
<td>MRV</td>
<td>493ml +/- 76.0ml</td>
<td>480ml +/- 21.9ml</td>
</tr>
<tr>
<td>PMRV</td>
<td>49.6cm +/- 2.5cm H2O</td>
<td>51cm +/- 1.7cm H2O</td>
</tr>
<tr>
<td>RC</td>
<td>10ml/cm +/- 1.7ml/cm H2O</td>
<td>9.1ml/cm +/- 0.5ml/cm H2O</td>
</tr>
</tbody>
</table>

HPZ = High pressure zone of the anal canal  
MRP = Maximum resting pressure in the anal canal  
MPstim = Maximum anal canal pressure during stimulation of S4 root  
RSR = Recto-sphincteric reflex inhibition of the internal anal sphincter  
RSR% = RSR fall in pressure as a percentage of the MRP  
MRV = Maximum rectal volume or capacity  
PMRV = Intrarectal pressure at MRV  
RC = Rectal compliance (during proctometrogram)
FIGURE II.3.2
Anal canal pressure at rest and during individual nerve root stimulation.
FIGURE II.3.3
Correlation of anal sphincter contraction pressure during S4 anterior root stimulation and duration of Brindley implant.

Maximum Anal Sphincter Contraction Pressure and Duration of Implant

Analy Sphincter Pressure (cm H2O)

Duration of Implant (yrs.)

\[ r = 0.7 \]

\[ p < 0.05 \]
FIGURE II.3.4

Recto-Sphincteric Reflex Pressure Reduction in Anal Canal as a Percentage of the Maximum Resting Pressure

% Reduction in Pressure

 Controls  Brindley

\[ p < 0.05 \]
Electromyography

The pudendo-anal reflex latency was within the accepted normal range of 38.5 ms +/- 5.8 ms (Bors and Blinn 1959, Rushworth 1967, Pedersen 1982, Swash 1982, Bilkey et al 1983, Smith and Varma 1985) both for Brindley subjects and controls (Table II.3.4). The pudendo-anal reflex response duration and response amplitude were not significantly different. The resting EMG was similar in both groups as was the motor unit potential duration of the external anal sphincter.

There was direct correlation between the pudendoanal reflex response amplitude and the duration of the stimulator \( r = 0.98, \ p < 0.001 \) (fig. II.3.5). There was also a direct correlation between the maximum external anal sphincter EMG in response to S4 stimulation and the duration of the stimulator implant \( r = 0.79, \ p < 0.05 \) (fig. II.3.6).
TABLE II.3.4

Results of EMG investigations in Brindley group and controls.

**Electromyography Results**

<table>
<thead>
<tr>
<th>Investigation</th>
<th>Brindley Group</th>
<th>Controls</th>
</tr>
</thead>
<tbody>
<tr>
<td>PAR</td>
<td>39.2ms +/- 2.0ms</td>
<td>37.8ms +/- 1.8ms</td>
</tr>
<tr>
<td>PARD</td>
<td>18.0ms +/- 1.4ms</td>
<td>17.1ms +/- 1.4ms</td>
</tr>
<tr>
<td>PARA</td>
<td>35.7uV +/- 3.4uV</td>
<td>30.0uV +/- 1.0uV</td>
</tr>
<tr>
<td>EMGrest</td>
<td>13.0uV +/- 1.7uV</td>
<td>12.1uV +/- 1.2uV</td>
</tr>
<tr>
<td>EMGstim</td>
<td>167.9uV +/- 13.0uV</td>
<td></td>
</tr>
<tr>
<td>MUPD</td>
<td>7.4ms +/- 0.9ms</td>
<td>7.4ms +/- 0.8ms</td>
</tr>
</tbody>
</table>

PAR = Pudendo-anal reflex latency
PARD = Pudendo-anal reflex response duration
PARA = Pudendo-anal reflex response amplitude
EMGrest = External anal sphincter EMG at rest
EMGstim = External anal sphincter EMG during stimulation of S4 root
MUPD = Mean motor unit potential duration of external anal sphincter
Correlation of pudendo-anal reflex EMG response amplitude and duration of Brindley implant.
FIGURE II.3.6
Correlation of external anal sphincter EMG amplitude during S4 root stimulation and duration of implant.

![Graph showing correlation between E.A.S. EMG (uV) and duration of implant. The correlation coefficient is r = 0.79 with p < 0.05.](image)
Radiology

The anorectal angles and pelvic floor calculations were carried out on the X-ray films as described above. An example of a pelvic floor study lateral pelvic X-ray film taken in a spinal cord injury subject without a Brindley stimulator is shown in fig. II.3.7. Films taken in a subject with Brindley a stimulator, at first inactive (fig. II.3.8), and then with the stimulator active (fig. II.3.9) are also shown.

The anorectal angles measured at rest showed no significant difference for the Brindley subjects and for control group (Table II.3.5). However during S4 stimulation there was a significant narrowing of the angle from rest (122.2° +/- SEM 1.8°) to (76° +/- 1.6°) (p < 0.01) (fig. II.3.10).

There was significantly less descent of the pelvic floor at rest in relation to the pubo-cocygeal line in the Brindley stimulator subjects (-22.8mm +/- 1mm) than in the spinal injury controls (-31.9mm +/- SEM 1.7mm) (p < 0.05) (fig. II.3.11). During stimulation of the S4 anterior root there was a significant elevation of the pelvic floor to 4mm above the pubococygeal line (p < 0.01) (fig. II.3.12).
FIGURE II.3.7
Photograph of a lateral Xray proctogram taken in the left lateral position in a spinal cord injury subject without a Brindley stimulator. Note also the hold up of transit markers at the pelvic brim in this subject. The fixed reference pubo-cocygeal line has been drawn.
FIGURE II.3.8

Photograph of a lateral X-ray proctogram taken in the left lateral position in a spinal cord injury subject with a Brindley stimulator while the stimulator was inactive. The reference pubo-coccygeal line has been drawn.
Figure II.3.9
Photograph of a lateral Xray proctogram taken in the left lateral position in the same spinal cord injury subject as in fig. II.3.8 while the Brindley stimulator was active. The reference pubo-coccygeal line has been drawn.
TABLE II.3.5

Results of radiological investigations in Brindley and control spinal cord injury subjects.

Radiology Results

<table>
<thead>
<tr>
<th>Investigation</th>
<th>Brindley Group</th>
<th>Controls</th>
</tr>
</thead>
<tbody>
<tr>
<td>ARArest</td>
<td>122.2 +/- 1.8</td>
<td>125.9 +/- 2.0</td>
</tr>
<tr>
<td>ARAstim</td>
<td>76.0 +/- 1.6</td>
<td></td>
</tr>
<tr>
<td>PFPrest</td>
<td>-27.7mm +/- 1.5mm</td>
<td>-31.9mm +/- 1.7mm</td>
</tr>
<tr>
<td>PFPstim</td>
<td>4mm +/- 1.1mm</td>
<td></td>
</tr>
</tbody>
</table>

ARArest = Ano-rectal angle at rest
ARAstim = Ano-rectal angle during stimulation of S4 root
PFPrest = Pelvic floor position at rest
PFPstim = Pelvic floor position during stimulation of S4 root.
FIGURE II.3.10

Ano-rectal angles at rest and during S4 anterior root stimulation.
FIGURE II.3.11
Position of the ano-rectal angle point (pelvic floor position) in relation to the pubo-coccygeal line at rest in spinal injury controls and Brindley stimulator subjects.
FIGURE II.3.12
Position of the ano-rectal angle point (pelvic floor position) in relation to the pubo-coccygeal line at rest and during anterior S4 root stimulation in Brindley stimulator subjects.
Discussion

The levator ani pelvic floor diaphragm stretches across the pelvic outlet and is perforated by the anorectum, vagina and urethra. The rectum lies above the pelvic floor and the anal canal lies below this level. The pelvic floor and striated external sphincter are in a state of continuous contraction to help maintain anal continence (Floyd and Walls 1953, Taverner and Smiddy 1959, Porter 1962). The puborectalis muscle is of paramount importance in maintaining faecal continence by sustaining an acute angle at the anorectal junction (Duthie 1971) where it forms a flap valve (Parks 1975). The puborectalis activity can normally be altered at will to suit the requirements of defaecation or continence as appropriate (Phillips and Edwards 1965). Innervation of all the pelvic floor muscles originates in the S234 spinal segments (Onuf 1901, Mannen et al 1982, Schroder 1980). This fact has been exploited by using the EMG activity of the easily accessed external anal sphincter as an indicator of the external urethral sphincter or puborectalis muscle activity (Bors and Blinn 1959, James et al 1979). The puborectalis muscle receives direct pelvic nerve fibres from above the pelvic diaphragm while the external anal sphincter receives fibres via the pudendal nerves from below the diaphragm (Percy et al 1981, Snooks and Swash 1986). The factors which maintain the tonic contraction of the striated sphincters are not yet fully understood but there is mediation via
a spinal reflex (Parks et al 1962) and activity may be related to muscle spindle response to stretch by descending abdominal pressure (Swash 1982). Using the cat model Bishop interrupted the S234 spinal reflex arc and demonstrated that sectioning of the posterior sacral nerve root rami abolishes spontaneous activity in the pelvic floor (Bishop 1959). Local anaesthetic block of the pudendal nerve can abolish activity in the external anal sphincter but reduces the anal canal pressure by only 15% (Duthie and Watts 1965, Frenckner and Von Euler 1975) The smooth muscle internal anal sphincter contributes up to 80% of the resting tone in the anal canal (Bennett and Duthie 1964) but is not under voluntary control. The rectosphincteric reflex relaxes the internal anal sphincter following rectal distension (Gowers 1877, Denny-Brown and Robertson 1935) with any residual anal sphincter pressure being due to the external sphincter. This reflex is present in spinal cord injured subjects (Gowers 1877) and allows rectal evacuation to proceed when intrarectal pressure is sufficient. The anal canal maximum resting pressure of the combined internal and external sphincter activity is not generally lowered in chronic paraplegics (Denny-Brown and Robertson 1935, Varma and Stephens 1972, Frenckner 1975) although it is significantly lower than non spinal injured controls when recorded in subjects with complete spinal cord lesions using perfused sleeve manometry (Wheatley et al 1977). The anal canal maximum resting pressure is also considerably reduced
in subjects with meningocele (Meunier and Mollard 1977), sacral nerve ablation (Gunterberg et al 1976), and during spinal anaesthesia (Frenckner and Ihre 1976).

The anal canal maximum resting pressure in the subjects studied here (68.3 and 53.6 cmH2O) (table x) is at the lower limit of the accepted normal for non spinal injured subjects (Read et al 1979, Matheson and Keighley 1981). The rectosphincteric reflex was present in all subjects studied here and the reflex fall in pressure response did not vary between the two groups (53.3 and 47.1 cmH2O), indicating similar internal anal sphincter activity. However when considering the fall in pressure as a percentage of the original resting pressure there was a significant difference between the fall of 78% +/- 4.9% for the stimulator group and 87.3% +/- 6.0% for the controls. This may indicate an increase in activity in the internal sphincter and/or a reduced level of resting activity in the external sphincter in the control group. The resting pressure in the external anal sphincter in the stimulator group contributed the normal residual 20% of the maximum resting pressure (Frenckner and Von Euler 1975). In contrast to this finding there was no difference reflected in the integrated resting EMG activity of the external anal sphincters between both groups. The mean motor unit potential duration and pudendoanal reflex latencies were within the accepted normal ranges for these muscles 5 - 7ms (Petersen and Franksson 1955, Bartolo et al 1983).
Prolongation of the pudendoanal reflex and motor unit potential duration are associated with neurogenic insult to the muscles of the pelvic floor giving reduced sphincter pressures and resulting in neurogenic faecal incontinence (Parks et al 1977, Beersieck et al 1979, Swash 1982). The two females studied here were not in the high risk group for neurogenic faecal incontinence which is usually associated with multiparity and middle age (Swash and Schwartz 1984).

The anorectal angle at rest showed no significant difference between the stimulator group (122.2° +/- 5°) and the controls (125.9° +/- 5.3°). However the difference in the position of the pelvic floor at rest in the two groups did achieve significance (p < 0.05) level with the stimulator group having less perineal descent (table x). Several methods are described in the literature for assessing the anorectal angle and pelvic floor descent including barium soaked gauze swabs (Parks 1975) and rubber tubing (Taggart 1966) with the patient in the lateral position. Balloon proctography with the patient sitting (Preston et al 1984) can show pelvic floor weakness. Synthetic barium impregnated stool made with potato starch is now used in dynamic defaecography studies (Mahieu et al 1984a). It is generally agreed that cine radiology with the patient in the sitting position and using this type of radio-opaque synthetic stool of similar water content to normal stool gives the most physiological conditions and allows repeatable objective
assessment of the function of the pelvic floor (Mahieu et al 1984b). The radiology can be combined with EMG and manometric studies to give a more complete picture of events (Womack et al 1985). The present pelvic floor studies were performed in the left lateral position and it is accepted that the pelvic floor is not particularly stressed in this position (Preston et al 1984). However an upright position although normally associated with an increase in pelvic floor activity in the non spinal injured subject (Taverner and Smiddy 1959) would accentuate any degree of perineal descent under the influence of gravity in the spinal injured subjects.

As described above, stimulation of the S2-34 anterior roots caused synchronous contraction of the pelvic floor, puborectalis and external anal sphincter with associated elevation of the pelvic floor, narrowing of the anorectal angle and rise in anal sphincter pressure. The S4 root had the dominant effect with decreasing effect from S2 and S3 (Brindley et al 1982, Binnie et al 1986). There was close correlation between the duration of the implant and the maximum pressure ($r = 0.81$) plus integrated EMG activity ($r = 0.93$) generated by the external anal sphincter in this study. This together with the observed similar correlation of the increase in amplitude of the pudendoanal reflex response with time of implant ($r = 0.98$) is in keeping with hypertrophy of the striated muscles of the pelvic floor during the time of the implant. The use of
electrical stimulation of intact lower motor neurons to paralysed or immobilized skeletal muscle is known to substantially reverse atrophy due to disuse and restore contractile force (Peckham et al 1976, Turk et al 1980). There is also increased resistance to fatigue with increased levels of oxidative enzymes used in aerobic metabolism and increased capillary density and a transformation of fast twitch Type II to slow-twitch Type I muscle fibres (Salmons and Hendriksson 1981, Pette and Vrbova 1985). Functional electrical stimulation (FES) is taken to mean the restoration of useful movement or sensation by the electrical stimulation of excitable tissue and many benefits have been claimed in a variety of conditions (McNeal 1977). The most well known FES is the cardiac pacemaker which was first implanted in 1957 and over a quarter of a million implanted worldwide within the next twenty-five years (Feruglio et al 1983). More directly applicable to spinal cord injury subjects are the FES of the diaphragm by phrenic nerve stimulation (Glenn and Phelps 1985), for standing and reflex walking (Vodovnik et al 1981, Kralj et al 1987), for restoration of hand function (Rebersek et al 1973, Peckham et al 1980a, 1980b, Kiwerski 1984), the Brindley sacral implant for electromicturition, continence and penile erection (Brindley 1986) and neurostimulation for the modulation of spasticity (Davis and Gesink 1974, Richardson et al 1979, Bajd et al 1985).

