CHAPTER 19

A Dual Theory of Nocturnal Enuresis

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Although enuresis has been recognised as a problem for many centuries, it is only during the last fifty years that the topic has been systematically studied, most of the major advances having occurred within the last twenty years. The earlier studies suffered from imprecision, in that the terms they used were ill-defined and they did not adequately explain or differentiate between the different forms of enuresis.

Treatment has always depended on the theories of origin which are in vogue at the time. The current sophisticated pharmacological and mechanical techniques of management have their primitive counterparts, just as the more humane psychogenic theories and therapies ushered in by Freud (1916) have their counterparts in the earlier magic ones.

Introduction to the Current Views of Enuresis

There have been a number of quite different, and at times contradictory explanations of enuresis, but recently it has become possible to discern some unifying threads. This paper constitutes an *exploratory* attempt to roughly weave these threads into the fabric of a dual theory, which proposes that nocturnal enuresis consists of two separate disorders, each with different origins.

Enuresis as a Psychological Disorder. Early in this century, the psychoanalytical explanation of enuresis as a disorder with a psychological basis received widespread approval, and many variants of the theory were elaborated. Unfortunately, the main theory failed to generate testable hypotheses, and, what is more, the possibility of the psychological disturbance being secondary to the enuresis was ignored.

Enuresis as a Disorder of Maturation. A second theory developed by psychologists is a behavioural one. This views enuresis 'not as a symptom of emotional disturbance, but as a failure to develop adequate cortical control over sub-cortical reflex mechanisms' (Lovibond and Coote 1971). Cortical control is seen as incorporating neural maturation and the development of a complex series of conditioned reflexes.

In addition to these two main theories, a number of more specific biological hypotheses have been advanced. Urinary tract defects have been reported (Campbell 1934, Barbour *et al.* 1963, Angell 1969), but are considered to be significant in only a small proportion of cases.

Physiological research (Muellner 1960) has lead to the hypothesis that enuretics have functionally small bladder capacities, which have to be increased to enable them not to wet the bed. Support for this theory has been qualified. However, an interesting modification of it has recently been advanced, namely that the limited functional bladder capacity is due to spasm (Gerrard and Zaleski 1969).

Other physiological research has been based on the alleged profoundness of sleep in a high proportion of nocturnal enuretics. Indeed, some workers have gone on to postulate that the basic defect consists of poor arousal mechanisms in the reticular system.

More recently, a concept has been evolving that views primary nocturnal enuresis mainly as a maturational disorder or deviation (Bakwin 1961, Barbour *et al.* 1963, Werry 1965, Kolvin *et al.* 1972), which has a genetic determination (Hallgren 1957, Barbour *et al.* 1963, Kolvin *et al.* 1972), and which shows an interaction with a number of other environmental influences. This concept does not preclude the possibility of different factors being responsible for the enuresis at different stages of development (Apley and Mac Keith 1968).

Definition and Classification of the Enuretic Disorders of Childhood

Early writers viewed enuresis as a homogeneous entity, and discussed the subject without any attempt at classification or differentiation. Only later was it appreciated that different types of enuresis might be associated with different or multiple aetiologies, natural histories, prognoses, and responses to therapy. Writers have not always made it clear whether they are reporting on cases of enuresis nocturna, enuresis diurna, or enuresis nocturna et diurna, nor whether their series include patients with both primary (or continuous) and secondary (or onset) types of nocturnal or diurnal enuresis. Even a simple classification into primary and secondary forms of enuresis provides some clinically relevant information, and has been shown to have some predictive value (Kolvin *et al.* this volume Chapter 26, Salmon *et al.* this volume Chapter 11)*. It is only since the introduction of classification that the previous confusion and ambiguities have given way to the emergence of more distinctive patterns.

Another complication is the fact that the term enuresis has been used to refer both to a disorder (or disease) and to a symptom (*i.e.* of some biological or psychological disorder) (Rutter 1965). Mac Keith (personal communication) advances the view that when considering a symptom it is necessary also to look further, not only for some underlying anatomical/physiological disorder, but also for the cause of this disorder. In psychodynamic literature, even primary enuresis is seen as a symptom, while elsewhere the term is used to refer to a disorder *sui generis*.

A more complex issue is what constitutes nocturnal enuresis. The issue is complicated by the fact that at age one bedwetting is undoubtedly normal or physiological, whereas at another age it is abnormal or pathological. By what criteria, then, should bedwetting be designated abnormal? In the first place, there is general agreement that urinary incontinence has to be excluded from consideration. Thereafter, there are various possibilities: we can define enuresis using an aetiological criterion, an age criterion, a statistical criterion, a functional criterion, an operational criterion, or a social anthropological criterion. An analysis of such criteria emphasizes their arbitrary nature; nevertheless, such distinctions still need to be made, to allow further research to be carried out in a systematic and meaningful way.

