SECTION VI

Theories of the Causation of Enuresis

CHAPTER 18

Bladder Function: Increased Frequency and Nocturnal Incontinence

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Increased Frequency of Micturition

Basically, the number of acts of micturition during any given period of time—the frequency—depends on the relationship between the urinary output during this period and the functional capacity of the bladder. The frequency of micturition can therefore be increased by either polyuria or a decrease in the functional bladder capacity.

In most older children and in nearly all adults, the functional capacity of the bladder can accommodate the urinary output during the night, so that nocturnal micturition is unnecessary.

Bladder Capacity

The true or structural bladder capacity is the capacity of the bladder divorced from nervous impulses, and depends, therefore, on local bladder structure. General anaesthesia has to be very deep to eliminate bladder contraction reflexes, so that the most accurate method of assessing true bladder capacity is by epidural or spinal anaesthesia; in practice, however, such accuracy is rarely required.

The functional bladder capacity is determined by the volume at which an evacuating bladder contraction is set up. Although one cause of a decreased functional capacity is naturally a decrease in structural capacity, in the vast majority of cases a decrease in functional capacity is due either to an increased discharge of sensory stimuli arising in the bladder or to defective inhibition by the higher levels of the central nervous system.

In cases of bladder irritability due to local inflammatory lesions (*i.e.* the group with decreased functional capacity due to an increase in sensory stimuli arising from the bladder), severe cystitis may involve the bladder muscle, and so eventually result in true structural bladder contracture.

An acquired reduction of the functional bladder capacity due to over-responsiveness to sensory stimuli or to lack of central inhibition, as a result of which the bladder is never allowed to become distended to its original anatomical limit, can lead to minor structural changes. These changes often appear to include the small vessels in the bladder submucosa, so that on distension under deep anaesthesia multiple petechial haemorrhages appear. In other cases, the bladder muscle structure appears to become 'set' at the maximum capacity allowed by the onset of reflex activity, and resists further distension at this volume. It is easy to picture how these states

may result in a vicious circle, whereby the increased reflex activity and the reduced functional bladder capacity are maintained.

The bladder capacity is often moderately reduced in children with enuresis (Starfield 1967, Gerrard and Zaleski 1969).

An Analysis of Urgency

There appear to be two basic types of urgency. In some cases, as has already been noted, it can be attributed to a dominance of sensory input over inhibition. In others, deficiency of reception of bladder sensations may result in the patient not perceiving the sensation of the desire to void until after the onset of the major bladder contraction (Fig. 1*b*). Descriptions by older children or adults with diurnal enuresis suggest that during the day the desire to void is not felt until the onset of the evacuating bladder contraction; as the patients themselves put it, 'the warning comes late—sometimes too late'. By the time the need to micturate has been perceived, attempts to prevent incontinence by simple voluntary inhibition are usually futile. The patient creates urethral resistance by voluntary contraction of the pelvic floor muscles or by crossing the legs. The power of the bladder contractions may then produce suprapubic colic. In some children, however, the bladder sensation appears to be so deficient that they may, especially if they are pre-occupied, wet themselves without being aware of it (Fig. 1). In such cases, the clinical impression that the need to void is not being appreciated until after the onset of the evacuating bladder contraction is sometimes supported by cystometry, which during filling often shows relatively powerful uninhibited contraction waves which do not produce any conscious desire to void. This