Muscle spasticity is difficult to quantify as it encompasses
increased muscle tone and stretch reflexes, involuntary movements and clonus. Walsh et al (1980) have used printed motors as torque generators in a technique to measure muscle tone. Using this technique of torque-induced motion analysis Douglas et al (1989) have assessed the muscle stiffness in quadriceps of paraplegics with and without electrical stimulation. They found that repeated isotonic contractions of thigh muscles induced by electrical stimulation produced an increase in muscle stiffness. Some paraplegic subjects had trained quadriceps from participating in a daily programme of FES (Petrofsky et al 1984). Their thigh muscle stiffness was not significantly different from that of untrained muscles. Stiffness of the trained muscles decreased immediately after passive movement and increased immediately after electrical stimulation in a similar way to untrained muscle. If the limb was not moved passively after stimulation then the stiffness was found to persist until the limb was moved by the physiotherapist, after which there was a quantifiable reduction in stiffness and in the EMG activity associated with the stretch reflex. The persistent stiffness was thought to be due to a plastic like moulding property of muscle, "phixotropy", at the chemical actin/myosin binding level which could maintain the muscle position or stiffness without EMG activity. This novel concept could be applied to the pelvic floor of the Brindley subjects in whom there appeared to be alteration in the position of the pelvic floor after stimulation.
without any increase in resting EMG activity. The only method of passively stretching the pelvic floor would be the downward transmission of abdominal pressure and without this the pelvic floor stiffness would persist. Other effects of such pelvic floor stiffness might be to hinder micturition immediately after electrical stimulation, in support of this hypothesis, three of the stimulator subjects have required urethral sphincterotomy to facilitate electromicturition with the stimulator.
CHAPTER II.4

THE ACTION OF CISAPRIDE ON THE CHRONIC CONSTIPATION OF PARAPLEGIA
Summary

Paraplegic subjects have intractable constipation associated with prolonged colonic transit time (Chapter 1.5). The agent cisapride significantly reduced the colonic transit time from 7.7 days to 5.1 days. Cisapride also improved the rectal tone, resulting in a significant reduction in maximal rectal capacity from 305.8 ml to 224.3 ml. There was also a reduction in residual urine volume associated with cisapride in some patients. The increased number of stools containing transit markers suggested that intraluminal mixing was increased by cisapride. Faecal water was not significantly increased. A possible complication after sudden withdrawal of the drug was retention of urine in one subject but this was subsequently avoided by its gradual reduction over two days in subjects thereafter.

Introduction

Spinal cord injury results in motor paralysis which is later complicated by bladder and bowel problems (Denny-Brown 1935, Connell 1963). The commonest bowel problem in the later phase is intractable constipation (Glick 1984). Release of acetylcholine (ACh) at the myenteric plexus by pharmacological means could be expected to stimulate colonic activity. The agent Cisapride (Janssen Pharmaceutical Ltd.) acts to stimulate gastrointestinal activity by releasing ACh at the myenteric plexus (Smout 1985). The present study investigates the effect of cisapride on colonic transit time and on rectal capacity in spinal injured subjects.
Patients

Ten subjects with complete, traumatic spinal cord injury were studied (Table II.A.1). There were 9 males and 1 female with a mean age of 34.1 years (range 20 - 45yrs). The level of cord injury varied from C4 to T10. The mean time since injury was 8.1 years (range 1 - 20yrs). All subjects gave informed consent and were investigated while in the spinal injuries unit. During the study each patient acted as his/her own control.
<table>
<thead>
<tr>
<th>Age</th>
<th>Sex</th>
<th>Level of Spinal Cord Injury</th>
<th>Time Since Cord Injury</th>
</tr>
</thead>
<tbody>
<tr>
<td>38</td>
<td>M</td>
<td>C6</td>
<td>16 years</td>
</tr>
<tr>
<td>32</td>
<td>M</td>
<td>C7</td>
<td>4 &quot;</td>
</tr>
<tr>
<td>34</td>
<td>F</td>
<td>C5</td>
<td>1 &quot;</td>
</tr>
<tr>
<td>34</td>
<td>M</td>
<td>T4</td>
<td>17 &quot;</td>
</tr>
<tr>
<td>20</td>
<td>M</td>
<td>C4</td>
<td>2 &quot;</td>
</tr>
<tr>
<td>29</td>
<td>M</td>
<td>C6</td>
<td>2 &quot;</td>
</tr>
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<td>M</td>
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<td>38</td>
<td>M</td>
<td>T10</td>
<td>3 &quot;</td>
</tr>
<tr>
<td>45</td>
<td>M</td>
<td>T10</td>
<td>15 &quot;</td>
</tr>
</tbody>
</table>
Methods

When patients entered the trial, the extent of their constipation was determined by first establishing their intestinal transit times. The ten subjects were fasted for a control period of at least 8 hours before being given an intravenous injection of 10mg cisapride. Rectal capacity and pressure studies done in the control period were now repeated. After an interval of at least 48 hours the subject was commenced on oral cisapride 10mg eight hourly. This was taken for 48 hours before the transit times were repeated. The cisapride was continued until the oro-anal transit time estimation was complete. At this time blood tests and were repeated. The post micturition residual urine volumes that were done were also done at this point.

Gastrointestinal Transit Time

The Oro-Caecal Transit Time (OCT) was estimated using the oral lactulose and expired breath hydrogen method (Bond 1975). A sample of end expiratory breath is taken as a baseline level of breath hydrogen. The subject swallows 10g of lactulose with a standard breakfast of 500 cal, having fasted for eight hours. Thereafter the breath is sampled every 15 minutes until the rise in hydrogen due to the lactulose reaching the caecum is observed. The Oro-Anal Transit Time (OAT) is estimated using radio-opaque polythene markers and Xraying of the stools (Hinton 1969). This allows a reasonable measure of transit time and avoids unnecessary exposure of the subject to xrays. The Colonic-Transit Time (CTT) was
calculated by subtracting the OCT from the OAT.

The method of stool collection was by manual rectal evacuation with the occasional spontaneous evacuation. A digital examination was performed each morning after breakfast and any rectal contents were evacuated into the collection bag. The patients were divided into two groups of five subjects. The group with spinal lesions C4 to C7 had the rectal examination and evacuation performed by an attendant while the second group with lesions T3 to T10 were able to perform the examinations and evacuations themselves.

Anal Sphincter Studies

The functional Sphincter Length (SL) of the combined internal and external anal sphincter was recorded in increments of 1cm together with the maximum resting pressure (MRP) (Varma 1984). The Recto-Anal Sphincteric Inhibitory Reflex (RSR) (Gowers 1877, Henry 1985) records the reflex induced fall in pressure due to the internal anal sphincter relaxing on rapid distension of the rectum with 50ml of air.

Ano-rectal Electrophysiological Studies

The integrity and latency of the Pudendo-Anal Reflex (PAR) (Smith 1984) involves a minor electrical stimulus over the dorsal genital nerve while recording from an anal plug electrode. Using this same anal plug electrode as a stimulating electrode, the integrity and latency of conduction in the spinal cord can be assessed by recording a Somatosensory Evoked Potential (SSEP)
with scalp electrodes (Lehmkuhl 1986).

Proctometrogram

The rectal capacity (MAXV) and rise in intraluminal pressure (MAXP) as changes in rectal volume are achieved is derived by this technique (Varma 1986). A large compliant rectal balloon is distended at a rate of 67 ml/min while the pressure in the balloon is recorded by a second device. The point of spontaneous expulsion of the rectal balloon or the detection of autonomic dysreflexia is taken as being at maximal rectal capacity. The ratio of MAXV to MAXP (RATIO V/P) gives an indication of the distensibility of the rectum.

Pulse Rate and Blood Pressure

These were recorded when intravenous cisapride was given and continued every five minutes for up to thirty minutes afterwards. The recordings were taken from the brachial pulse just proximal to the antecubital fossa with a "Dynamap" automatic pneumatic cuff recording device.

Blood Count, Urea/Electrolytes and Liver Function Tests

These blood tests were carried out in the hospital laboratories in the normal way.

Residual Urine Volume

This was carried out in the subjects who did not have an
indwelling catheter, both before and at the end of administration of oral cisapride (RUV).

Faecal Water Content

The percentage water content (\%H2O) of the faeces was derived by weighing the samples before and after a freeze drying technique (Eastwood 1984).

Transit Marker Dispersion

The number of separate stools containing transit marker pellets (STM) was recorded for each subject, giving an indication of intraluminal mixing.

Statistical Analysis

Statistical significance was determined by a Student's t test after logarithmic conversion of the data.
Results

A summary of the results is given in Table II.4.2a and 2b.

Transit Studies

The oro-caecal transit time before cisapride (3.4hrs +/- SEM 0.4hrs) was reduced but not significantly so after cisapride (2.6hrs +/- SEM 0.2hrs) (P > 0.05) (fig. II.4.1).

The oro-anal transit time before cisapride (187.3hrs +/- SEM 27.7hrs) was significantly reduced after cisapride (125hrs +/- SEM 24.3hrs) (p < 0.01) (fig. II.4.2).

The derived colonic transit before cisapride (185hrs +/- SEM 27.3hrs) was significantly reduced after cisapride (123hrs +/- SEM 24.1hrs) (p < 0.01) (fig. II.4.3).
### Results of Investigations (Mean +/- S.E.M.)

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Control</th>
<th>Cisapride</th>
</tr>
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<tbody>
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<td><strong>Transit Time</strong></td>
<td></td>
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<tr>
<td>OCT</td>
<td>3.4 +/- 0.4hr</td>
<td>2.6 +/- 0.2hr</td>
</tr>
<tr>
<td>OAT</td>
<td>187.3 +/- 27.7hr</td>
<td>125.0 +/- 24.3hr</td>
</tr>
<tr>
<td>CTT</td>
<td>185.0 +/- 27.3hr</td>
<td>123.0 +/- 24.1hr</td>
</tr>
<tr>
<td>HPZ</td>
<td>3.0 +/- 0.2cm</td>
<td>3.0 +/- 0.3cm</td>
</tr>
<tr>
<td>MRP</td>
<td>116 +/- 10.4cm H2O</td>
<td>125 +/- 9.1cm H2O</td>
</tr>
<tr>
<td>Pressure and Volume</td>
<td></td>
<td></td>
</tr>
<tr>
<td>RSR</td>
<td>65 +/- 6.2cm H2O</td>
<td>72.5 +/- 7.9cm H2O</td>
</tr>
<tr>
<td>MAXV</td>
<td>305.8 +/- 29.6ml</td>
<td>224.3 +/- 17.5ml</td>
</tr>
<tr>
<td>MAXP</td>
<td>75.5 +/- 5.9cm H2O</td>
<td>76 +/- 6.7cm H2O</td>
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<tr>
<td>Ratio V/P</td>
<td>4.3 +/- 0.4</td>
<td>3.1 +/- 0.3</td>
</tr>
<tr>
<td>RUV</td>
<td>51.5 +/- 5.3ml</td>
<td>27.7 +/- 2.6ml</td>
</tr>
</tbody>
</table>

OCT = Oro-Caecal Transit Time  
OAT = Oro-Anal Transit Time  
CTT = Colonic Transit Time  
HPZ = Anal Canal High Pressure Zone  
MRP = Anal Canal Maximum Resting Pressure  
RSR = Recto-Sphincteric Inhibitory Reflex  
MAXV = Maximum Rectal Volume  
MAXP = Pressure at MAXV  
Ratio V/P = Ratio of Maximum Rectal Volume/Pressure  
RUV = Residual Urine Volume
<table>
<thead>
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<th>TABLE II.4.2b</th>
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<tbody>
<tr>
<td><strong>Electrophysiology</strong></td>
</tr>
<tr>
<td>PAR</td>
</tr>
<tr>
<td>SSEP</td>
</tr>
<tr>
<td><strong>Faeces</strong></td>
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<tr>
<td>% H2O</td>
</tr>
<tr>
<td>STM</td>
</tr>
<tr>
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</tr>
<tr>
<td>SYST</td>
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<tr>
<td>DYST</td>
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<tr>
<td>HR</td>
</tr>
</tbody>
</table>

PAR = Pudendo-Anal Reflex  
SSEP = Somato-Sensory Evoked Potential  
% H2O = Percentage Water Content  
STM = Stools Containing Markers  
SYST = Systolic Blood Pressure  
DYST = Diastolic Blood Pressure  
HR = Heart Rate.
FIGURE II.4.1
Oro-caecal transit time before and after cisapride (oral).

Oro-Caecal Transit Time

Transit Time (hrs.)

Control

Oral Cisapride

$\rho > 0.05$
FIGURE II.4.2

Oro-anal transit time before and after cisapride (oral).

Oro-Anal Transit Time

Transit Time (hrs.)

Control    Oral Cisapride

$P < 0.01$
FIGURE II.4.3
Colonic transit time before and after cisapride (oral).
Rectal Volume and Compliance

The maximum rectal volume before cisapride (305.8ml +/- SEM 29.6ml) was significantly reduced with cisapride (224.3ml +/- SEM 17.5ml) (p < 0.01) (fig. II.4.4).

The rectal compliance or volume/pressure ratio before cisapride (4.3ml/cmH2O +/- SEM 0.4ml/cmH2O) was significantly reduced with cisapride (3.1ml/cmH2O +/- 0.3ml/cmH2O) (p < 0.05) (fig. II.4.5).

Ano-rectal Electrophysiology

Pudendo-anal Reflex

The pudendo-anal reflex was found to be present in all subjects with a mean latency of 63.8ms +/- SEM 1.5ms.

Somatosensory Cortical Evoked Potential

The SSEP was absent in all cases when the stimulus was applied via the anal plug electrode.

Cardiovascular Monitoring

There were no significant changes in heart rate, systolic or dyastolic blood pressure although there was a slight fall in dyastolic pressure with intravenous cisapride.

Page 159
FIGURE II.4.4

Maximum rectal volume of capacity before and after cisapride (I.V.).