*The question needs to be asked whether enuretics should always also be categorised into those with and those without emotional disturbance, irrespective of any other classification.

An aetiological criterion leads either to a cul-de-sac, because of inadequate knowledge of aetiology, or to a sterile debate about what is pathological. At its simplest level, the statistical frequency criterion would define as enuretic any child whose bladder functioning was unusual as compared with that of most children of his age. The arguments for and against using either an age criterion or a statistical frequency criterion are ably presented by Werry (1965). He points out that the smoothly declining shape of the age prevalence curve makes any dividing line essentially arbitrary.

The solution adopted by Poussaint and Ditman (1965) was to utilise a functional definition, which avoided the necessity of stipulating either a minimum age or frequency; they defined nocturnal enuresis as a lack of nocturnal bladder control in a child in whom the act of voiding takes place in the normal way. Poulton and Hinden (1953) offered a similar definition. Such a functional criterion does not take into consideration the period after acquisition of day-time control when, for some months, night-wetting is accepted as normal.

There are two main operational definitions. Werry (1967) suggests that a practical solution is to define enuresis as night-wetting after the commencement of school, firstly because at this age the enuretic lies along the tail-end of the distribution curve and spontaneous remission is at its minimum, and secondly because the child himself is becoming concerned by his enuresis. However, even if one agrees on such age cut-offs, there still remains the problem of minimum frequency. Does occasional bed-wetting constitute enuresis? Such questions have led Hallgren (1956*a*) to operationally define enuresis as wetting occurring at least once a month.

Finally there are the social anthropological definitions, which are based on the attitudes of the parents or culture. Brazelton's definition (this volume Chapter 28) is a good example of this, as he views bedwetting as abnormal only when it produces overt concern in the parents (with or without a secondary deleterious effect on the child).

Elaboration of the Main Theories of Nocturnal Enuresis

A Biological Theory of Primary Nocturnal Enuresis

Some children who persistently wet at night beyond the age of five years appear normal in all other respects. This leads to the question of whether an 'isolated' delay in the achievement of bladder control can occur as with the so-called *developmental speech delays*. Indeed, a case has been made by a series of medical authors for regarding primary nocturnal enuresis as a developmental disorder, relating to difficulties in controlling the emptying of the bladder. Hallgren (1956*a* and *b*) and Bakwin (1961) believe that this disorder has a hereditary basis.

Barbour *et al.* (1963) speculate that the primary fault is a type of neurophysiological immaturity of structures subserving bladder control. A child who had not reached the necessary stage of maturation at an age when most children achieve bladder control would be particularly vulnerable to failure due to other factors, for example due to minor physical abnormalities of the urinary tract leading to difficulties in learning bladder control, or due to the inhibition of learning by emotional factors.

Partly following the argument put forward by de Jonge (1969), the case in favour of a maturational disorder can be summarised as follows.

(1) The half bell-shaped appearance of the age prevalence curve (de Jonge 1969) has led Barbour *et al.* (1963) to consider age the crucial factor in achieving dryness. But Mac Keith (1968, 1972*a*) argues against maturational delay being a common explanation beyond the age of six.

(2) There is a strong tendency to spontaneous improvement (Bakwin 1961, de Jonge 1969).

(3) There is some evidence to suggest that genetic factors influence the age of becoming dry (Bakwin 1961, this volume Chapter 9, Kolvin *et al.* 1972). Gunnarson and Melin (1951) report finding evidence of a genetic loading in primary as compared with secondary enuretics. Hallgren (1957) also reports finding that nocturnal enuretics are more likely to have a family history of enuresis.

(4) The relative somatic immaturity of bedwetters also points to a maturational disorder. For example, there is evidence of an increased risk of bedwetting in low-birthweight children (Oppel *et al.* 1968), and more specifically in small-for-dates children (Kolvin *et al.* 1973); Douglas (this volume Chapter 15) has found an excess of speech defects and, in older boys, of infantile secondary sex characteristics, and Miller (this volume Chapter 5) has suggested that their physical growth tends to be poor; there are also reports of significant immaturity of the EEG records of persistent enuretics (Gunnarson and Melin 1951, Salmon *et al.* —this volume Chapter 11).

