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Leading article

Neurophysiological assessment of the pelvic floor

The improbable marriage of neurophysiology to proctology has proved highly profitable in that the two disciplines have combined to study clinical areas in common, to the mutual benefit of each. The starting point has been anorectal incontinence, but later the emphasis has been extended to include other functional disorders such as constipation.

Gowers could be described as the first physiologist to display scientific interest in anal canal function and was the first to describe the rectoanal inhibitory reflex.[1] Reflex responses affecting the internal anal sphincter were later studied in more detail by Denny Brown and Robertson[2] and the first studies of external anal sphincter function were by Beck[3] and Floyd and Walls.[4] Interest in the anal sphincter by non-clinicians virtually ceases at this point. Clinical contributions to anal sphincter function were made by Kerremans,[5] Schuster,[6] Frencker and Euler[7] who largely used manometric techniques. Such methods are relatively crude and are largely restricted to determining smooth muscle activity, that is to say internal anal sphincter function. Investigation of the striated component using conventional electromyographic techniques, showed that these muscles are tonically active at rest and during sleep[4] and contraction is reflexly evoked by coughing, sneezing or lifting heavy weights.[8] Such activity is facilitated by means of a spinal reflex arc[9] and as such is maintained in paraplegics if the level of cord section is above S2. Porter[10] was able to show that in patients with rectal prolapse the conventional EMG was abnormal, in that action potentials were of low voltage and there was a poor interference pattern during periods of attempted voluntary contraction.

The interpretation of these observations was possible thanks to the combination of a proctologist, a neurophysiologist, and a neuropathologist who showed evidence of denervation in the pelvic floor muscle biopsies of patients with faecal incontinence and rectal prolapse using elegant enzyme histochemistry.[11] The technique required visual assessment of the muscle under the microscope, however, and was open to observer error. An objective assessment of the innervation of the pelvic floor was then approached by studying the latency of the anal reflex in response to electrical stimulation of the perianal skin.[12] This method often produced paradoxical results, however, because of the suprasegmental nature of the reflex. A much more satisfactory approach to objective investigation of somatic muscle function has been by single fibre EMG.[13] The technique can be adapted to the study of pelvic floor musculature and has unequivocably shown that many patients with faecal incontinence have denervation damage to these muscles.[14]

Having established the presence of denervation, the next step was to

identify the anatomical site of the neurological damage. Because the efferent pathway can be considered to comprise a central component (within the spinal cord) and a peripheral component (the pudendal nerves) methods to test each were required. The former was achieved by transcutaneous stimulation of the lumbar spine using a method devised by Merton.[15] A single impulse of 800 to 1500 volts decaying with a time constant of 50 microseconds is delivered through two 1 cm in diameter saline soaked gauze pads placed over the spinous process of L1 and then repeated at the L4 position. In patients with peripheral nerve damage both latencies are increased more or less to an equal degree and the ratio of L1: latency L4 remains a constant. In patients with cauda equina disease however, the latency when the cord is stimulated at L1 is relatively longer than that at L4 and the ratio is increased.[16] By using such nerve stimulation techniques it can be shown that most patients with faecal incontinence have denervation of the pelvic floor secondary to peripheral nerve damage: spinal cord disease occurring in only approximately 24% of patients.[17]

Sensory aspects of anorectal function have been previously explored using relatively crude methods.[18] Roe *et al*[19] have developed a device for testing anal sensation using a probe to which is delivered a current of constant value and which is not determined by the nature of tissue in contact with the electrode. Using this method Rogers *et al* describe, in this issue, a group of patients with faecal incontinence who have a sensory deficit in the anal canal which correlates closely with the motor deficit.

Have these techniques advanced our comprehension of anorectal continence? These are tests largely of the integrity of the motor unit supply to the pelvic floor and have not, in truth, advanced our understanding of the dynamics of anorectal function. The spinal stimulation latencies to the puborectalis and external anal sphincter muscles are dissimilar, so confirming initial theories that these muscles have separate innervations.[20] The puborectalis is probably innervated on its pelvic surface by direct branches from the sacral plexus, while the external anal sphincter receives its supply from the pudendal nerves. In the research environment a neurophysiological approach has identified causative factors in many pelvic floor disorders. Denervation has not only been identified in rectal prolapse and faecal incontinence, but may also be a factor in constipation[21] and solitary rectal ulcer syndrome.[22] The important roles played by childbirth and defecation straining in initiating the denervating process are now well established.[23-25]

Many proctologists will claim that sophisticated neurophysiological investigation of the pelvic floor offers as much information as can be gained by educated digital examination of the anal canal. In routine practice this is partly true. An assessment of pelvic floor function based on squeeze pressure is unreliable, however, as many normal patients experience considerable difficulty in complying with a request to contract maximally. In certain cases, spinal stimulation studies will reveal the small subgroup of patients with spinal disease who might require a neurosurgical referral in preference to pelvic floor surgery. It has to be stated, however, that the present development of methodology is far from complete. In patients with severe atrophy of the pelvic floor musculature there may be very limited electrical activity generated and single fibre estimations may be misleading. Nerve stimulation studies can be uncomfortable for the patient and accurate electrical localisation of the pudendal nerves is not always achieved. There is

a continuing requirement then, to develop accurate techniques to quantify anorectal function. In the future, assessment of the total bulk of functioning sphincter muscle will almost certainly need the application of computer technology to neurophysiological techniques and it is hoped that the educated digital assessment of function will finally be rendered redundant.

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