Rectal Volume (mLs)

Control  I.V. Cisapride

p<0.01
FIGURE II.4.5
Rectal compliance (ml/cmH2O) before and after cisapride (I.V.).
Full Blood Count, Electrolytes and Liver Function Tests.

None of these investigations were adversely affected by the course of cisapride administration.

Faecal Water and Intracolonic Mixing

The faecal water was not significantly increased.

The mean number of stools containing transit marker pellets was significantly increased from 1.9 to 3.1 stools per subject (p < 0.05).

Discussion

The colon has an intrinsic myenteric nerve plexus which has a degree of spontaneous activity and causes autonomous colonic muscle activity (Christensen 1981). This myenteric plexus can also be stimulated by the extrinsic nerve supply of the colon (Bayliss 1900, Wood 1981). The constipation in spinal injury subjects may be due to interruption, at the spinal level, of the extrinsic nerve supply to the distal colon. The extrinsic parasympathetic nerve supply to the distal colon emerges from the central nervous system with the anterior sacral roots S 2, 3 and 4. Direct electrical stimulation of these sacral nerve roots has been shown to affect the pelvic colon and ano-rectal function (Varma, Binnie et al 1986). As neuroprosthetic stimulation to increase the activity of the colon is not yet as appropriate as it’s use for bladder stimulation, a suitable pharmacological preparation capable of stimulating intestinal motility would have more
immediate application. The agent cisapride is a synthetic substituted benzamide which has been shown to have a potent stimulation effect on isolated rabbit colonic smooth muscle, probably through a muscarinic receptor (Snape 1985). There is a similar effect on guinea pig ileum. Using binding studies for dopamine receptors on rat striatum and when tested against apomorphine induced vomiting in dogs, there was no anti-dopaminergic properties (Van-Nueten 1985). The effect on motor activity may be mediated in part by blocking type 2 serotonin receptors (Moriarty 1985) and the possible release of other gastrointestinal hormones (Koop 1986). The action of this agent has been studied in humans, on oesophageal motility (Wienbeck 1984, Corazziari 1985, Smout 1985). These authors have shown that this agent reduces gastro-oesophageal reflux by increasing the pressure in the region of the gastro-oesophageal junction. In gastric motility (Jian 1985, Thomforde 1985, Edwards 1987) cisapride stimulates gastric emptying. As a consequence of jejunal activity (Stacher 1986) and stimulation of colonic motility (Miller-Lissner 1985, Lee 1984, Lederer 1985) it is said to reduce transit time.

The present study shows that in spinal injured subjects cisapride significantly reduces both colonic transit time and the maximum rectal capacity. The intraluminal mixing of contents is increased, as detected by the increased dispersion of markers in stool collections. The faecal water content is little changed. The internal anal sphincter reflex relaxation caused by rectal
distension is not inhibited by cisapride. There appeared to be no adverse effects on heart rate or blood pressure although there was a slight reduction in diastolic pressure. No side effects on parameters of full blood count, urea, electrolytes and liver function tests were noted. In the first patient, who normally emptied his bladder by suprapubic tapping, the unwanted side effect of acute retention of urine was noted on the morning after abrupt cessation of oral cisapride. This resolved after one day of intermittent catheterisation. In subsequent subjects the cisapride was tailed off at 10mg twice and 10mg once per day for one day each before stopping the drug completely. A reduction in residual volume while taking cisapride was noted in seven of the remaining subjects but a more formal study would be required to determine the significance, if any, of this finding.

Regarding the long term effects of cisapride, there are to date several subjects, including two from this study, who have taken the drug for periods in excess of one year. The beneficial effects on colonic function are maintained and will be formally reported at a later date. We have not detected any deleterious long term effects of oral cisapride 10mg thrice daily on modalities of blood pressure or heart rate, full blood count, urea, electrolytes and liver function tests. We did however record one case of retention of urine after abrupt cessation of the drug and we have subsequently recorded increased frequency of micturition in three subjects with incomplete spinal lesions being treated with cisapride for colonic transit problems. This increased frequency
was troublesome in one female subject and was corrected by reducing the dosage of cisapride to 10mg twice per day.
CHAPTER II.5

DISCUSSION OF SECTION II
The gastrointestinal transit studies carried out in spinal cord injury subjects confirm the presence of a basic problem with delayed colonic transit. This results in constipation with reduced frequency of defaecation and reduced faecal water content. It may be that the colonic transit delay is predominantly related to dysfunction of the left colon (Menardo et al. 1984) as electrical stimulation of S234 anterior nerve roots and the parasympathetic nerve supply to the left colon improves the delayed colonic transit and frequency of defaecation although the faecal water content is further reduced. Colonic manometric investigations reveal that the electrical stimulation of the S234 anterior nerve roots increases the left colon pressure wave height and motility index above basal activity in all regions from the transverse colon to the rectum. The region of the left colon which was most responsive to nerve root stimulation was the splenic flexure and the greatest motility response followed the S3 anterior root stimulation which produced a gradient of activity away from the splenic flexure in both proximal and distal directions. This motility gradient could produce an area of transit delay at the splenic flexure but give more rapid distal transit beyond the splenic flexure towards the rectum. This apparent effect could be physiological in that there appears to be two such areas of hold-up at the splenic flexure and the recto-sigmoid junction in non-spinal injured subjects (Holdstock et al. 1970, Kirwan and Smith 1974, Krevesky et al. 1986). Physiological retro-pulsion from the splenic flexure should increase exposure time in the caecum and right colon.
for water absorption (Cummings 1975, Christensen 1981). The recto-sigmoid delay retains faecal material in the left colon proximal to the rectum until times of defaecation when left colonic activity delivers the stool into the rectum prior to evacuation.

The maximum rectal pressure induced by electrical stimulation of the anterior sacral nerve roots is in response to S4 root stimulation although some of the pressure recorded may reflect contraction of the pelvic floor. The pelvic floor and external anal sphincter are maximally activated by S4 anterior root stimulation causing elevation of the pelvic floor and high pressure contractions of the external anal sphincter. A similar contraction of the external urethral sphincter is used to maintain urinary continence (Brindley 1977). The pelvic floor and external anal sphincter contractions will obstruct the outflow of rectal contents during periods of stimulation but rectal evacuation might proceed after stimulation since the rectal pressure remains elevated after the striated sphincters have relaxed (Brindley, Polkey, Rushton 1988). The drug cisapride is known to act generally throughout the gastrointestinal tract in the oesophagus, stomach, small bowel and in the colon. In the paraplegic subjects studied here with normal upper gastrointestinal transit times, preserved by an undamaged vagal innervation the main effect of the drug cisapride was to reduce colonic transit time and increase frequency of defaecation but without increasing faecal water content (Binnie et al 1988).

It would therefore appear that the left colonic dysfunction associated with spinal cord injury is amenable to some correction
by manipulation of the extrinsic neural control of the left colon by localised electrical nerve stimulation of the sacral parasympathetic outflow and also by a more generalized pharmacological intrinsic neural stimulation by the prokinetic agent cisapride. By selective S4 anterior root stimulation faecal continence can be maintained by the pelvic floor and the external anal sphincter. The future prospect remains of further manipulation of the stimulation parameters to allow for possible controlled electro-defaecation as well as the already available electro-micturition in these paraplegic subjects.
SECTION III
CHAPTER III.1

A DORSAL GENITAL NERVE ELECTRICAL STIMULATOR
Summary

A portable dorsal genital nerve stimulator was designed to stimulate the external anal sphincter via the pudendo anal reflex. The stimulator was intended for use in subjects with neurogenic faecal incontinence with an intact pudendo-anal reflex arc. The device was assessed in a group of eight such women who were found to have neurogenic faecal incontinence and an intact pudendo-anal reflex arc at presentation. When the stimulator was activated there was a significant increase in the anal canal pressure (p < 0.01) accompanied by a significant rise in the EMG activity (p < 0.01) of the external anal sphincter. As a result of these effects the possibility arises of using this stimulator as an exercise over a period of time with repeated stimulations in an attempt at improving the external anal sphincter pressures and improve anal continence in these subjects.

Introduction

One of the modern EMG investigations for neurogenic faecal incontinence is the pudendo-anal reflex latency (Varma and Smith 1985). The pudendo-anal reflex arc incorporates the dorsal genital nerve, the afferent fibres to the cord, the S234 portion of the conus medullaris and the efferent nerve fibres to the pelvic floor and external anal sphincter in the pudendal nerve (fig. III.1.1). The latency from the dorsal genital nerve stimulus artifact to the external anal sphincter EMG response can be reliably used as an indicator of the degree of neuropathy. Elicitation of the reflex
results in a contraction response of the pelvic floor and external anal sphincter. A portable stimulator (fig. III.1.2) was designed to provide a train of stimuli to the dorsal genital nerve in an attempt to produce a sustained response in the external anal sphincter. The aim of this study was to assess the acute effects of the dorsal genital nerve stimulator on the anal canal pressure and external anal sphincter EMG activity.

Patients

Eight female subjects with a mean age of 47.5 years (range 32 - 65) were involved in this study (Table III.1.1). Parity ranged from 1 to 5 with a mean of 3.1. All presented with faecal incontinence and were incapacitated socially by this, having to wear incontinence pads at all times. Their initial examination in the ano-rectal laboratory confirmed the neurogenic nature of the incontinence and all subjects were found to have an intact pudendo-anal reflex although the latency was prolonged. They were informed of this study and invited to participate with each subject acting as her own control.
TABLE III.1

Patients presenting with neurogenic faecal incontinence and in whom the dorsal genital nerve stimulator was assessed.

<table>
<thead>
<tr>
<th>Age</th>
<th>Duration of Presenting Complaint</th>
<th>Parity</th>
<th>Pudendo-Anal Reflex Latency</th>
</tr>
</thead>
<tbody>
<tr>
<td>45</td>
<td>6 yrs.</td>
<td>3</td>
<td>48.7 ms</td>
</tr>
<tr>
<td>38</td>
<td>3&quot;</td>
<td>2</td>
<td>47.2</td>
</tr>
<tr>
<td>32</td>
<td>2&quot;</td>
<td>1</td>
<td>69.4</td>
</tr>
<tr>
<td>55</td>
<td>8&quot;</td>
<td>4</td>
<td>57.1</td>
</tr>
<tr>
<td>43</td>
<td>5&quot;</td>
<td>3</td>
<td>53.7</td>
</tr>
<tr>
<td>65</td>
<td>16&quot;</td>
<td>5</td>
<td>56.4</td>
</tr>
<tr>
<td>49</td>
<td>7&quot;</td>
<td>3</td>
<td>54.3</td>
</tr>
<tr>
<td>52</td>
<td>11&quot;</td>
<td>4</td>
<td>60.5</td>
</tr>
</tbody>
</table>
FIGURE III.1.1
Diagrammatic representation of the pudendo-anal reflex neural arc.

Pudendo-anal Reflex

S2,3,4

Dorsal Genital Nerve

External anal sphincter
FIGURE III.1.2

Photograph of the portable dorsal genital nerve stimulator with the felt stimulating electrodes in an insulated hand piece.
Methods

Anorectal Manometry

The anal canal high pressure zone and maximum resting pressure were measured by a station pull through technique using a water filled micro balloon system (Varma and Smith 1984). The point of maximum resting pressure was the site at which the sphincter pressure response to dorsal genital nerve stimulation was also recorded (fig. III.1.3).

Electrophysiology

The pudendoanal reflex latency (Varma, Smith and McInnes 1985) was recorded after applying over one hundred consecutive synchronised impulses to the dorsal genital nerve using the MS92a EMG multifunction apparatus (Medelec, Woking, Surrey, England). The mean motor unit potential duration (MUPD) of the external anal sphincter was calculated from the mean of twenty motor unit potential durations taken at four sites around the anal sphincter circumference (Bartolo et al 1983). These were obtained with a concentric needle electrode and using the signal trigger and delay function of the EMG recording equipment. The number of phases in each motor unit potential was counted and the fraction of polyphasic units recorded. Each part of a motor unit which lies between two crossings of the baseline including the part of the potential between the onset and the first crossing is termed a phase (Buchthal 1977). A polyphasic unit has greater than four
phases. Anal sphincter mapping was performed at the time of MUPD acquisition using a concentric needle electrode and showed that the external anal sphincter ring was intact in all cases.

An anal plug electrode was used with an EMG integrator to obtain measurements of the external anal sphincter resting EMG and later the EMG during dorsal genital nerve stimulation (fig. III.1.4).

Electrical Stimulator

A portable stimulator (fig. III.1.2) (Appendix 1) was designed to provide a train of square wave stimuli to the dorsal genital nerve with a fixed frequency of 1 Hertz and pulse duration of 0.1ms. Saline soaked felt electrodes were used to apply the skin stimulus in the midline at the base of the clitoris, identical to that used for the pudendoanal reflex test. A submaximal tolerable stimulation voltage of 120 - 150V which was between two and three times the sensory voltage was used. The portable, compact stimulator device had a rechargeable nickel cadmium battery power source. The duration of stimulation for this study was for ten seconds although the reflex response did not attenuate with up to five minutes of continuous stimulation at 1Hz.

Statistics

Statistical analysis of the data was by the Wilcoxon signed rank sum test for paired data.
MAXIMUM ANAL CANAL PRESSURE AT REST AND DURING STIMULATION OF THE DORSAL GENITAL NERVE.
INTEGRATED EMG ACTIVITY OF THE ANAL SPHINCTER AT REST AND DURING STIMULATION OF THE DORSAL GENITAL NERVE

STIMULATION OF THE DORSAL GENITAL NERVE

AT REST  BEGIN  STOP  AT REST
Results

Ano-rectal Manometry

Subjects showed a short high pressure zone in the anal canal (1.9cm +/- SEM 0.2cm) with a low maximum resting pressure (49.1cmH2O +/- SEM 11.4cmH2O). There was however a significant rise in anal canal pressure to 89cmH2O +/- SEM 10.4cmH2O on activation of the dorsal genital nerve stimulator (p < 0.01) (fig. III.1.5).

Ano-Rectal Electrophysiology

The mean pudendo-anal reflex latency (55.9ms +/- SEM 2.4ms) was prolonged compared to the accepted normal range (38.5 +/- SEM 1.2ms) (Varma and Smith 1984). The external anal sphincter MUPD (12.7ms +/- SEM 1.7ms) was also prolonged compared to the normal range for this investigation (6.3ms +/- SEM 0.3ms) (Bartolo et al 1983). There was a high mean number (25.9% +/- SEM 3.3%) of polyphasic motor unit potentials recorded from these subjects compared to the accepted 12% for normals (Buchthal 1977).