(5) Biering (1959) suggests that bedwetters are emotionally immature, and Gunnarson and Melin (1951) report that children with infantile and immature behaviour occur twice as frequently amongst persistent enuretics. Hallgren (1957) also describes a significant association between enuresis, speech delay and immature behaviour. MacFarlane *et al.* (1954) report that persistent enuretics lose and acquire age-related behaviour significantly more slowly than non-enuretic children.

(6) Gender differences are reported (Hallgren 1957, Bakwin 1961, de Jonge 1969), which will be discussed later.

Another view of primary enuresis is that taken by the behaviour psychologists (Lovibond 1964, 1971), who place particular emphasis on the development of a complex series of conditioned reflexes in the process of learning bladder control. In other words enuresis is viewed as deficient learning of a habit pattern.

Like Werry (1965), we consider the above two theories (*i.e.* of a maturation disorder and of poor conditioning) to be complementary rather than alternatives to each other. Werry (1967) points out that the development of bladder control has two facets—maturation of the physical substrate and an experiential or learning component. Some would question the absolute necessity of the latter component, and would argue that, just as it is probable that walking skills can appear without any teaching (or learning), so it is feasible that, at times, bladder control can mature and develop without any teaching (Mowrer 1950, Mac Keith, this volume Chapter 20).

The limitation of the maturation theory on its own is that it does not adequately take into account such concepts as poor conditionability or environmental factors leading to poor or faulty learning or conditioning; similarly, learning theorists often

inadequately recognise the fact that non-learning is not necessarily a result of faulty learning, but could be due to the developing organism not yet being at a stage when it has the equipment for this particular form of learning (Kolvin *et al.* 1972). If the equipment were not yet 'mature', the acquisition of the habit pattern would at least be delayed. Further, the notion of a specific 'sensitive' period for the emergence of dryness, with its personal and social implications for the child and the family, has recently been advanced (Mac Keith 1964, 1968, 1972, and this volume Chapter 1).

We propose that many of the phenomena associated with primary nocturnal enuresis are explicable in terms of the biological theory, which takes into account both maturational and learning factors, though we also recognise that certain aspects of the theory may require modification with the advancement of knowledge.

A Psychodynamic Theory of Secondary Enuresis

In the secondary form of nocturnal enuresis, the child starts bedwetting again after a reasonably long period of bladder control. This breakdown of an acquired habit merits an explanation different from that put forward to account for primary nocturnal enuresis. Evidence currently available is not, however, unequivocally in favour of a psychogenic basis.

The psychogenic case is argued most cogently by Werry (1967), who points out that both he and Hallgren (1956*b*) have offered some evidence of emotional factors contributing to the development of secondary enuresis. He goes on to suggest that anxiety may cause irritability of the bladder, and that, consequently, a high level of chronic anxiety, in addition to its major disruptive effect on learning, could also interfere with bladder control in a child who was vulnerable to enuresis because of hereditary or constitutional factors. In other words, Werry suggests that, by itself, high chronic anxiety cannot cause secondary enuresis, but that a constitutional predisposition is also necessary.

Very little research on this subject has been conducted with reasonable scientific strictness. The most important study is that undertaken by Hallgren (1957). His series derives from psychiatric departments and, as might be expected, cases of psychological/psychiatric disorder are over-represented. Hallgren therefore concludes that his findings cannot be generalised to the enuretic population as a whole. Further, his criterion for statistical significance of $p < 0{\cdot}01$ is particularly rigorous, and as a result his two groups differ on only two of over thirty variables. His explanation for his rigour is the degree of uncertainty inherent in this type of data. But this is no greater or less than in any other data of this kind, so his data merit re-analysis using the usual criterion of $p < 0{\cdot}05$. If this is done, we find significant differences between the groups on five of the variables: gender, frequency of wetting, certain symptoms of nervousness, and familial incidence in both parents and sibs.

The other important study is that of Novick (1966), which points to a whole range of significant differences between primary and secondary enuretics in their response to treatment.

Hypotheses

On the basis of the above theories, two main hypotheses are generated. The first

is that primary nocturnal enuresis has a mainly biological basis; the second is that secondary or 'onset' enuresis has important psychological determinants.

However, it must always be remembered that signs of emotional disturbance do not, in themselves, preclude physical disease. These two hypotheses do not preclude the possibility that lesser psychological factors may be operative in primary nocturnal enuresis, or that lesser biological factors may be operative in secondary enuresis. The rest of this paper will be devoted to an examination of the hypotheses using evidence drawn from the literature and current research.

A parsimonious hypothesis that life-long and onset enuresis are both due to anxiety provoking stresses has been advanced by Mac Keith (1968).

