Early and late components in the human anal reflex

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SUMMARY Perianal electrical stimulation induces reflex activity in the superficial part of the external anal sphincter muscle. Several components of differing latency can be recognised. It is suggested that these correspond with the ripples of contraction observed clinically after perianal scratch stimuli. Some of our earlier studies suggested an erroneously short latency for the first component of this cutaneously-elicited reflex. The reasons for this are discussed.

The anal reflex is usually elicited by pricking or scratching the perianal skin. After a brief delay a flicker of contraction in the superficial part of the external anal sphincter muscle is observed on the side stimulated and, to a lesser degree, on the opposite side. Rossolimo localized the anal reflex to the sacral segments of the spinal cord and more recent clinical and anatomical studies have suggested that the fourth sacral segments are particularly important. The anal reflex is absent in lesions of the cauda equina and it is also often absent in patients with idiopathic faecal incontinence. We have studied the latency of the anal reflex, recorded by an electrophysiological technique, in patients with idiopathic faecal incontinence, as part of our histopathological and physiological investigations of this problem. In our studies of normal subjects we have recorded a much shorter latency than that described by Pedersen and colleagues. Like other cutaneously-elicited reflexes, for example the blink reflex, the anal reflex probably consists of oligosynaptic and polysynaptic components of short and long latency, respectively. In this report the various components of the anal reflex recorded by our technique in a group of normal subjects are described, and the differences between our results and those of Pedersen et al are discussed. The latencies of the first component of this response are very short and various reasons for this are considered.

Methods

The anal reflex was elicited in the manner described previously. The patient was placed in the left lateral position and a ground electrode was strapped to the dependent thigh. A standard concentric needle EMG electrode was inserted, without anaesthesia, into the superficial portion of the external anal sphincter complex in the midline, 1 cm posterior to the anal verge. The perianal skin, 1 cm to 2 cm from the anus, was stimulated supramaximally (usually with a stimulus of about 90 V and 0.1 ms duration) with a bipolar surface stimulating electrode, and the reflex response recorded by the concentric needle EMG electrode was displayed on the oscilloscope of a standard EMG apparatus (Medelec EMG MS6). Stimuli were applied at a rate of 1 Hz. Using the storage mode of the oscilloscope it was then possible to distinguish the reflex response from random basal action potentials by its larger size, and by its constant latency (fig 1). The latency of the reflex response was measured from the stimulus to the first deflection of the reflex muscle action potential complex (fig 1). The latency of the first component decreases slightly with repeated stimuli and with increasing stimulus voltage and care was therefore taken to allow the latency to stabilise at the shortest value obtainable, before recordings were made, by suitable adjustment of the stimulus. Recordings of the anal latency in which the oscilloscope sweep occupied 50 ms or more were available in 27 of the larger group of normal subjects in whom we have studied this reflex. Most of these subjects were clinically normal relatives of patients referred for treatment of ano-rectal incontinence and all gave their informed consent for the recordings to be made. In these recordings late components were accepted if they appeared synchronously in all of the five consecutive traces made during each study, if they appeared more than 5 ms after the cessation of the initial complex of EMG activity in the anal sphincter muscle, and if they resembled the initial action potential complex in form and amplitude (fig 1).

Results

The latency of the first component of the anal reflex ranged from 6 to 13 ms (mean 9.3 ± 2.53 ms). A second component was identified in each of these 27 recordings. This second component followed the
first component after an interval of 15 to 33 ms (mean 23.9 ± 4.78 ms), at a latency from the stimulus of 23 to 45 ms (mean 33.2 ± 5.6 ms). These results are illustrated in fig 2. A third component was recognised in three recordings, occurring 5 ms, 7 ms and 9-5 ms respectively after the second component (fig 1).

Discussion

Clinical testing of the anal reflex is traditionally performed by pricking the perianal skin repetitively, or by dragging a roughly pointed object such as an orange stick across the perianal skin on either side of the anal orifice. These stimuli usually cause a brief contraction of the superficial and deep parts of the anal sphincter, resulting in several rapidly consecutive ripples of contraction in the muscle on the side of the stimulus. Clinical observation thus suggests that the anal reflex consists of several phases of activity in the sphincter muscle. In previous reports of our observations of the latency of the anal reflex following perianal electrical stimulation an early response with a latency of 8.3 ± 1.7 ms was recorded. The evidence that this early response is of reflex origin is discussed in our earlier reports.

In dissections of two female cadavers we found that the length of the pudendal innervation of the external anal sphincter was 20 and 28 cm respectively. Chantraine et al found that the pudendal conduction was 56 m/s. These figures give a theoretical value for the anal reflex latency, in women, of 8.6 to 11.5 ms, allowing for a synaptic delay of 1.5 ms. However, direct percutaneous stimulation of the spinal cord in two normal male subjects, reported in this volume by Marsden et al, gave a latency for conduction from L1 to the anal verge of 7 ms. Although this latency is probably shorter in most women because of their smaller stature it is likely that the very short anal reflex latencies recorded in some of our normal subjects, for example, 6-5 ms, are not reflex in origin, but must be due to spread of stimulus current directly to small branches of the pudendal innervation of the external sphincter muscles. The biphasic nature of the histogram of the first component of these latency measurements (fig 2) is consistent with this suggestion, perhaps indicating that only the second peak at 11 to 14 ms in this first component represents a true reflex response to perianal cutaneous electrical stimulation. The changes we have found in this latency in patients with pelvic floor disorders provide support for our contention that there is a short-latency component of the electrically elicited anal reflex in man, although the method we have used for studying this reflex seems not always to produce a clear reflex response. Indeed, in all our recent work on pelvic floor disorders we have preferred to use single fibre EMG fibre density measurements as a parameter of external anal sphincter function.

Our recent recordings, made using a slow oscilloscope sweep, have revealed a second component at 33.2 ± 5.6 ms, and sometimes a third component of this reflex response (figs 1 and 2). Pedersen et al in independent investigations of the anal reflex using a similar electro-physiological technique, found an anal reflex response with a mean latency of 50 ± 10.5 ms with no earlier component. In these investigations a cut-off relay of 15 to 20 ms duration was employed in the input to the preamplifier, making it impossible for the earlier component of the anal reflex to be identified. Pedersen et al noted that the latency of the reflex response they recorded varied with different stimulus parameters. Their stimulus, unlike ours, consisted of a train of five square wave pulses each 1 ms in duration, and each separated by an interval of 1 ms. It seems likely that the second component we have recorded at 33.2 ms is related to the response recorded by Pedersen et al, the difference in latency being accounted for by the difference in experimental technique. In our recordings, the earlier component was recorded in response to re-
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Repeated single shock stimuli applied at 1 s intervals. Our experiments were performed as control studies during our investigations of patients with anorectal incontinence and we have not had opportunity to investigate the possibility that this second component of the anal reflex, presumably representing polysynaptic activity in the anterior horn in the region of Onuf's nucleus, or the result of ascending relays within the cord, occurs bilaterally as is the case with the second component of the blink reflex.12 14

Finally, it should be noted that there are other possible ways of investigating the reflex control of the anal sphincter. For example, sudden rectal distension causes cessation of ongoing spontaneous activity in the external sphincter muscle, the rectosphincteric reflex, a phenomenon which accounts, in part, for the patulous anal orifice found in patients with faecal impaction. The latency of this response has not, so far, been determined but might prove a useful investigation in patients with lesions of the sacral cord, cauda equina, or sacral nerve plexus.

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References

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