The resting integrated EMG (11.6uV +/- SEM 1.7uV) of the external anal sphincter was significantly increased (44.9uV +/- SEM 4.9uV) during activation of the stimulator (p < 0.01) (fig. III.1.6).
FIGURE III.1.5
Maximum pressure recorded in the anal canal at rest and during dorsal genital nerve electrical stimulation.
FIGURE III.1.6
Maximum amplitude of integrated EMG recorded from the external anal sphincter at rest and during dorsal genital nerve stimulation.
Discussion

The pelvic floor and external anal sphincter are normally in a state of tonic contraction at rest (Floyd and Walls 1953, Taverner and Smiddy 1959). The tonic EMG activity is dependent on a spinal reflex arc (Parks et al 1962) and although the pubo-rectalis and external anal muscles contain several muscle spindles (Winckler 1958, Swash 1982) the exact mechanism which maintains or perpetuates the tone is not yet clear. The integrity of the entire arc can be tested by the pudendo-anal reflex as described earlier (Varma and Smith 1985). Interruption of the S234 afferent nerve root fibres abolishes spontaneous activity in the pelvic floor and external sphincter (Bishop 1959). A similar cessation of spontaneous activity in the external anal sphincter follows local anaesthetic block of the S234 efferent pudendal nerve fibres (Frenckner and Von Euler 1975). Traction injury to the pudendal and pelvic nerves is associated with excessive straining and perineal descent such as occurs during a protracted second stage of labour (Parks et al 1977, Snooks, Barnes and Swash 1984, Snooks, Barnes et al 1984, Snooks, Barnes et al 1985, Jones et al 1987). In neurogenic faecal incontinence the terminal portion of the pelvic and pudendal nerves are the common site of injury (Kiff and Swash 1984) and might therefore selectively denervate the puborectalis, external anal sphincter and external urethral sphincter leading to faecal and urinary incontinence respectively.

Over the last century various electrical devices have been designed with the aim of investigating the innervation or
functional contractions of the external anal sphincter and the pelvic floor.

Galvanic and Faradic electrical stimulation tests were used to differentiate between normal innervated muscle and denervated muscle by surface stimulation tests. The denervated muscle would contract to direct current (galvanism) shocks lasting for about one second but was unlikely to respond to the short impulses produced by induction coil at the limits tolerated by the patient. The innervated muscle is excited through its nerve supply and therefore responds to brief faradic shocks and is also responsive to galvanic shock. As it was not practicable to define intensity and duration of impulses the test was of qualitative nature only. Moreover Ritchie (1954) showed that if the induction coil had a high enough output then the test had only 50% chance of being correct. The test similarly had no prognostic value for follow-up examinations as there was no quantitative measure of the outputs produced. This may explain why the empirical use of faradic stimulation for pelvic floor descent and stress incontinence of urine has had such variable success, particularly if it was being used in subjects with markedly denervated pelvic floor muscles. Caldwell described the use of faradic stimulation of the anal sphincter as a degrading and unpleasant procedure and without much effect (Caldwell 1963).

Caldwell and Fleck designed an implantable device with electrodes which were directly inserted into the external anal sphincter and which were activated by a radiofrequency current from a secondary coil over the iliac region and stimulated the external anal
sphincter (Caldwell and Fleck 1963). Caldwell measured rises in anal pressure with a Burge tube balloon and showed that pressures of 15mmHg were easily achieved but that pressures of 30mmHg led to pain. The device signal/response was calibrated on a normal patient’s external anal sphincter after insertion of the electrodes through the skin during an operation for pilonidal sinus.

Aiming to avoid implanted materials Hopkinson and Lightwood made an hour glass shaped anal plug electrode which activated the external anal sphincter muscle via two circumferential electrode plates, one at the level of the puborectalis and the other at the anal margin (Hopkinson and Lightwood 1966). There was however a lack of objective ano-rectal manometry regarding the effects of the Hopkinson plug as the anal sphincter pressures were assessed by contraction onto the examiners digit in the anal canal. This considerably limits the value of any comparative study. However the basic shape of the Hopkinson anal plug electrode has stood the test of time and is to his credit (Section IV.1). Hopkinson assessed the likelihood of success with the anal plug stimulator by the patients ability to retain the anal plug stimulator in the anal canal while standing upright. This may have acted as a very crude indicator of the degree of denervation of the muscle with the badly denervated sphincters being unable to retain the electrode. The Hopkinson device most likely worked by stimulating the muscle via the intramuscular portion of its intact peripheral nerve supply (Allin 1986) as this requires about one tenth the strength of electrical stimulus required to stimulate muscle tissue directly. More success
might therefore have been expected had there been better patient selection with exclusion of those patients with complete denervation of the external anal sphincter in whom the device would most likely not work. Some initial success was reported with both the Caldwell and Hopkinson stimulators but neither have not found continued use in this field (Duthie 1971) due to wire breakages in the implant requiring removal of the entire device and a combination of problems with the anal plug such as anal discomfort, occasional ulceration of the anal canal and the need to retain the device in the anal canal at all times to be effective.

More recently Brindley introduced an implantable anterior sacral root stimulator for electromicturition and contraction of the striated external urethral and anal sphincters by anterior S234 sacral nerve roots in selected paraplegics (Brindley 1982). Apart from bladder effects this device also has profound effects on the pelvic floor and external anal sphincter (Section II.2) (Varma, Binnie et al 1986). Implantation of this stimulator requires a major surgical procedure and is generally unsuitable for use if afferent spinal conduction is intact due to the presence of some afferent autonomic and somatic fibres in the anterior nerve roots. The Brindley stimulator would thus be inappropriate in its present form for use in non paraplegic subjects for either Bladder stimulation or pelvic floor and anal sphincters stimulation.

The dorsal genital nerve stimulator was designed to produce stimuli similar to those used in the test situation as a clinical observation showed that in subjects with an intact reflex arc,
eliciting the pudendo-anal reflex produced contractions of the external sphincter and pelvic floor although the subjects with neurogenic faecal incontinence were often unable to feel this contraction or reproduce the degree of contraction voluntarily. The reflex was also noted to show no signs of habituation over several minutes stimulation as in agreement with other authors (Pedersen 1982). This study has shown that the stimulator is able to produce a significant rise in the anal canal pressure associated with a significant rise in the external anal sphincter EMG activity. The possibility of using the stimulator to try and improve the anal canal pressures over a longer period of time in these subjects now arises (Chapter III.2).
CHAPTER III.2
DORSAL GENITAL NERVE PUDENDOANAL REFLEX ELECTRICAL STIMULATION FOR
THE TREATMENT OF NEUROGENIC Fecal INCONTINENCE
Summary

A group of eight women were shown to have neurogenic faecal incontinence. Ano-rectal manometric investigations revealed a low maximum resting pressure in the anal canal together with a poor reflex contraction pressure response to cough and also a low maximum voluntary squeeze contraction of the external anal sphincter. Ano-rectal electrophysiological investigations revealed that the external anal sphincter ring of muscle tissue was intact on EMG mapping. There was low integrated EMG activity at rest in all subjects and all had an intact but prolonged pudendoanal reflex arc. A portable dorsal genital nerve stimulator (Chapter III.1) was used to provide repetitive train stimuli which gave a regular series of contractions of the external anal sphincter. After an eight week course of electrical stimulation, ano-rectal continence was regained in all but one case who continued to have incontinence of flatus only. There was a significant increase in the maximum resting pressure in the anal canal \( p < 0.01 \) together with cough \( p < 0.01 \) and squeeze pressure responses \( p < 0.01 \) in all cases. The improved continence persists at one year after completing the electrical stimulation therapy in all but the one case who has since undergone a surgical postanal repair.

Introduction

With modern manometric and electrophysiological techniques neurogenic faecal incontinence is an increasingly recognised problem. Formerly known as idiopathic faecal incontinence the
causation is now known to be due to trauma to the pelvic and pudendal nerves and is frequently associated with prolonged or difficult labour (Parks 1975). The recognizable signs include reduced function at rest in the pelvic floor and external anal sphincter as well as impairments in the reflex and voluntary control of continence. Previously various electrical stimulators have been designed with the aim of returning function to the external anal sphincter and pelvic floor using a surface anal plug electrode with two concentric ring electrodes (Hopkinson and Lightwood 1966) or with implanted stimulators activated by a radio frequency link outside the body and subcutaneous leads to the electrodes in or near the striated anal sphincters (Caldwell 1963). Although some have been successful initially (Duthie 1971), these stimulators have for various reasons not found continued use in this field for example due to wire breakages in the implants and anal pain or mucosal ulceration with the anal plugs. One of the modern EMG investigations for neurogenic faecal incontinence is the pudendoanal reflex latency (Smith and Varma 1985). The latency from the stimulus artifact to the sphincter response can be used as an indicator of the degree of neuropathy in the standard neurogenic faecal incontinence cases (allowing for the exclusion of other causes of faecal incontinence). Elicitation of the reflex results in a contraction response of the pelvic floor and external anal sphincter. A portable stimulator (Appendix 1) (fig. III.1.2) was designed to provide a train of stimuli to the dorsal genital nerve and was used three times per day for eight weeks. The aim of this
study was to harness repetitive stimulation of the pudendoanal reflex to try and restore continence and observe any alterations in appropriate anorectal manometric and electrophysiological measurements.

Patients

Eight female subjects (Table III.2.1) with a mean age of 47.5 years (range 32 - 65) were involved in this study. Parity ranged from 1 to 5 with a mean of 3.1 +/- SEM 0.4. All presented with faecal incontinence with a mean duration of 7.25yrs +/- SEM 4.5yrs and were incapacitated socially having to wear incontinence pads of one type or another. As a result of the initial examination it was felt that all subjects would benefit from a surgical post anal repair procedure and were therefore offered this operation. While awaiting surgery they were informed of this study and invited to participate with the understanding that they would remain on the waiting list should surgery still be required after the trial period. After the stimulator had been validated the subjects were recalled to the laboratory to commence this study. During the study each subject acted as her own control. All subjects had an intact pudendo-anal reflex arc with a mean latency of 55.9ms +/- SEM 2.4ms.
### TABLE III.2.1

Patients Presenting with Neurogenic Faecal Incontinence and Treated with the Dorsal Genital Nerve Stimulator.

<table>
<thead>
<tr>
<th>Age</th>
<th>Duration of Presenting Complaint</th>
<th>Parity</th>
<th>Pudendo-Anal Reflex Latency</th>
<th>Self Assessment of Degree of Incontinence: Pre-Treatment : Post-Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>45</td>
<td>6 yrs.</td>
<td>3</td>
<td>48.7 ms</td>
<td>complete *</td>
</tr>
<tr>
<td>38</td>
<td>3 &quot;</td>
<td>2</td>
<td>47.2</td>
<td>“</td>
</tr>
<tr>
<td>32</td>
<td>2 &quot;</td>
<td>1</td>
<td>69.4</td>
<td>“</td>
</tr>
<tr>
<td>56</td>
<td>8 &quot;</td>
<td>4</td>
<td>57.1</td>
<td>“</td>
</tr>
<tr>
<td>43</td>
<td>5 &quot;</td>
<td>3</td>
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<td>3</td>
<td>54.3</td>
<td>“</td>
</tr>
<tr>
<td>52</td>
<td>11 &quot;</td>
<td>4</td>
<td>60.5</td>
<td>“</td>
</tr>
</tbody>
</table>

*complete = solids, liquids and flatus.
Methods

All measurements were performed at the initial presentation and repeated after completing the eight week course of electrical stimulation.

Manometry

A standard water filled microballoon system with external transducer was used to measure the anal canal pressure with a lcm station pullthrough technique (Varma and Smith 1984). The anal sphincter high pressure zone or functional anal sphincter length was measured and the maximum resting pressure recorded. The reflex contraction pressure of the external sphincter during a cough was recorded at the point of maximum resting pressure as was the maximum voluntary squeeze pressure of the sphincter. The rectosphincteric reflex fall in pressure on distending the rectal ampulla rapidly with a balloon with 50ml of air was also recorded at the point of maximum resting pressure in the anal canal (Smith and Varma 1986). The external anal sphincter pressure response during dorsal genital nerve stimulation was also recorded at this point.

Electrophysiology

An anal plug electrode was used with an EMG integrator to obtain measurements of the external anal sphincter resting EMG and later the EMG during dorsal nerve stimulation. The pudendoanal reflex latency (Varma and Smith 1985) and response amplitude were recorded.
after applying over one hundred consecutive synchronised impulses to the dorsal genital nerve using an EMG multifunction apparatus (Medelec MS92a, Woking, Surrey, England). The mean motor unit potential duration of the external anal sphincter was calculated from the mean of twenty motor unit potential durations taken at four sites around the anal sphincter circumference (Bartolo et al 1983). These were obtained with a concentric needle electrode and using the signal trigger and delay function of the recording equipment. The number of phases in each motor unit potential were counted and the fraction or overall percentage of polyphasic units was calculated for each subject. Each part of a motor unit which lies between two crossings of the baseline including the part of the potential between the onset and the first crossing is termed a phase (Buchthal 1977). A polyphasic unit has greater than four phases. Anal sphincter mapping was performed at the time of MUPD acquisition using a concentric needle electrode and showed that the external anal sphincter ring was intact in all cases.

Electrical Stimulator

The portable stimulator provided a train of square wave stimuli to the dorsal genital nerve with a fixed frequency of 1 Hertz and pulse duration of 0.1ms. Saline soaked felt electrodes were used to apply the skin stimulus in the midline at the base of the clitoris, identical to that used for the pudendoanal reflex test. A submaximal tolerable stimulation voltage of 135V +/- SD 15V which was two to three times the sensory voltage was used. As stated in
the previous chapter the immediate effect of activating the stimulator was to cause a significant rise in the external anal sphincter EMG activity (fig. III.1.6) from 11.6μV +/- SEM 1.7μV to 44.9μV +/- SEM 4.9μV (p < 0.01) with a corresponding significant rise in the anal canal pressure (fig. III.1.5) from 49.1cmH2O +/- SEM 4.0cmH2O to 89cmH2O +/- SEM 10.4cmH2O (p < 0.01). The portable, compact stimulator device had a rechargeable power source which could last for about ten days when in use. The duration of treatment was for eight weeks, using the device for five minutes on three occasions per day. The subjects were trained in the use of the stimulator in the laboratory before being given the device to take home for domiciliary use. At the end of the course of stimulation the external anal sphincter EMG response and pressure response to activation of the stimulator were retested.