Evidence From Physiology and Learning Theory

Muellner's (1960) theory is based on the supposition that enuretics have lower functional bladder capacities than non-enuretics. He postulates that an ability to hold urine for longer and longer periods goes with a gradually increasing tolerance for higher bladder volumes and, as a consequence, an increased functional bladder capacity. This control gradually extends to the sleeping state. Muellner sees the ability to hold more urine as being dependent upon maturation, and upon the exercise of consistent voluntary control. He claims that full voluntary control over bladder capacity occurs naturally, though Yeates (this volume Chapters 3 and 18) considers that both voluntary and involuntary factors are involved. Muellner does not consider the possibility that a low functional bladder capacity could be *due* to enuresis (Hallman 1950, Bakwin 1961), nor does his theory explain why some children with reduced day-time bladder capacities do not wet the bed. Nevertheless, two basic mechanisms in becoming dry at night appear to be the child becoming able to hold urine for longish periods during the day and the extension of this ability to the sleeping state (Lovibond 1971). Muellner's contention that enuresis is one manifestation of a general disorder of sensory-motor co-ordination is not supported by the sensory-motor study of Werry (1965). However, Werry used motor tests which do not necessarily reflect neural maturation, so the case rests on the circumstantial evidence previously summarised.

Whatever the current evidence in support of other theories of enuresis, a learning theory explanation cannot easily be set aside when predictions based on learning theory have, in the main, been fulfilled. Furthermore, Lovibond (1964, 1971) has produced evidence that when children are treated using the bell-and-pad apparatus the learning of nocturnal continence to prevent the bell ringing may be a voluntary process. This constitutes what is described as operant conditioning. But there is no general agreement among learning theorists as to how nocturnal continence is learned. If learning does play a part it must involve complex neurophysiological processes, and somehow mechanisms associated with diurnal bladder control must be extended to the sleeping state.

If enuresis is the result of faulty learning, the question arises of why poor learning occurs in some children and not in others. The most plausible explanation is offered by Mac Keith (1964, 1968), who invokes the concept of a sensitive period for the emergence of dryness as an explanation for the fact that the acquisition of dryness is particularly

common during the period from one and a half to four and a half years, but is subsequently more infrequent. He argues against simple maturational delay as a sufficient explanation (Mac Keith 1972), and states that delay in maturation is an unlikely explanation for primary enuresis persisting after the age of six. Mac Keith distinguishes between: (1) the development of control in the sensitive period (for which maturation is essential); and (2) the later acquisition of dryness. Even a transient interference with acquisition during the sensitive period may leave the child without control for a number of years, during which acquisition will apparently be more difficult. When poor bladder control persists, new anxieties may be preventing the emergence of dryness.

On the other hand, the weight of evidence in support of a theory of maturational delay is overwhelming, and cannot be ignored.* Kolvin *et al.* (1972) postulate that an extreme variation in maturation may lead to a delay in the *readiness* of the child to acquire a new skill. In other words, the equipment necessary for the learning of a particular skill may only be available at a later, possibly genetically determined, stage of development. This explanation would be unsatisfactory to those who claim that the mechanisms are mature in those enuretics who do not wet the bed in unfamiliar surroundings, such as in hospital (Mac Keith 1968). So far, no one has speculated that the lack of bladder control in such children and adolescents may be due to immaturity at the level of the reticular formation resulting in a relative insensitivity to ascending stimuli, and that in unfamiliar surroundings this 'block' may be overcome by the existence of unusual arousal stimuli.

If we accept that learning does play a part in the acquisition of nocturnal continence, we have to consider those factors which are likely to inhibit or promote learning, both during the second, third and fourth years when control is usually acquired, and later on as well.

Firstly, simple factors need to be considered; for example, some enuretic children are found to come from very poor homes where not even elementary toilet training techniques have been applied. In such cases it is often hypothesised that the wetting is a direct consequence of the poor training: hence we have the concept of the 'under-trained' bedwetter. There is support for this concept in the literature, both from the clinical experience of Bakwin (1961), and the research of Blomfield and Douglas (1956). Bakwin (1961) describes the following factors as being conducive to the learning of dryness: a home where cleanliness is prized; a toilet close at hand; a mother who is sensibly solicitous; sensible regulation of the child's fluid intake; regular 'lifting' at night, and a home which is warm at night. Blomfield and Douglas (1956) found a correlation between early training and early bladder control, irrespective of social class. Brazelton (1962) emphasises how dryness is promoted by an absence of parental pressure. Douglas (this volume Chapter 15) has shown that a number of deleterious factors in three- and four-year-olds may be associated with a higher incidence of bedwetting at later ages.