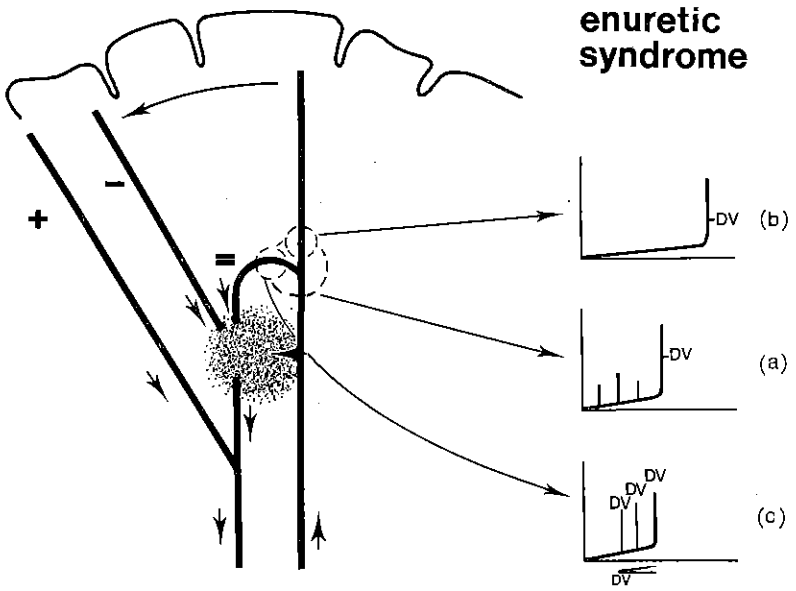

Fig. 1. Working hypothesis of dysfunction in the enuretic syndrome. Large dotted circle = original site of dysfunction (a). Small dotted circle = residual dysfunction: (b) sensory, (c) motor (defective unconscious inhibition).

'late warning' (hypaesthetic) mechanism of urgency (Fig. 1*b*) can not, however, account for any associated increased frequency of micturition—the latter can be attributed only to an input/inhibition imbalance mechanism (Fig. 1*a*).

An Analysis of the Enuretic Syndrome

Micturition during sleep must be due to deficient production or conduction of sensory stimuli from the bladder, or to a failure to receive, appreciate or react to them. The first of these possibilities (*i.e.* deficient production) seems to be highly improbable, and can be rejected if we accept that bladder contractions in response to distension normally produce a desire to void.

For micturition to occur during sleep, the bladder must fill to its maximum functional capacity, and the triggering stimuli be produced, before the night is fully passed. The mechanism of this early filling may, in occasional cases, be polyuria, but this is too exceptional to be accepted as being of basic importance in more than a very small proportion of bedwetters. Therefore, in addition to any possible deficiency of central reception of or reaction to afferent stimuli from the bladder, there must also be a factor of reduced functional bladder capacity. This reduced functional bladder capacity can be attributed only to a deficiency in the central inhibitory response to sensory input from the bladder.

Recovery from the most severe form of the enuretic syndrome, in which the patient is incontinent every night, and has frequency, urgency and precipitancy during the day, usually follows a set pattern. First the frequency starts to improve, but the diurnal and nocturnal incontinence persist. Then, as the frequency further decreases, the urgency and the diurnal incontinence also begin to disappear, but the nocturnal incontinence still remains unchanged. Finally, the nocturnal incontinence begins to improve and eventually disappears. This pattern suggests that the major dysfunction is sensory, the higher central nervous system failing to appreciate low level stimuli from the bladder, and responding only to the major stimuli produced by strong contraction of the bladder wall; by comparison, the deficiency in inhibition (*i.e.* the failure of the hypothalamus or subcortical centres to respond to stimuli from the bladder with suitable bladder inhibitory messages) would appear to be the more minor factor.

In other cases the nocturnal incontinence disappears, but the tendency to urgency and frequency, particularly during the day, persists into adult life—and may possibly be permanent. In such patients it would appear that only the afferent mechanisms had completely matured (Fig. 1*c*), the inhibitory mechanisms remaining deficient.

Cases of persistent nocturnal incontinence without urgency and frequency during the day, especially those in which the nocturnal incontinence has always been the sole symptom, possibly come into a different category from those with the complete syndrome. The genetic studies of Hallgren (1957) showed that this type of enuresis in particular has a strong genetic component. It is tempting to attribute the persistence of nocturnal incontinence in this group to habit (see Fig. 2*d*), and this suggestion is supported by the ease with which some of these cases can be cured.