Statistics

The method of statistical analysis for the results of measurements of the high pressure zone (HPZ), maximum resting pressure (MRP), maximum voluntary squeeze contraction pressure (MVC) and cough reflex contraction pressure (CRP) is that of a Student's t test after logarithmic conversion of the data of the paired observations. This is mathematically valid because the effects of the treatment are putatively proportional rather than additive. In this situation the logarithm stabilises the variance as the coefficient of variation is constant. Transforming back, the mean numbers are antilogged and these are now called the geometric
The remaining data were analysed by the Wilcoxon signed rank sum test for paired data.

Results

Manometry

The anal canal high pressure zone (HPZ) representing the functional anal sphincter length was increased from 1.9cm +/- SEM 0.2cm to 2.6cm +/- SEM 0.3cm after the period of stimulation but not significantly so (p > 0.05).

The anal canal pressure reduction on reflex relaxation of the internal sphincter, recto-sphincteric reflex (RSR) was not significantly altered by the course of dorsal nerve stimulation from 43.8cmH2O +/- SEM 3.5cmH2O to 45.4cmH2O +/- SEM 3.2cmH2O (p > 0.05).

The maximum resting pressure (MRP) in the anal canal (fig. III.2.1) was significantly increased from 49.1cmH2O +/- SEM 4.0cmH2O to 61.2cmH2O +/- SEM 4.5cmH2O (p < 0.01) after the course of electrical stimulation.

The pressure generated by reflex contraction of the external anal sphincter in response to coughing (CRP) (fig. III.2.2) was significantly increased from 80.6cmH2O +/- SEM 8.2cmH2O to 106.1cmH2O +/- SEM 11.2cmH2O after the period of stimulation (p < 0.01).

The maximum voluntary squeeze contraction (MVP) of the external
anal sphincter (fig. III.2.3) was also increased significantly from 112cmH2O +/- SEM 12.1cmH2O to 150.9cmH2O +/- SEM 17.3cmH2O after the period of stimulation (p < 0.01).

There was also a significant increase in the external anal sphincter pressure response to dorsal genital nerve stimulation at the end of the course of treatment (128cmH2O +/- SEM 13.3cmH2O) (fig. II.2.) from the stimulation pressure at the beginning of the therapy 89cmH2O +/- SEM 10.4cmH2O (p < 0.01).
FIGURE III.2.1

Maximum resting pressure recorded in the anal canal before and after the course of dorsal genital nerve stimulation.
FIGURE III.2.2
Maximum pressure recorded in the anal canal during the reflex response to a cough before and after the course of dorsal genital nerve stimulation.
FIGURE III.2.3

Maximum pressure recorded in the anal canal during a maximal voluntary squeeze contraction of the sphincter before and after the course of dorsal genital nerve stimulation.
FIGURE III.2.4
The maximum pressure recorded in the anal canal during dorsal genital nerve stimulation before and after the course of electrical stimulation.

Anal Canal Maximum Pressure
With Stimulator

M.P. (cm H2O)

Before  After Treatment

+ p<0.01
Electrophysiology

The pudendoanal reflex latencies (PARL) with a mean of 55.9 ms +/- SEM 2.4 ms were prolonged when compared to this laboratory normal range of 38.5 ms +/- SEM 0.94 ms (Varma and Smith 1985) and were indicative of neurogenic injury. The motor unit potential durations (MUPD) of the external anal sphincter with a mean of 12.7 ms +/- SEM 1.2 ms were prolonged or widened when compared to the accepted normal range 6.9 ms +/- SEM 0.2 ms. There was also a high mean number of polyphasic motor unit potentials recorded from the external anal sphincters in these subjects with a mean of 25.9% +/- SD 9.4% and the accepted norm being 12%. Both of which were in keeping with reinnervation of the muscle after a previous neurogenic insult (Bartolo et al 1983). The MUPDs were not affected by the course of electrical stimulation with a mean of 12.7 ms +/- SEM 1.2 ms before and 12.8 ms +/- SEM 1.2 ms after (p > 0.05).

The pudendoanal reflex response amplitude (PARAmp) (fig. III.2.5) was significantly increased from 37.5 uV +/- SEM 3.7 uV to 49.5 uV +/- SEM 3.3 uV (p < 0.01) after the period of stimulation.

The resting or basal integrated EMG (EMGR) of the external anal sphincter (fig. III.2.6) was increased significantly from 11.6 uV +/- SEM 1.7 uV to 26.9 uV +/- SEM 2.6 uV after the course of stimulation (p < 0.01).

There was a significant increase in the EMG response of the external anal sphincter during dorsal genital nerve stimulation at the end of the period of treatment 72.3 uV +/- SEM 5.4 uV (fig. III.2.7) from that at the beginning 44.9 uV +/- SEM 4.9 uV (p < 0.01).
FIGURE III.2.5
The pudendo-anal reflex response amplitude before and after the course of dorsal genital nerve electrical stimulation.
FIGURE III.2.6
The external anal sphincter EMG at rest before and after the course of dorsal genital nerve electrical stimulation.
FIGURE III.2.7

The maximum integrated EMG recorded from the external anal sphincter during dorsal genital nerve stimulation before and after the course of dorsal genital nerve electrical stimulation.

![Diagram showing EMG recordings before and after treatment](image-url)
Discussion

The pelvic floor and external anal sphincter are in a state of continuous tonic contraction at rest. The tonic EMG activity is dependent on a spinal reflex arc although the exact mechanism which maintains or perpetuates the tone is not yet clear. The integrity of the entire arc can be tested by the pudendoanal reflex. The pudendoanal reflex arc includes the afferent dorsal genital nerve, the conus medullaris and the efferent fibres from the S2,3,4 roots to the pelvic floor and external sphincter. Elicitation of the pudendoanal reflex produces a measurable contraction in these muscles and the latency from stimulation to response can be used as an indicator of neurogenic damage. Traction injury to the pudendal and pelvic nerves is associated with dysfunction of the pelvic floor and external anal sphincter which can result in faecal incontinence. Injury to these nerves occurs during excessive straining with perineal descent such as occurs during labour. With partial denervation the remaining nerve fibres attempt to reinnervate the muscle fibres by sprouting. The process of reinnervation by any one nerve fibre is limited and beyond this there will be no further sprouting by the nerve to the muscle fibres which will therefore remain denervated. Reinnervation of the pelvic floor can take up to two years to be complete depending on the site and degree of nerve injury. Percutaneous stimulation of the cauda equina and transrectal pudendal nerve terminal motor latencies can help to locate the site of efferent nerve injury. In neurogenic faecal incontinence the terminal portion of the pudendal
nerve is the common site of injury. The majority of cases of partial pudendal nerve neuropathy after delivery will spontaneously recover sufficiently to correct the faecal or urinary incontinence. However particularly difficult and/or multiple deliveries can result in cumulative damage which may be beyond the powers of spontaneous recovery precipitating faecal incontinence from that time onwards. Similarly if sufficient nerve fibres are lost at the time of insult then the natural loss of further nerve fibres with the passage of time may result in a critical insufficiency of neuromuscular units to maintain continence at some time after the insult and thus present with neurogenic faecal incontinence in middle age or later life. The subjects in this study all had reduced spontaneous activity in the anal sphincter at rest. This was reflected in a low spontaneous integrated EMG activity. There was also a weak reflex response to coughing and a reduced voluntary squeeze contraction. This manometric picture coupled with the EMG evidence of prolonged PAR latency, prolonged mean MUPD and increased polyphasic potentials confirmed the neurogenic nature of the external anal sphincter dysfunction. With a normal rectosphincteric reflex to relax the internal anal sphincter there was little pressure left to prevent faecal incontinence at rest and a reduced voluntary and stress reflex safety margin.

Various treatments have been advocated for neurogenic faecal incontinence and to some extent they reflect the spectrum of the condition from occasional soiling to frank faecal incontinence.
Alterations in diet and medications may be required to give sufficient predictability of bowel habit to avoid incontinence in some subjects with little or no control over the striated anal sphincters (Munn 1972, Habeeb and Kallstrom 1976). Pelvic floor exercises with physiotherapy are commonly used for urinary stress incontinence (Castleden 1984, Klarsov 1984, Wilson 1984) and could equally well be applied in mild degrees of anal incontinence. Biofeedback has found a place in the treatment of faecal incontinence (Almy 1979, Cerulli 1979) by addressing the problem from two angles i) the problem of reduced anorectal sensation or awareness and ii) the poor response of the voluntary sphincters. Anorectal awareness is improved by operant conditioning of the rectosphincteric response using pneumatic training devices (Engel 1974, Olness 1980, Constantides 1983) while the voluntary sphincter response can be improved with similar training devices (Cerulli 1979, Wald 1981). Biofeedback has also proved effective in correcting inappropriate contraction of the pelvic floor during straining at stool (Bleijenberg 1974). The surgical procedure of postanal repair was devised by Parks to reconstitute the anorectal angle and improve resting tone plus squeeze pressures in the anal canal with 72% fully continent and 12% continent for solid stool only (Browning and Parks 1983). Keighley reported similar figures for full continence of 63% and a further 21% continent for solid stool only (Keighley 1984). The operation of gracilis muscle transposition (Pickrell et al 1952) to reconstruct the external anal sphincter apparatus may be considered in some cases with
additional loss of external sphincter muscle due to trauma or disuse (Corman 1980, 1984).

The use of electrical stimulation as an adjunct to diagnosis in cases on denervated muscle has been practised for over one hundred years (Erb 1883). Later the Galvanic and Faradic stimulation tests were used to differentiate between normal innervated muscle and denervated muscle. The denervated muscle would contract to direct current (galvanism) shocks lasting for about one second but was unlikely to respond to the short impulses produced by induction coil at the limits tolerated by the patient. The innervated muscle is excited through its nerve supply and therefore responds to brief faradic shocks and is responsive to galvanic shock also. As it was not practicable to define intensity and duration of impulses the test was of qualitative nature only. Moreover Ritchie (1954) showed that if the induction coil had a high enough output then the test had only 50% chance of being correct. The test similarly had no prognostic value for follow-up examinations as there was no quantitative measure of the outputs produced. This may explain why the empirical use of faradic stimulation for pelvic floor descent and stress incontinence of urine has had such variable success, particularly if it was being used in subjects with markedly denervated pelvic floor muscles. Caldwell described the use of faradic stimulation of the anal sphincter as a degrading and unpleasant procedure and without much effect (Caldwell 1963). Together with Flack he then designed an implantable device for anal sphincter stimulation. A subcutaneous coil at the iliac spine was
activated by a radiofrequency current from a second coil taped to the overlying skin. The cables were led subcutaneously to the electrodes inserted in the external sphincter muscle. Rises in anal pressure were measured with by a Burge tube balloon. Success was reported with return of continence and after 4 months further stimulation was not required as sufficient anal tone could be maintained without the device (Caldwell 1963). Although problems with displacement of one electrode were recorded with the first case the more serious technical failure of cable breakage requiring removal of the entire device was reported by Alexander and Rowan. They describe only 5 out of 29 implants still working after four years (Alexander and Rowan 1970). The problem of cable breakage due to movement has been overcome in modern neuroprosthetic devices such as the Brindley sacral root stimulator (Brindley 1982) by the use of the helically coiled "Cooper wire" (Donaldson 1983). Hopkinson was encouraged by Caldwell's initial success but was committed to minimal implanting of apparatus and so proceeded to design an anal plug electrode for direct stimulation of the external anal sphincter and puborectalis (Hopkinson 1972). This hourglass plug electrode was placed in the anal canal and remained there while the sphincter was stimulated. Monophasic current caused blackening of the anode and occasional ulceration of the anal canal. These problems were avoided by the use of biphasic current. Treatment was given for two hours per day until symptoms resolved or until the subject gave up with lack of progress. Unfortunately the anal sphincter pressures were only
assessed digitally by contraction onto the examiners digit in the anal canal. The test which correlated best with the prognosis of treatment was the ability to retain the anal plug while walking. In retrospect this may have acted as a very crude indicator of the degree of denervation of the muscle, although some of the subjects had rectal prolapse which itself could have caused further weakening of the anal sphincters due to dilatation. The subjects who were able to retain the electrode were accepted for treatment but had to live with the electrode permanently in place during treatment to guarantee continence. This could be unacceptable even to the most highly motivated subject.

The present method of stimulating the pelvic floor and external anal sphincter via the pudendoanal reflex using the dorsal genital nerve stimulator was provoked by clinical observations on the pudendoanal reflex. It was noted in subjects with an intact reflex arc that elicitation of the pudendoanal reflex produced contractions of the external sphincter and pelvic floor although the subjects with neurogenic faecal incontinence were often unable to feel this contraction or reproduce the degree of contraction voluntarily. The reflex was also noted to show no signs of habituation (Pedersen 1982) over several minutes stimulation. The stimulator was therefore designed to produce stimuli similar to those used in the test situation. This study has shown that repeated use of the stimulator on a thrice daily basis over the course of several weeks serves to return function to the pelvic floor and external anal sphincter in that there is significant
improvement in the maximum resting pressure, cough and squeeze pressures of the anal canal. The maintenance of this striated sphincter activity is returned to the subject by a combination of sphincter awareness and the ability to contract the pelvic floor voluntarily or by reflex during coughing. This is reflected in the improved resting EMG and maximum resting pressure in the anal canal. This together with the normal muscle tone is what presumably helps to maintain spontaneous activity in the normal situation and is analogous to the effect of biofeedback in similar circumstances. The mode of action of this return of pelvic floor function appears to be mainly by hypertrophy of the innervated muscle fibres and subsequent awareness of their contraction. There was widening of the MUPD in some cases but this was not significant indicating that the mechanism is unlikely to be due to further reinnervation of the sphincters. Loss of tone in the pelvic floor could perpetuate the lack of spontaneous activity in the spinal reflex arc as afferent input to the sacral cord is essential for this activity to persist (Bishop 1959). Simple tightening of the puborectalis portion of the pelvic floor by a post anal repair would not persist unless the muscle could to some extent actively maintain this position afterwards. EMG studies show increased resting activity after postanal repair in keeping with reactivation of the pudendoanal reflex arc. In summary, all subjects in this study were shown to have neurogenic faecal incontinence due to pudendal nerve neuropathy with a prolonged but intact pudendo-anal reflex. After eight weeks of treatment seven out of eight subjects
became fully continent of faeces and flatus while one remained incontinent of flatus only. There was no change in the pudendo-anal reflex latency or the MUPD in the external anal sphincter. The maximal resting anal canal pressure was increased above the continence threshold and the protective cough and squeeze pressures were significantly improved in the same seven out of eight subjects.