*An opposite case is presented by Mac Keith *et al.* (this volume Chapter 1), who argue that in bedwetting by children over five maturational delay is not the cause, though in children under five it may be the reason why they do not develop nocturnal bladder control.

Werry (1965) has described two sets of factors which delay the emergence of nocturnal continence: (1) physical factors, which interfere with learning at the structural level; and (2) emotional tension, which interferes with the learning processes. These are discussed elsewhere.

The hypothesis concerning the poor conditionability (Eysenck 1957) of enuretics is discussed elsewhere (Kolvin *et al.*, this volume Chapter 26), and the conclusion is reached that, while such a hypothesis cannot be discounted, it receives only limited support from the research cited. The possibility remains, however, that in some enuretics poor conditionability may play a part in hampering the emergence of dryness.

Finally, the influence that may be exercised on a pre-existing developmental delay by a combination of other factors is emphasised by Barbour *et al* (1963), and elaborated by Kolvin *et al.* (1972): 'it [the delay] may be exaggerated by "errors" of learning at the toilet-training era, by inhibition of learning by transient emotional factors (Mac Keith 1968), or by the presence of minor anatomical abnormalities creating increased difficulties in learning'.

Secondary enuresis can quite easily be accounted for in terms of both learning and dynamic theory, and the subject is more appropriately discussed in a subsequent section.

Physical Factors

Physical defects of the urinary tract have been emphasised as a cause of enuresis by some authors (Campbell 1934). On the other hand, others have denied both their significance and their presence (Barbour *et al.* 1963, Angell 1969), a view supported by the finding of Kuzemko (1967) that bedwetting is not particularly common amongst children with clear organic pathology. In spite of the negative evidence from the literature of an association between physical defects of the urinary tract and enuresis, it seems reasonable to hypothesize that primary enuretics will be found to be more likely to have physical defects which hamper rather than prevent learning, and there is an important need for studies to see whether there are differences between primary and secondary enuretics in this respect. In support of the above hypothesis is the evidence of Oppel *et al.* (1968), who demonstrated that children who showed some abnormality on clinical neurological examination were more likely to fall into their 'never dry' category.* It is also interesting to note that children with primary nocturnal enuresis wet more frequently than those with acquired nocturnal enuresis (Hallgren 1957).

The relationship between epilepsy and enuresis has not been adequately established, most research workers finding neither a higher incidence of epilepsy amongst their enuretic patients (Hallgren 1957, Poussaint and Greenfield 1966), nor a higher incidence of enuresis amongst epileptics (Poussaint and Greenfield 1966). Only Salmon *et al.* (this volume, Chapter 11) have so far attempted to distinguish between

*While the proportion of enuretics with proven urinary tract infection is low, it has been customary to carefully check persistent cases for such infection. Whether urinary tract infection is causative of enuresis is the subject of another chapter (Stansfeld, this volume page 102), but it is important to mention here that practising paediatricians should pay particular attention to this possibility in the case of secondary enuresis.

primary and secondary enuretics, and their general conclusion is that epileptic recordings are not common in either group.

Deep Sleep Patterns

One of the most widely propagated theories concerning enuresis is that the basic fault is poor arousal to bodily or environmental stimuli due to unusually deep sleep (Bostock 1962). As Bostock's research was undertaken on a very small number of cases, his findings are not convincing. However, Braithwaite (1956) also reported an association between enuresis and deep sleep, finding an inordinate depth of sleep significantly more frequently in his large group of enuretics than in a group of controls. Other evidence to support the deep sleep theory is the fact that some primary enuretics cease to wet the bed in unfamiliar surroundings, a finding which may be attributable to heightened sensitivity at the reticular level to arousal stimuli.

The only clinical data available comparing the depth of sleep of primary and secondary enuretics is that of Hallgren (1957) and Kolvin *et al.* (this volume, Chapter 26). Such enquiries suffer from the handicap that they are entirely dependent on parental accounts, which are considered by some to be particularly susceptible to 'expectation' distortions. Hallgren does not describe any significant differences. Kolvin *et al.* report only a moderate correlation (0·5) between parental reports of depth of sleep and crude evidence of nocturnal restlessness, measured in terms of the amount of body movement. It is noteworthy that deep sleep significantly correlated with a series of health measures including psychiatric normality, and that a relative absence of nocturnal restlessness correlated significantly with primary enuresis. These correlations point to less restlessness in the primary, and more restlessness in the secondary enuretic. Also, the variable 'depth of sleep' correlated at a marginally lower level with primary than with secondary enuresis. This finding suggests that greater depths of sleep are generally associated with primary nocturnal enuresis and lesser depths of sleep with secondary enuresis.