The most likely site of origin of the functionally immature behaviours (including

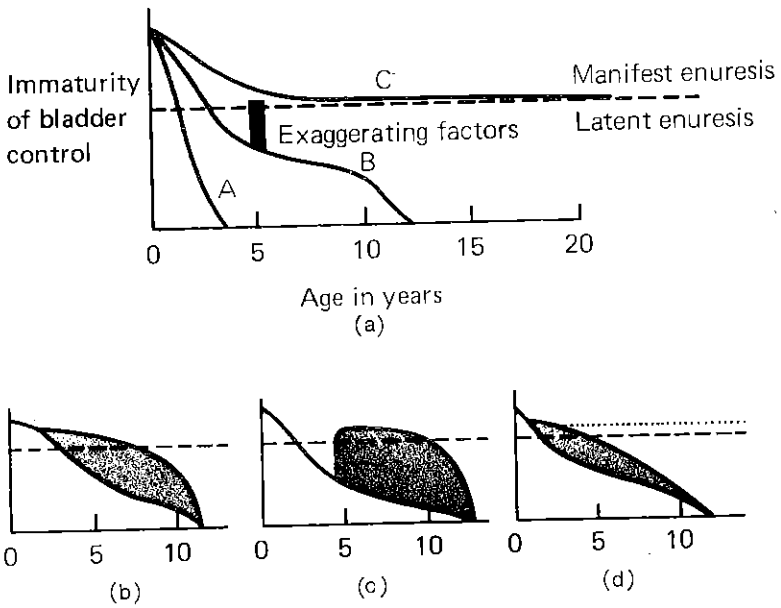

Fig. 2. Diagrammatic representation of concept of manifest and latent enuretic states. Interrupted line indicates enuresis threshold in each. Height of shading indicates extent of exaggerating factors. (a) Three rates of maturation of bladder control: A = normal; B = average delay (showing principle of exaggerating factors raising level above enuresis threshold); and C = failure to mature (enuresis always manifest). (b) Enuresis kept manifest till the age of 8 years by exaggerating factors. Latent till the age of 12 years. (c) Onset enuresis—enuresis made manifest at the age of 4 years by the occurrence of an exaggerating factor before maturation is complete. (d) Habit enuresis—manifest enuresis continues (dotted line) even after other factors have decreased or ceased to act. (*From* Yeates 1965.)

possible sleep disorders) which present as bedwetting seems to be between the cortex and the hypothalamus.

Concept of Latent Enuresis

The phase of unstable micturition control seen in the developing normal child may, in the apparently cured enuretic, persist for a number of years; that is to say, the condition may cease to be manifest but still be latent. If there is already an underlying deficiency of bladder sensation, a child with 'latent' unstable micturition control may become enuretic as a result of any condition ('exaggerating factor') which reduces the functional bladder capacity (Figs. 2*a*, line B, and 2*b*). According to this hypothesis, 'onset' enuresis can be thought of as latent enuresis which has been revealed (in a patient with bladder hypaesthesia) by the addition of an exaggerating factor (Fig. 2 *c*) (Yeates 1965).

Organic Findings in Enuresis

The absolutely negative findings on ordinary clinical and endoscopic investigation

in the vast majority of enuretic cases make it likely that abnormalities such as urinary tract infection and threadworms are merely associated with and are not causes of bedwetting. At most, some of them might make latent enuresis manifest. For example, severe cystitis might make evacuation during the night necessary when it would not otherwise have been so, by reducing the functional bladder capacity.

Cases of enuresis persisting for many years may show bladder trabeculation and an apparent obstruction of the bladder outlet by a bar at the bladder neck. The trabeculation may well be the result of oft-repeated voluntary urethral obstruction at the external sphincter level during bladder contraction. The appearance of a posterior bladder neck 'bar' might easily be the result of a posterior blow-out of the urethral wall between the contracted external sphincter and the more resilient bladder neck, this being a common finding in neuropathic bladder disorders with pelvic floor spasm. In any case, the bar at the bladder neck seems to be a consequence and not the cause of enuresis.

Conclusion

It would be unrealistic to suggest that every case of enuresis can be 'explained' on the basis of the above mechanisms, or that the physical findings, and the results of cystometry and cystoscopy always correspond with the clinical state. Bladder dysfunctions are still explicable only on working hypotheses—which do not always work.

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