In conclusion, with neurogenic faecal incontinence the pudendo anal reflex may be present albeit prolonged. This reflex does not fatigue on electrical stimulation and may be harnessed by repetitive stimulation to improve the external anal sphincter resting pressure and also cough and squeeze pressures. This is associated with restoration of control of normal faecal continence which persists in some cases for over one year. The investigations described allow judgements to be made on the external anal sphincter but since the afferent dorsal genital nerve stimulates the S234 region of the conus medullaris there may also be effects on the pubo-rectalis and pelvic floor in general as they are supplied by the same nerve roots. Investigation of the effects of the dorsal genital nerve stimulator on the pelvic floor would be best studied in a dynamic radiological fashion with simultaneous EMG recordings with the subject in the sitting position (Womack et al 1985). Such a study is now underway in this laboratory.
SECTION IV
CHAPTER IV.1

BIOFEEDBACK MEASURES TO INFLUENCE OUTLET OBSTRUCTION CONSTIPATION

IN MAN
Summary

Fifteen subjects presenting with intractable constipation associated with prolonged straining at stool were shown to have anismus, inappropriate contraction of the pelvic floor and external anal sphincter when straining at stool. The internal anal sphincter recto-sphincteric inhibitory reflex was found to be normal in all cases thus excluding Hirschprung’s disease. The mean duration of the presenting complaint was 8.8 years +/- SEM 1.8yrs. The functional abnormality of the external anal sphincter was revealed with standard EMG apparatus and an electromyographically derived index was devised to grade the degree of anismus. A self applied EMG biofeedback device was then used to allow observation of the external anal sphincter EMG by the subjects. This enabled relearning of higher control to suppress the non-relaxing pelvic floor. Biofeedback training was continued for a mean time of 3.1 weeks and resulted in a significant reduction in the degree of anismus as graded by the anismus index from 69.9 +/- 7.8 before biofeedback to 14.0 +/- 3.1 after (p < 0.01). There was an associated reduction in time spent straining at stool and in the degree of difficulty encountered in passing stool. The correction of the external anal sphincter EMG response to straining at stool and the clinical benefit to the patients has persisted at a mean follow up time of 6.2 months.

Introduction

The external anal sphincter is in tonic continuous activity
maintained by spinal reflexes and further controlled by supraspinal centres which increase tone to maintain continence and inhibit tone to allow defaecation. An area in the pons has control over distal colonic and ano-rectal motility (Weber et al 1985). Higher control of the external anal sphincter and achievement of socially acceptable continence is a learned phenomenon which varies from about the age of 2 years (Fritz and Armbrust, 1982) up to age 6 years (Whiting and Child 1953) depending on the different cultures. The EMG activity of the external anal sphincter has been studied by surface electrodes in children of different ages (Molander and Frenckner 1983) and has shown that supraspinal control is not achieved until about the age of 2.5 years. Complete spinal cord injury results in loss of supraspinal control of the pelvic floor and external anal sphincter and is often associated with dyssynergy of the external urethral sphincter (Jurascheck et al 1980, Wyndaele 1987). Urethral sphincter dyssynergy may require a sphincterotomy to allow micturition to proceed beyond the non-relaxation or more correctly the inappropriate contraction of the external urethral sphincter (Klein et al 1969, Wyndaele et al 1985, Wyndaele et al 1987). In non-spinal injured subjects anismus is the inappropriate contraction of the pelvic floor musculature when straining to defaecate. This failure of inhibition of the pelvic floor causes a functional outlet obstruction of the ano-rectum resulting in constipation (Wasserman 1964, Preston and Lennard-Jones 1985). The affected subjects strain excessively at stool and higher centres are unaware of the incoordination of the
pelvic floor. Anismus or inappropriate external anal sphincter contraction can be detected by modern non-invasive EMG techniques (Chapter 1.5). The claim has been made that with appropriate biofeedback training subjects can re-learn suppression of the non relaxing pelvic floor (Bleijenberg 1987). A self applied EMG device, Myotron 120, (Physiological Feedback Systems, Vijftig Bunderweg 1, Dorst (NB), The Netherlands) allows the subject to see or hear the external anal sphincter muscle EMG response during straining and it is this feedback of biological information which is the basis of the training technique (Almy 1979). The aim of this study was to assess the effect of using such an EMG biofeedback device in the treatment of a group of subjects with anismus.

Patients

The fifteen subjects (Table IV.1.1) included twelve females and three males with a mean age of 45 years (age range 22 - 76 years). The universal presenting complaint was that of constipation with excessive straining at stool and difficulty in evacuating the rectum. The mean duration of the presenting complaint was 8.8 years +/- 7.1 years. All subjects had some degree of perineal discomfort at defaecation and one subject had apparent anterior rectal mucosal prolapse at presentation. All subjects underwent proctoscopy and rigid sigmoidoscopy and showed no signs of solitary rectal ulceration or other anorectal pathology.
### Patients Presenting With Anismus And Treated With E.M.G. Biofeedback

<table>
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<th>No.</th>
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</tr>
<tr>
<td>2</td>
<td>F</td>
<td>46</td>
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<td>3</td>
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<td>1 year</td>
</tr>
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<td>F</td>
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<td>20 years</td>
</tr>
<tr>
<td>5</td>
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<td>6</td>
<td>M</td>
<td>61</td>
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<td>7</td>
<td>F</td>
<td>42</td>
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</tr>
<tr>
<td>8</td>
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<td>F</td>
<td>50</td>
<td>7 years</td>
</tr>
</tbody>
</table>
Methods

Ano-Rectal Manometry

Standard manometric methods using a water filled microballoon system were used to measure the length of the anal sphincter high pressure zone, the maximum resting pressure in the anal sphincter and the rectosphincteric reflex relaxation of the internal sphincter (Varma and Smith 1984). A continuous infusion proctometrogram technique was used to assess the threshold volume for sensory awareness, the maximum rectal capacity and rectal compliance (Varma and Smith 1986). In a repeat test the balloon was filled to the level of sensory awareness and the patient invited to attempt to expel the balloon voluntarily. The ability of the subject to expel the balloon or not was recorded. This was known as the Balloon Expulsion Test (BET).

Ano-Rectal Electrophysiology

The pudendoanal reflex latency was recorded with an anal plug electrode after dorsal genital nerve stimulation (Varma, Smith and McInnes 1986). The external anal sphincter motor unit potential duration was measured using a standard concentric needle electrode (Bartolo et al 1983).
FIGURE IV.1.1
External anal sphincter integrated EMG in normal response to straining and with anismus.
Detection of Anismus

The subject lay in the left lateral position with a ground electrode wrapped around the right thigh. An anal plug electrode (Chapter 1.5) connected to an isolated EMG integrator was placed in the anal canal. The resting or basal EMG from the external anal sphincter was recorded and the subject was then asked to contract the external sphincter and the maximum squeeze EMG was recorded. The subject was then asked to strain as if at stool and the strain EMG was recorded (fig. IV.1.1). Using these results the anismus index was calculated using the method previously described from this laboratory (Kawimbe, Binnie and Smith 1988).

Anismus Index =

\[
\frac{(\text{Strain EMG} - \text{Rest EMG})}{(\text{Squeeze EMG} - \text{Rest EMG})} \times 100.
\]

EMG Biofeedback

A short course of training in self treatment was planned for the subjects about to enter the study and a structured plan was made for the duration of that period of biofeedback was (fig. IV.1.2). The subjects were given a two or three day period of training with the device before being allowed to take the Myotron 120 home for domiciliary use (fig. IV.1.3). During training the subjects came to the anorectal laboratory and met the female technician involved in this study with the doctor concerned. The subjects were shown a diagrammatic representation of their problem and the objectives of the biofeedback treatment were explained. The anal plug electrode was placed in the anal canal and connected to
the biofeedback device, the Myotron 120 (fig. IV.1.4). The Myotron was calibrated so that the resting EMG was reading as zero. The subject now contracted the external sphincter and noted the squeeze EMG which was displayed in uV in excess of the original resting EMG. The subject then strained as if at stool and took particular note of the external anal sphincter EMG response again displayed as uV in relation to the resting EMG. The EMG activity could also be monitored continuously by means of a built in loudspeaker, the frequency of the sound varied proportionally with the EMG level. The facility of having the sound feedback given when the EMG was above or below the set threshold was also available. At successive attempts and by altering the method of straining as required the subject endeavoured to reduce the straining EMG down to or below the resting EMG and thus correct the anismus dysfunction. Subjects were then allowed to take the device home to be trained with for two, five minute sessions per day. They were to attempt defaecation only on the early morning occasion, the second session being seen as a reinforcement exercise only. The Myotron had attached to it a simple set of instructions for the procedures involved lest some patients should forget or became confused by the sequence of events. The duration of domiciliary biofeedback treatment was in multiples of two weeks depending on the patients symptoms and whether they felt they would benefit from continuing biofeedback therapy. On the whole they were highly motivated patients who were distressed by their symptoms yet all went through the learning exercise and period of training. They
appreciated the availability of the female technician in the team for help at all times. Eight used the device for two weeks, six used it for four weeks and one used it for six weeks. The subjects also kept a diary record of the number of stools passed per week, the time spent straining at stool, the degree of difficulty felt in passing stool and finally any perineal pain or discomfort (fig. IV.1.5). The latter two indices were scored on an analogue scale and then categorised into 0, 1 = mild, 2 = moderate, 3 = severe groups. The subjects kept the diary for at least two weeks before commencing biofeedback and continued throughout the course of treatment. Prior to review the subjects again recorded these factors for two weeks prior to attending the laboratory.

Follow-up

All subjects were reviewed between 3 - 6 months after the assessment at the end of the course of biofeedback when the tests were repeated. Two were seen again at just under 1 year and a further two have been reviewed at over 15 months giving an overall mean follow-up time of 6.2 months +/- 4.6 months.

Statistics

Statistical analyses were performed on the data using the non-parametric test of the Wilcoxon signed rank test for paired data.
FIGURE IV.1.2
Algorithm for the biofeedback study.

Biofeedback Treatment of Anismus

Methodology

- Diagnosis
- Pre-treatment Diary
- Hospital Treatment
- Home Treatment
- During-Treatment Diary
- End of treatment evaluation
FIGURE IV.1.3

Plan for training period with Myotron 120 biofeedback device.

Biofeedback Treatment of Anismus

Hospital Treatment

Day I
- Repeat EMG
- Explain technique and equipment
- Proctometrogram balloon
  Expulsion exercise

Day II
- Explain
- Balloon expulsion exercise
- Patient uses biofeedback equipment on the ward

Day III
- PT demonstrates ability to use equipment
- ± balloon expulsion exercise
- Discharge home
FIGURE IV.1.4
Photograph of the Myotron 120 EMG biofeedback device. The control box measures 15 x 10 x 3cm while the electrode has the same dimensions as the electrode in chapter I.5 (page 43).
FIGURE IV.1.5

Items recorded in patient diary.

Biofeedback Treatment of Anismus

Diary

- Number of bowel motions
- Time spent in the loo
- Time spent on the loo straining
- Defaecation pain
- Degree of defaecation difficult
Results

Ano-rectal Manometry

The results of the standard ano-rectal manometric investigation are shown in Table IV.1.2. These investigations were carried out at the first visit to the laboratory and repeated at the end of the biofeedback training and also at the time of later follow-up review. There were no significant differences between any of the results on the pre- or post-biofeedback tests or between the pre-biofeedback and follow-up tests at the laboratory i.e. p > 0.05. Of note, there was no direct correlation between the duration of symptoms, and presumably the condition of anismus, and maximum rectal capacity (r = 0.12, p > 0.1).
## TABLE IV.1.2

Ano-rectal Manometry and EMG results (Mean +/-SEM).

<table>
<thead>
<tr>
<th>Test</th>
<th>Biofeedback</th>
<th>Biofeedback</th>
<th>Follow-up</th>
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</thead>
<tbody>
<tr>
<td>HPZ</td>
<td>3.1cm +/- 0.2cm</td>
<td>3.0 +/- 0.2</td>
<td>3.1 +/- 0.2</td>
</tr>
<tr>
<td>MRP</td>
<td>92.3cmH2O +/- 5.1cmH2O</td>
<td>91.4 +/- 5.6</td>
<td>92.9 +/- 5.5</td>
</tr>
<tr>
<td>RSR</td>
<td>66.4cmH2O +/- 4.6cmH2O</td>
<td>66.2 +/- 4.5</td>
<td>66.1 +/- 3.8</td>
</tr>
<tr>
<td>MRV</td>
<td>448ml +/- 21ml</td>
<td>443 +/- 22</td>
<td>445 +/- 23</td>
</tr>
<tr>
<td>RC</td>
<td>7.6ml/cmH2O +/- 0.4ml/cmH2O</td>
<td>7.6 +/- 0.5</td>
<td>7.7 +/- 0.5</td>
</tr>
<tr>
<td>PAR</td>
<td>42.3ms +/- 1.7ms</td>
<td>42.4 +/- 1.7</td>
<td>42.5 +/- 1.8</td>
</tr>
<tr>
<td>MUPD</td>
<td>8.8ms +/- 0.6ms</td>
<td>8.7 +/- 0.7</td>
<td>8.9 +/- 0.8</td>
</tr>
</tbody>
</table>

HPZ = High pressure zone in anal canal  
MRP = Maximum resting pressure in the anal canal  
RSR = Recto-sphincteric reflex  
MRV = Maximum rectal volume of capacity  
RC = Rectal compliance ratio of volume/intraluminal pressure  
PAR = Pudendo-anal reflex latency  
MUPD = Motor unit potential duration of the external anal sphincter
Ano-rectal Electrophysiology

The pudendo-anal reflex was found to be present in all cases with a mean latency of 42.3ms +/- 6.7ms (Table IV.1.2).

The mean motor unit potential duration of the external anal sphincters was 8.8 +/- 2.5ms with the more widened motor unit potentials in the subjects with the longer history of anismus. There was a significant correlation between the duration of presenting complaint and the motor unit potential duration of the external anal sphincter (r = 0.82, p < 0.01) (fig. IV.1.6).  

Anismus and Biofeedback

A summary of the anismus index before and after EMG biofeedback and at the latest follow up are given in Table VI.1.3. Also tabulated are the results of recorded stool frequency, perineal discomfort at time of defaecation, difficulty with defaecation, time spent in the toilet and time spent straining at stool. On analysing the results after biofeedback and at follow-up compared to the pre-biofeedback items the anismus index (fig. IV.1.7) was significantly decreased at (p < 0.01) as was the degree of pain at defaecation (fig. IV.1.8), the degree of difficulty at defaecation (fig. IV.1.9), the time spent straining at stool (fig. IV.1.10) and the overall time spent in the toilet (fig. IV.1.11). The frequency of defaecation was increased less significantly after biofeedback (p < 0.05) and did not persist at follow-up. Prior to biofeedback only 2 subjects could expel the rectal balloon while 13 out of the 15 could do so after biofeedback training.
FIGURE IV.1.6

Correlation of external anal sphincter mean motor unit potential duration and duration of symptoms of anismus.
TABLE IV.1.3

Results of anismus index plus records from patient diary before and after biofeedback and at later follow-up (Mean +/- SEM).