In view of the above findings it would appear important to ascertain the electrophysiological levels at which enuretics wet, and whether there are differences in this respect between the different types of enuretics. In contradistinction to clinical impressions, enuretics wet at all electrophysiological stages of sleep (Ditman and Blinn 1955, Broughton and Gastaut 1963, Ritvo *et al.* 1969), except during rapid eye movement (REM) sleep. One of the patterns described by Ritvo *et al.* (1969) fits well with the hypothesis that enuretics tend to sleep more deeply than non-enuretics; this was a pattern of non-arousal enuresis associated with minimal maladjustment, regular wetting, a nonchalant attitude, profound depth of sleep and a strong positive family history of enuresis. Unfortunately, the significance of such tantalising findings is questionable because of the smallness of the series.

In summary, the attractive neurophysiological theory that attributes primary enuresis to profound sleep and poor reception of sensory stimuli at the level of the reticular formation receives only marginal support from clinical research, and so far receives almost no support from electrophysiological experimental work. Nevertheless, further research is essential to ascertain whether meaningful clusters or patterns of

familial, behavioural and physiological variables can be uncovered which could make a scientific contribution to the classificatory exercise.

Psychological Evidence

Psychological evidence has to be evaluated in relation to the different types and origins of nocturnal enuresis. The first question to be asked is whether nocturnal enuretics show an excess of behavioural and personality abnormalities and, if they do, whether the disturbance is specific or general. We must then look for differences in personality between primary and secondary types of enuretics. Recent research has provided irrefutable evidence of an excess of psychological disturbance in enuretics (Werry and Cohrrsen 1965, Shaffer *et al.* 1968, Kolvin *et al.* 1972). The Isle of Wight Survey (Rutter *et al.* this volume, Chapter 17) has provided evidence that there is 'a moderately strong association between enuresis and emotional or behavioural disturbance in girls, but the association is less marked in boys'.

On the other hand, it must be emphasised that in the Newcastle Study (Kolvin *et al.* 1972) the vast bulk (71 per cent) of the enuretics proved to be psychiatrically normal, and personality-wise the enuretics differed very little from the norm on the Cattell Personality Questionnaire (C.P.Q.). Nevertheless, the incidence of emotional disturbance in nocturnal enuretics was high (29 per cent).

Oppel *et al.* (1969) report that 'never dry' children are significantly more emotionally sensitive, less ambitious, more withdrawn and more likely to suppress their feelings than their permanently dry counterparts. They also provide information on relapsed cases. However, their definition of relapse is wetting after a minimum of one month's dryness, so their findings tell us nothing whatsoever about secondary enuresis as it is currently defined.

Other detailed information on the behaviour of primary and secondary enuretics is provided by the data of the Newcastle survey. As reported by Kolvin *et al.* (1972), the children were rated by a psychiatrist on seventeen behavioural items, each of which was scored on a four point scale, with points three and four being indicative of clear-cut deviance. Further investigation, using a simple classification into two groups, *i.e.* those with no or little behavioural deviance (score 1 and 2) and those with clear-cut deviance (score 3 and 4), has revealed certain differences between the primary and secondary enuretics, in that on a number of items a higher proportion of secondary than of primary enuretics was classified as showing clear-cut deviance. On only three items (solitariness, fearfulness and obsessiveness), however, were the differences of statistical significance, although on two others they were almost significant (Table I). The greater behavioural deviance of the secondary enuretics was also reflected in their higher mean score for all seventeen items added together, although here again the difference fell short of statistical significance (Table I).

The above recent findings are of importance in view of the negative correlations previously reported by Lapouse and Monk (1959), who did not find any association between enuresis and behaviour disorders. However, these authors did not classify their cases of enuresis into primary and secondary types. Other negative evidence was provided by MacFarlane *et al.* (1954) and Bakwin (1961), who did not describe any signs of psychological disturbance as being characteristic of enuresis. The evidence

TABLE I

Behaviour

Feature	*Primary enuretics*				*Secondary enuretics*				*Significance*
	1 + 2*		3 + 4†		1 + 2*		3 + 4†		
	No.	*Per cent*	*No.*	*Per cent*	*No.*	*Per cent*	*No.*	*Per cent*	
Sensitive	28	38·4	45	61·6	4	19	17	81	N.S.
Solitary	66	90·4	7	9·6	15	74·4	6	25·6	$\chi^2 = 4{\cdot}93$, $p < 0{\cdot}05$
Fearful	60	82·2	13	17·8	12	57	9	43	$\chi^2 = 4{\cdot}75$, $p < 0{\cdot}05$
Obsessive	60	82·2	13	17·8	12	57	9	43	$\chi^2 = 4{\cdot}75$, $p < 0{\cdot}05$
Highly strung	51	70	22	30	11	52·4	10	47·6	N.S
Mean total score on 17 items	30·4 (S.D. = 6·14)				33·42 (S.D. = 6·86)				$t = 1{\cdot}93$, $p < 0{\cdot}1$