<table>
<thead>
<tr>
<th>Test</th>
<th>Pre-Biofeedback</th>
<th>Post-Biofeedback</th>
<th>Follow-up</th>
</tr>
</thead>
<tbody>
<tr>
<td>AI</td>
<td>69.9 +/- 7.8 %</td>
<td>14.0 +/- 3.9</td>
<td>14.6 +/- 3.7</td>
</tr>
<tr>
<td>PPAD</td>
<td>2.3 +/- 0.2</td>
<td>0.5 +/- 0.2</td>
<td>0.4 +/- 0.2</td>
</tr>
<tr>
<td>DAD</td>
<td>2.8 +/- 0.1</td>
<td>1.1 +/- 0.2</td>
<td>0.6 +/- 0.1</td>
</tr>
<tr>
<td>TSAS</td>
<td>12.7 +/- 1.2 min</td>
<td>5.6 +/- 0.8</td>
<td>4.5 +/- 0.5</td>
</tr>
<tr>
<td>BM/wk</td>
<td>5.2 +/- 0.8</td>
<td>8.8 +/- 1.0</td>
<td>7.4 +/- 0.7</td>
</tr>
<tr>
<td>BET</td>
<td>2</td>
<td>13</td>
<td>11</td>
</tr>
</tbody>
</table>

AI = Anismus index (%)

BM/wk = Bowel motions per week

PPAD = Perineal pain at defaecation

DAD = Difficulty at defaecation

TSAS = Time spent straining at stool (minutes)

BET = Ability to expel rectal balloon or balloon expulsion test
Anismus Index Pre-, and Post Biofeedback

Anismus Index

Pre- Biofeedback  Post- Biofeedback  Follow-up

$ p < 0.01 $
FIGURE IV.1.8

Degree of Pain at Defaecation

Pain at Defaecation

Pre-Biofeedback  Post-Biofeedback  Follow-up

p < 0.01
FIGURE IV.1.9

Difficulty at Defaecation

Difficulty at Defaecation

Pre-Biofeedback  Post-Biofeedback  Follow-up

+ p < 0.01
FIGURE IV.1.10

Time Spent Straining at Stool

Time (mins.)

Pre-Biofeedback  Post-Biofeedback  Follow-up

+ p<0.01

Page 238
Time Spent in the Toilet

- **Pre-Biofeedback**
- **Post-Biofeedback**
- **Follow-up**

Note: $p < 0.01$
Discussion

Faecal continence is maintained at rest by the tonic contraction of the puborectalis muscle together with the activity of the internal and external anal sphincters (Taverner and Smiddy 1959, Parks et al 1966). The internal anal sphincter relaxes on distension of the rectum by an involuntary myenteric reflex independent of the spinal cord (Denny-Brown and Robertson 1935). The external anal sphincter remains contracted until much larger volumes distend the rectum (Parks et al 1962). The puborectalis muscle and the external anal sphincter can be relaxed under voluntary control (Whitehead et al 1981) to allow defaecation to proceed when appropriate after raising intra-abdominal pressure and adopting the correct posture (Phillips and Edwards 1965). Anorectal outlet obstruction can result from the absence of the rectosphincteric reflex relaxation of the internal anal sphincter in rectal aganglionosis of Hirschprung's disease (Faverdin et al 1981). Outlet obstruction is also present if the normal inhibition of the pelvic floor does not occur during straining at stool (Rutter 1974). The failure of relaxation of the pelvic floor and external anal sphincter is compounded by inappropriate contraction of the pelvic floor during straining at stool (Womack et al 1987). The descriptive term "anismus" has been applied to this condition (Preston and Lennard-Jones 1985).

Anismus should be suspected if the history given by the patient includes excessive straining at stool sometimes to the point of exhaustion. The patient may volunteer the need for self
digitation to accomplish rectal evacuation (Martin et al 1981). Objectively the condition can be diagnosed by anorectal manometry showing a rise in sphincter pressure on straining (Preston and Lennard-Jones 1985); EMG studies showing increased activity on straining (Rutter 1974); radiological investigations showing contraction of the pelvic floor (Kuijpers et al 1986) and defaecography showing accentuation of the puborectalis indentation on straining (Mahieu et al 1984); or a combination of these with pressure and EMG superimposed on a videoproctogram study (Womack et al 1987). Anismus was diagnosed in all subjects in this study using a surface anal plug electrode and the EMG technique described above.

The immediate effect of inappropriate contraction of the pelvic floor and external anal sphincter when straining to defaecate is to cause anorectal outlet obstruction (Preston and Lennard-Jones 1985). The consequent constipation leads to the ineffectual consumption of laxatives (Turnbull et al 1986). As the rectum becomes more chronically impacted there is diminution in the sensory awareness (Meunier et al 1979, Loening-Baucke and Younoszai 1982) and a flaccid megarectum may ensue (Nixon 1967, Oppe 1967, Lane and Todd 1977). With continued rectal fullness the internal and external sphincters are inhibited at rest and this leads to faecal soiling or encopresis particularly so in children (Levine 1971). There is a strong association between anismus and anorectal abnormalities caused by excess straining (Kuijper et al 1986). One of the long term effects of anismus with prolonged straining at
stool is that the anterior anorectal mucosa is forced against the puborectalis bar and may become oedematous and tend to prolapse (Lane 1974, Rutter and Riddell 1975). This mucosa is traumatised and may bleed or become ulcerated as a solitary rectal ulcer (Kuijpers et al 1986). As many of the anismus subjects self digitate to evacuate the rectum it was thought that anterior solitary rectal ulcers were due to digital trauma. This is not the case as the solitary rectal ulcer can occur in cases who do not digitate (Martin et al 1981). All subjects in this study underwent proctoscopy and rigid sigmoidoscopy but no evidence of solitary rectal ulceration was seen. Prolonged excessive straining against a closed pelvic floor sphincter mechanism leads to perineal descent. Traction on the pelvic and pudendal nerves causes neurogenic damage to the striated sphincters (Parks et al 1977, Neill et al 1981, Snooks et al 1984). It is known that peripheral nerves are damaged if stretched beyond 12% of their resting length (Sunderland 1978). Reinnervation occurs in the muscles supplied by the traction damaged nerve (Henry et al 1982) and an indication of the degree of reinnervation can be related to the prolongation of the mean motor unit potential duration of the affected muscle (Bartolo et al 1983). There was a significant correlation between the duration of the presenting complaint of excessive straining at stool in the subjects in this study and the mean motor unit potential duration of the external sphincter. This indicates that there is continuing damage to the pudendal nerves the longer the condition persists and is consistent with the findings of Snooks et
al (1985) and Bartolo et al (1983). Pudendal nerve neuropathy may also result in perineal descent with alteration in the relative positions of perineal anatomy and so partially obstruct defaecation (Bartolo et al 1985). Subjects with this problem find that bilateral peri-anal digital support of the perineum or posterior vaginal digital support eases defaecation. If the neurogenic insult is sufficient then frank faecal incontinence will ensue (Parks et al 1977). It therefore appears that there is a spectrum of conditions from simple anismus, solitary rectal ulcer syndrome, perineal descent, rectal prolapse and frank neurogenic faecal incontinence which have many separate causes but which could be due in some cases to an initial problem of anismus which has gone unnoticed.

The cause of anismus is not known but probably a combination of several factors act by different routes to culminate in the end result of anismus. Anxiety, mental stress and other psychological disorders may be associated with the onset of anismus as they are found in many patients attending with other functional gastrointestinal disorders (Switz 1976, Thomson and Heaton 1980). Various spinal abnormalities congenital and acquired (Wyndaele 1987, Binnie et al 1988) may be associated with anismus and may be coupled with similar dysfunction of the external urethral sphincter (Wyndaele 1987). Specific antagonist muscles contribute to the "let go" of the flexor digitorum muscles in the forearm on relaxation (Matthews 1988) while the lack of specific antagonists to the pelvic floor may be a predisposing factor to the occurrence of
spasm in the pelvic floor muscles.

The treatment of anismus will depend to some extent on the age of the subject and the degree or stage of the condition. Correct toilet training in infancy is of prime importance (Pinkerton 1958). The deliberate retention of stool by some children, although not anismus, may indicate psychological or psychiatric abnormalities which should be treated before directing attention to the bowel problems. Anismus can be recognised in children (Meunier 1985) but conditions such as Hirschprung's disease and other congenital anorectal anatomical abnormalities should be excluded (Lawson and Nixon 1967). The coexistence of urological problems with outlet obstruction should be considered (Bannister 1988) and checks made for lumbosacral dysraphism such as spina bifida occulta (Yip et al 1985, Fidas et al 1989) as the possibility of surgical release of the tethered cord may arise (James et al 1979).

Pharmacological blockade of the sympathetic innervation to the bladder neck can be achieved by the use of alpha-1-adrenoreceptor antagonists in spinal cord injury subjects troubled by vesico-urethral dyssynergy (Awad et al 1977, Gosling et al 1977, Scott and Morrow 1978, Cramer 1989). However these effects are directed at the sympathetic tone of the posterior urethra smooth muscle sphincter (McGuire et al 1977) and although they might have effects on the sympathetic nervous influence on the internal anal sphincter (Carlstedt et al 1988) they would not be so applicable to the somatic innervation of the external anal sphincter in anismus. Puborectalis relaxation by the local injection of Botulinum A
toxin has been shown to be effective in correcting anismus (Hallan et al 1988). The effect however is relatively short lived and requires repeated local injections to the posterior portion of the muscle under EMG guided control to maintain sufficient relaxation.

The anismus problem of the non-relaxing puborectalis muscle has been tackled by various surgical methods. Division of the puborectalis muscle has been advocated to allow unobstructed defaecation to proceed (Wasserman 1964, Wallace and Madden 1969, Barnes et al 1985). Against this approach to an essentially functional problem is that the puborectalis is of prime importance in maintaining continence at rest by a flap valve (Parks 1966) in these anismus subjects as much as it is in normal non-anismus patients. Dividing the muscle will lead to loss of the anorectal angle and could therefore result in faecal incontinence in a similar way to other causes of traumatic faecal incontinence (Christiansen and Pedersen 1987). Surgery may be necessary in some cases where anismus is associated with a megacolon and slow transit constipation (Yoshioka and Kieghley 1989, Bleijenberg and Kuijpers 1987). Colectomy with ileorectal anastomosis has been advocated in these cases with acceptable functional results despite a continuing need to strain (Preston et al 1984). The semi-liquid ileal effluent is easier to pass per-rectum than dry inspissated stool. Rectal prolapse can be corrected by various forms of rectopexy including ivalon songe rectopexy (Penfold and Hawley 1972) but this does not correct the functional anismus dysfunction. Similarly with the solitary rectal ulcer syndrome rectopexy may allow healing of the
ulcer and avoid the risk of bleeding (Schweiger and Alexander-Williams 1977) by correcting the prolapse but again would not correct any functional abnormality of the sphincter and pelvic floor. The problem of perineal descent with anismus raises a particular quandary. Pelvic floor repair and post anal repair will restore the relative anatomy and reduce the relative obstruction due to distortion by the rectocele. However the position and function of the puborectalis will also be restored somewhat by the correction of the perineal descent (Browning and Parks 1983) and therefore once again the puborectalis may become a cause of obstruction if the anismus persists. One of the cases in this study suffered from a combination of anismus and rectal prolapse. She underwent biofeedback correction of the anismus prior to having the rectal prolapse corrected by a trans-abdominal ivalon sponge rectopexy. Thereafter she continued to be free of symptomatic anismus when reviewed six months after the rectopexy and ten months after completing the original biofeedback training.

As anismus subjects are unaware of the incoordination of the pelvic floor, biofeedback offers a fairly simple and minimally invasive technique for relearning suppression of the nonrelaxing pelvic floor (Almy 1979). A pneumatic manometric biofeedback device is available but this is more suited to relearning anorectal awareness of contents and thus avoiding faecal soiling or incontinence than it is for learning to inhibit the pelvic floor during straining (Constantinides 1983). On the other hand the Myotron 120 EMG device is ideally suited for retraining anismus
subjects (Bleijenberg and Kuijpers 1987). It is small and compact, easily operated and relatively inexpensive. It is important however that an experienced tutor instructs the subjects on what they are required to do and on what they are aiming to achieve with the device. The clinical aim is that of trouble free defaecation which may not always mean complete resolution of the anismus abnormality. We may have to accept that a minor degree of EMG anismus is acceptable so long as it remains asymptomatic.
CHAPTER V.1

DISCUSSION OF THESIS
There can be no doubt that spinal cord injury has a devastating effect on the human body (Guttmann 1973). However with improved medical and nursing care paraplegic subjects are now able and indeed expected to live for a normal lifespan (Kinnersley 1986). The main advances in management have been in initial care following the trauma (Swain et al 1985), reduction in sepsis and avoidance of renal failure (Grundy and Russell 1986). One of the quests of modern medicine is to improve the quality of life and, as paraplegic subjects now live longer they also deserve any improvements in quality which can be obtained for them. One such improvement would be a degree of voluntary control over micturition and continence. The Brindley S234 anterior sacral nerve root stimulator is intended to improve bladder emptying and reduce infections in the residual urine while giving continence of urine between between episodes of electromicturition (Brindley 1977). The Brindley stimulator fullfills these expectations and has the added benefit of enabling some male subjects to achieve penile erection sufficient for intercourse (Brindley 1982). The sacral S234 anterior nerve roots also carry the pelvic parasympathetic nerve supply to the left colon and rectum and the somatic nerve supply to the pelvic floor and external anal sphincter (Varma, Binnie, Smith et al 1986). As such the Brindley stimulator provides a unique opportunity to study the effects of direct electrical stimulation of the S234 nerve roots on left colon, rectum, pelvic floor and external anal sphincter function.

Investigations show that paraplegics have a severe constipation
related to significant delay in colonic transit time. There is improvement in colonic transit time in subjects with a Brindley stimulator showing that manipulation of the parasympathetic innervation of the colon has beneficial effects. Manometric studies show an increase in colonic activity in all regions of the left colon from the transverse colon to the rectum with the greatest activity at the splenic flexure. The S3 root stimulation has the most profound motility effects on the left colon with a motility gradient away from the splenic flexure in both directions proximal and distal. The distal one emptying the left colon but the proximal one creating a barrier to flow. There is a second area of functional barrier to transit at the recto-sigmoid junction. These two regions correspond to physiological regions of hold-up in the normal colon and may simply indicate regions of functional delay in colonic transit to enable normal colonic functions to be achieved (Holdstock et al 1970, Kirwan and Smith 1974, Krevsky et al 1986).