*Score 1 + 2 = little or no deviant behaviour.
†Score 3 + 4 = clear-cut deviant behaviour.

that secondary enuretics are more psychologically disturbed than primary enuretics (Hallgren 1956 *a* and *b*, Beaucham *et al.* 1965, Oppel *et al.* 1969, Kolvin *et al.* 1972) is inconsistent (Rutter *et al.* this volume Chapter 17).

The theory that in most cases enuresis has a deep-seated psychogenic or psychological origin has very little hard factual evidence in its favour. Available evidence is mainly derived from uncontrolled clinical impressions, which are mostly unsupported by empirical facts, so it is difficult to know what weight to attach to them. That an enuretic displays signs of a psychiatric disturbance is not proof of a psychogenic origin, as the disturbance may be a reflection of the enuresis. A necessary condition which must be met before a psychogenic explanation can be anything more than speculative is that the postulated precipitating stress should have preceded or coincided with the onset of secondary enuresis, or, in cases of primary enuresis, have been operative at a time when the child would normally have been learning to be dry.

In spite of these difficulties, the most widely expressed view of enuresis is that it is a psychogenic manifestation. Kanner (1957) sees secondary enuresis as part of a more general regression into infantile modes of behaviour. Paediatricians and child psychiatrists (Wolff 1969), on the basis of their clinical experience, commonly describe recognisable signs, which they report as having antedated or coincided with the onset of the enuresis. However, the results of systematic research do not consistently support such clinical findings. De Jonge (1969), in his epidemiological study, has systematically looked for possible psychological causes in 34 cases of secondary enuresis. By careful enquiry, admittedly retrospective, he was only able to identify disturbing events in half the cases. Those disturbing events which he did find proved to be fairly diverse, non-specific and universal phenomena, (*e.g.* the birth of a sibling), and there were no grounds for attributing the enuresis to them.

On the other hand, Paulett and Tuckman (1958) reported maternal pregnancy as occurring more frequently in children who afterwards developed 'onset' enuresis than in an age-matched population of non-enuretics, and Hallgren (1957) describes emotional precipitants as occurring in 55 per cent of his cases of 'acquired' enuresis. The Isle of Wight research (Rutter *et al.* this volume Chapter 17) reveals that 50 per cent of the children who were dry at five years but wet at seven years (*i.e.* secondary enuretics) showed deviant behaviour at five years, whereas, of those who were dry at five years and still dry at seven years, only 11 per cent had behavioural disorders. Thus there was an apparent tendency for an excess of deviant behaviour to *precede* the appearance of secondary enuresis. Rutter *et al.* consider their findings incompatible with the theory that behaviour disorders found in secondary enuretics are reactive, *i.e.* that behaviour disorders in cases of onset enuresis are secondary to the enuresis. However, their findings do not appear to be inconsistent with the theory that onset enuresis is a secondary reaction to, or a symptom of, long-standing anxiety.

The ambiguity, vagueness, complexity and esoteric nature of the concepts upon which the earlier psychogenic theories were based frequently led to their outright rejection as explanations of primary nocturnal wetness. The psychogenic theory has recently been revived and modernised by Mac Keith (1968), who differentiates between earlier, often transient, adverse psychological experiences which have prevented emergence of nocturnal bladder control at the age when most children become dry, and the enuretic's existent disturbances, sometimes secondary to enuresis, which at a later age are preventing him from learning bladder control. In his parsimonious theory, Mac Keith postulates that transient anxiety at the 'sensitive stage' of learning bladder continence may lead to a perpetuation of wetting, but leave no residual emotional scars. He seeks evidence from the epidemiological research of Douglas (this volume Chapter 15) to demonstrate an excess of psychological stresses during the third year of life in children who later fail to become dry or who become wet after a period of having been dry.

Some mention must be made of the children who appear to wet deliberately. They seem consciously to empty their bladders in bed or in other inappropriate circumstances, in order to seek attention or in order to manipulate the situation to meet their needs, or to express aggression or hostility. If such wetting could be positively associated with secondary enuresis, this would provide an important prop for the psychogenic theory. However, such cases are rare, and, as yet, no-one has indicated what their association is, if they have one, with primary or secondary enuresis.