All paraplegics are not suitable for the Brindley sacral implant (Brindley, Polkey and Rushton 1988) and other means have been sought to aid them. The method of clean intermittent self catheterisation has revolutionised bladder management (Maynard and Glass 1987). For the bowel and for the first time in paraplegia an advance is that the gastrointestinal prokinetic agent cisapride is a possible treatment for the chronic constipation of paraplegia or a means of avoiding this chronic state (Binnie et al 1988). This agent selectively stimulates the gastrointestinal cholinergic myenteric plexus without any peripheral side effects likely to produce
autonomic dysreflexia or nausea. Transit studies show that cisapride reduces colonic transit time significantly without producing diarrhoea and incontinence in paraplegic subjects.

Stimulation of S4 anterior root by the Brindley implant produced dramatic contractions of the pelvic floor and external anal sphincter. Similar contractions of the urethral sphincter have previously been shown to occur following similar stimulation (Cardozo et al 1984). This effect of the stimulator would tend to produce an outlet obstruction to defaecation unless it could be arranged by appropriate stimulation that the rectal contractions could be maintained after relaxation of the external sphincter for example by deleting S4 effects or by increasing S3 effects, in which case defaecation could be produced.

As described earlier the agent cisapride generally increases gastrointestinal motility in paraplegia with an overall reduction in colonic transit time and some though not significant increase in faecal water content. On the other hand the electrical stimulation of the left colon parasympathetic nerve supply results in reduction of the colonic transit time but with a significant reduction in faecal moisture content. The difference between the results of the two types of stimulation is interesting and raises the question as to why this should be so. The studies of the left colon show that electrical stimulation of this area causes an emptying pressure gradient but there also appears to be an in-built control mechanism to hold up contents which therefore does not allow excessive amounts of liquid stool to overload the distal left colon
and sphincter mechanisms. In this situation the Brindley stimulator would appear to be activating an intrinsic control mechanism which already exists and merely requires some fine tuning from external neural sources to function correctly.

Faecal incontinence is recognised as one of the early problems after spinal cord injury but this quickly changes to a protracted constipation which has been investigated in this thesis. The problem of neurogenic faecal incontinence in the contemporary surgical scene is gaining in importance (Parks 1975, Neill et al 1981, Keighley 1984, Snooks, Barnes, Swash et al 1985). This type of incontinence is related to pelvic and pudendal nerve neuropathy often in association with prolonged straining and descent of the perineum such as occurs in the second stage of labour (Parks et al 1977, Snooks, Swash, Setchell et al 1984). Various neurophysiological investigations are required to establish the neurogenic cause of the incontinence (Swash 1985), including the pudendo-anal reflex latency (Varma, Smith, MacInnes 1986). This reflex test involves a stimulus over the dorsal genital nerve and recording the EMG response in the external anal sphincter. The latency from stimulus through the S234 portion of the conus medullaris to the external anal sphincter EMG response correlates well with the degree of neuropathy (Varma, Fidas et al 1987). A new anal plug surface electrode was designed with longitudinal electrode plates to give an improved pickup of external anal sphincter EMG compared to an older anal plug with the circumferential electrode plates. During this neurophysiological
investigation an original clinical observation was made which was that elicitation of the pudendo-anal reflex resulted in a visible contraction of the external anal sphincter onto the new anal plug electrode. The reflex and the response did not fatigue with a stimulation frequency of 1Hz. Moreover the subjects were generally unaware of the contractions and were not able to reproduce such strong contractions voluntarily afterwards. As a result of this observation a portable dorsal genital nerve stimulator was designed to reproduce the stimulus parameters used during the test situation and allow repeated stimulations of the muscle over a period of time provided the pudendo anal reflex arc was intact. A group of eight women with neurogenic faecal incontinence were treated with the stimulator with x3 five minute sessions per day for eight weeks. Seven out of the eight patients regained full control of faecal continence while the eighth failed to gain control of flatus only. The clinical benefits persist in excess of a mean of one year.

Higher neural control of the pelvic floor and external anal sphincter for both increased contraction and voluntary inhibition of spontaneous activity is lost in spinal cord injury. The pontine control of the lower spinal centres influencing the external anal sphincter (Weber et al 1985) is learnt or acquired at about the age of 2.5yrs (Holander and Frenckner 1983). There is however loss of this higher control of inhibition in subjects with inappropriate contraction of the pelvic floor during straining at stool (anismus) (Wasserman 1964, Barnes and Lennard-Jones 1985). This functional
abnormality can be diagnosed with the aid of the new surface anal plug electrode and is commonly associated with a high obstruction constipation often with an empty rectum and of course the paradoxical EMG response in the external anal sphincter during straining at stool. The control mechanism inherent since childhood for inhibiting the pelvic floor and absent in anismus can be relearnt by a biofeedback training using external anal sphincter EMG signals (Bleijenberg 1987). Fifteen subjects underwent a period of EMG biofeedback training and all achieved a significant improvement in their outlet obstruction with reduction in the anismus index. The benefits to the subjects persisted for at least six months after completing the biofeedback training.

Taking a wider view in the modern age of increasing trauma resulting in many spinal cord and neural injuries and with the expanding geriatric population with abnormalities of continence control, the need for a coordinated approach to bowel and continence problems is clear. As the new generation of investigative and therapeutic appliances are introduced and validated they should be seen not only as a means of treating the immediate clinical problem but also as providing tools for the further study of the physiology of the colon and perhaps leading to a better understanding of the disease processes.

The development of the new appliances described in this thesis and the opportunity to test them out has depended on multidisciplinary co-operation involving general surgeons, spinal cord injury surgeons, neurophysiologists, gastroenterologists,
bioengineers and computer experts. This reflects the challenging nature of the problems in this field and the skills that are available to help bring about their solution.
CHAPTER V. 2

CONCLUSIONS
The following short paragraphs summarise the conclusions drawn from the results presented in the previous sections as indicated.

Chapters I.1 to I.4 restate aspects of the problems of bowel function in spinal injury and the control of distal function in man, concentrating on aspects of constipation and incontinence with reviews of anatomy, physiology and methodology.

Chapter I.5. A new anal plug electrode with the electrode plates orientated in the long axis of the anal canal enables more sensitive recording of external anal sphincter EMG when compared to an older electrode with circumferential electrode plates.

The new electrode does not cause inappropriate contraction of the external anal sphincter by its presence in the anal canal and can therefore be used with confidence to test for anismus with genuine inappropriate contraction of the external anal sphincter when straining as if at stool.

Chapter II.1. Paraplegic subjects have a slow transit type of constipation due to prolonged colonic transit time which is associated with a reduced frequency of defaecation and reduced faecal water content.

The Brindley S234 anterior nerve root stimulator reduces the colonic transit time and increases the frequency of defaecation but is paradoxically associated with a reduced faecal water content. The latter may be due to relatively longer right colon transit time despite an overall reduction in complete colon transit time.
Chapter II.2 The Brindley stimulator increased colonic pressure wave height above basal levels in all areas from the transverse colon to the rectum. The greatest increase in the colonic mean motility index was at the splenic flexure.

The greatest peak wave height and mean motility index response was to S3 anterior nerve root stimulation which caused a motility gradient distally.

Stimulation of the S4 anterior nerve roots caused the greatest increase in intrarectal pressure.

Chapter II.3 Stimulation of the S4 anterior nerve roots caused the greatest increase in external anal sphincter pressure and EMG activity with decreasing effect from S2 and S3 anterior roots.

There was direct correlation with the maximum pressure generated by the external anal sphincter during S4 anterior root stimulation and the duration of the implant in keeping with hypertrophy of the external sphincter muscle.

The relative degree of reflex relaxation induced in the anal canal by the recto-sphincteric inhibitory reflex was less in the Brindley stimulator patients than in non-stimulator paraplegics indicating increased contribution by the external anal sphincter to the maximum resting pressure in the anal canal in the Brindley subjects at rest.

Chapter II.4 The agent cisapride significantly reduces colonic transit time in paraplegic subjects and increases intraluminal mixing of colonic contents in the process.

Cisapride also decreases the maximum rectal
capacity by increasing intrarectal tone and may have a similar
effect on the bladder reducing the residual volume of urine in
some cases. Abrupt withdrawal of oral cisapride may however result
in acute retention of urine in some paraplegics.

Chapter III.1 In subjects with neurogenic faecal incontinence
due to pudendal nerve neuropathy, the pudendo-anal reflex if
demonstrably present, can be repeatedly stimulated by a portable
non-invasive dorsal genital nerve stimulator to induce a
significant rise in the pressure and EMG activity of the external
anal sphincter, provided the reflex arc is intact.

Chapter III.2 The portable non-invasive stimulator can be
repeatedly used over an eight week period to significantly improve
the maximum resting pressure of the anal canal as well as the cough
and squeeze pressures of the external anal sphincter in patients
presenting with neurogenic faecal incontinence but in whom the
pudendo-anal reflex remains intact.

The resting or spontaneous EMG activity of the
external anal sphincter was significantly increased after the
course of stimulation.

The improvement in sphincter function was
accompanied by return of ano-rectal continence in almost all cases
treated. The improved continence persists for over one year after
completing the course of electrical stimulation treatment.

Chapter IV.1 In intractable obstructive constipation in subjects
with a non-relaxing pelvic floor when straining at stool (anismus),
an anismus index was derived by relating the rise in EMG during
straining to that during squeeze. The degree of anismus was assessed by the anismus index which was also used to assess the improvement produced by electrical biofeedback training.

The reduced degree of inappropriate contraction of the pelvic floor and external anal sphincter is accompanied by a reduction in time spent straining at stool and in the degree of difficulty encountered in passing stool. The benefits to the patients persist for at least six months after completing the EMG training exercises.
CHAPTER V.I.I

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CHAPTER VII.1

APPENDIX 1. INFORMATION ON EQUIPMENT
The list below itemises materials used in the work for this thesis giving the catalogue number and source from which they were bought.

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<tr>
<th>ITEM</th>
<th>CATALOGUE NO.</th>
<th>SOURCE</th>
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<td>Finetech-Brindley Stimulator</td>
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<td>Finetech Ltd 13 Tewin Court Welwyn Garden City Herts. AL7 1AU</td>
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<td>G.M.I. Breath Hydrogen Monitor</td>
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<td>G.M.I. Medical Ltd. Inchinnan Estate Renfrew Scotland PA4 9RG</td>
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<td>Radio-opaque transit pellets</td>
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<td>Portex Ltd 499/000/000 Portex Ltd 499/000/000</td>
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<td>Hythe Kent Hythe Kent</td>
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<td>800/002/065</td>
<td>England CT21 6JL 800/002/065 England CT21 6JL</td>
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<tr>
<td>Narrow bore 2mm tubing for motility triple balloons.</td>
<td>800/200/250/100</td>
<td></td>
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<td>Disposable manometer line.</td>
<td>200/490/200</td>
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<td>Small 3mm balloon for anal pressure profile.</td>
<td>3514</td>
<td>Precision Dripping Ltd Stover Trading Estate Yate 3514</td>
</tr>
<tr>
<td>Medium 7mm balloon for colonic motility.</td>
<td>3515</td>
<td>Bristol England BS17 5PG 3515</td>
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<tr>
<td>Large balloon for proctometrogram.</td>
<td>D 266 . 200</td>
<td>London Sheath Company D 266 . 200</td>
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<tr>
<td>(Capable of holding 4000ml with only 10 cmH2O rise in intrinsic pressure.)</td>
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England GL7 1PT

High Speed Chart Recorder
2 Channel MX 216 Ormed Ltd
6 Channel MX 6 Welwyn Garden City

Isolated Preamplifiers for
Ano Rectal Pressures 4820 Hertfordshire
Integrated EMG 4880 England AC2 3AW
Cystography 4912
Volume by weight transducer 3559
2 Channel Pre Amplifier MX2P
Housing Cabinet MX216

Multi function EMG M S 92 a Medelec Ltd
equipment with 2 channels,
averaging facility, chart
recorder and computer interface.

Ground electrode GE-V 53058
Single concentric needle MCDT 50 53002
Single fibre needle SF 37 53032
Needle holder cable NH 1 53021
Silver/silver chloride
Disc electrode kit E/RE - K 53065
Large bipolar stimulator LBS 53051
Electrode Gel 53078

Anal Plug Electrode 13 K 78 DISA
13 K 79 Dantec Electronics Ltd
Cable for anal plug
13 L 03 Redcliffe Way
electrode. Bristol

Digitimer Stimulator for
D 180 - 33 Digitimer Ltd
trans cutaneous lumbar
Welwym Garden City
stimulation.
Hertfordshire
England

BBC B Microcomputer
Double disc drive 40/80 switchable
CUB colour monitor medium resolution
Universal second processor
Sidewise ram expansion board
View
Viewstore
Viewsheet
ViewsPELL
Viewplot
ViewIndex
Dorsal genital nerve stimulator and Anal plug electrode
Patents pending (Appendix 2)
CHAPTER VII.2

APPENDIX 2. INFORMATION ON PATENTS APPLIED FOR ON NEW APPLIANCES
The new anal plug electrode and the dorsal genital nerve stimulator designed during work for this thesis were registered by Unived Technologies Ltd. as Proceedings under the Patents Act 1977 in The Official Journal (Patents), No. 5154 on the 14th of October 1987. Page 2444:

Anal Plug Electrode No. 8721254

Single Channel Stimulator No. 8721255
CHAPTER VII.3

SCIENTIFIC COMMUNICATIONS
The following presentations resulted from work carried out for this thesis.


July '88. Colon Club Workshop, Anorectal Physiology Methodology, Bristol. "Noninvasive EMG recordings" (anal canal).


May '89. Annual Clinical and Scientific Meeting of the Royal College of Surgeons of Edinburgh. Invited speaker at main symposium on the Surgery of Incontinence. "Neurogenic Faecal Incontinence."

May '89. Pelvic Research Group, Edinburgh "Noninvasive EMG diagnosis of anismus."

July '89. Surgical Research Society of Great Britain and Ireland, The University of Newcastle. "EMG Biofeedback as a domiciliary treatment of anismus."
CHAPTER VII.4

CONTRIBUTION TO TEXTBOOK AND PUBLICATIONS
The following publications resulted from work carried out for this thesis.

Smith A N, Binnie N R. Neural Disorders; in Clinical Measurements in Coloproctology: Editors; D. Kumar, D Waldron, N Williams. Springer-Verlag, London 1989