Evidence From Therapy

Finally, if the theories we have offered to explain the two types of enuresis have any validity, then one would expect predictions based on such theories to be fulfilled. Though research to assess the effects of psychotherapy has in general produced negative findings (Werry 1965), White (1968) has claimed achieving a good response to 'psychological treatment' amongst a group of secondary enuretics. The significance of White's results is impossible to evaluate, as the author did not specify what the treatment consisted of, nor did she specify what constituted improvement.

Shaffer *et al.* (1968) have also reported a better response to psychological treatment amongst secondary enuretics, though the work of Kolvin *et al.* (this volume Chapter 26) suggests that the response of secondary enuretics to behaviour therapy is less favourable than their (temporary) response to imipramine treatment. Kolvin *et al.* also report that, irrespective of the regime, psychologically more stable children respond better to treatment. The above findings cannot be taken to imply that imipramine does not work in primary enuretics, since researchers such as Kardash *et al.* (1968) have demonstrated that it is clinically and statistically superior to a placebo in controlling primary enuresis; furthermore, Kolvin *et al.* have demonstrated only a non-significant trend for behaviour therapy to be more effective than imipramine in cases of primary enuresis.

Both these pieces of research suffer from a deficiency in the number of secondary enuretics investigated (in the study of Kolvin *et al.* this deficiency is specified and in that of Shaffer *et al.* it is presumed). It is of course difficult to assemble a large sample of onset enuretics. The largest and most comprehensive is that of Novick (1966), who studied 22 primary and 23 secondary enuretics. However, his results appear contradictory. While he describes statistically significant differences between the two groups in response to treatment, he surprisingly reports no significant differences between them on a series of psychological, social and background variables. It seems difficult to see how such discrepant results in relation to aetiology and therapy can co-exist.

Unfortunately, Novick's paper contains no simple statistical information regarding the psychological, social and background variables investigated, so we have no opportunity for examining possible trends in respect of these factors. As the two groups contained few cases, any aetiological differences elicited, even though not statistically significant, could have been large and worthy of further consideration. As it is, all that can be concluded is that Novick's research provides no useful information on psychological, social and background variables.

As far as the results of treatment are concerned, Novick reports that in comparison with his primary enuretics his 'acquired' enuretics showed a more favourable response to routine supportive therapy alone; they took fewer days to reach the criterion of cure; they wet less frequently prior to reaching the criterion of cure; they showed a faster rate of decrease in wetting prior to reaching the criterion of cure; at follow up, after cure, they were wetting significantly more frequently; they had more new symptoms after treatment; and, according to the mothers' reports, they showed a deterioration of behaviour in more areas. Such differences in response to treatment were not reflected on three other variables investigated. Novick concludes that his findings confirm the hypothesis that acquired and life-long enuretics:

(1) differ in their response to symptomatic treatment;
(2) differ in respect of the long-term effectiveness of the cure produced by this type of treatment; and
(3) differ with regard to the behavioural after-effects of symptomatic treatment.

While further research is required before the issue can be unequivocally settled, studies of differences in response to therapy do appear to lend some support to the theory that there are two types of enuresis and that secondary enuresis has a psychogenic basis.

Epidemiological, Social and Parental Factors

Epidemiological evidence (de Jonge 1969, Miller this volume Chapter 5) favours the view that the two types of enuresis found in children over the age of five have different natural histories. In primary enuresis, the pattern between the ages of five and ten years is of a fairly steady decrease in numbers; with secondary enuretics, there is a marked increase in numbers at about six years, and thereafter children with this form consistently account for a relatively higher percentage of all enuretics, although the exact proportion of primary and secondary enuretics varies erratically.

Rowntree (1955) has demonstrated a correlation between certain types of broken home and high rates of enuresis, and this finding has been confirmed by de Jonge (1969) and Douglas (this volume Chapter 15). Douglas has also suggested that certain types of family crisis have occurred more frequently in the third year of life in enuretics. This is an area of enquiry in which further epidemiological research is likely to prove rewarding.

A series of researchers (Hallgren 1956 *a* and *b*, Stein and Susser 1966, Kolvin *et al.* 1972, Miller this volume Chapter 5) have demonstrated that bedwetting is more common in the lower social classes. Other recent research (Rutter *et al.* this volume Chapter 17) describes only a weak and inconsistent association. The crucial question is: if social factors are important, how do they exert their influence? While some (Stein and Susser 1966) suggest that operative social influences transcend all other aetiologies, there is no generally agreed explanation of how these influences are mediated. It would seem reasonable to speculate that the higher rate of enuresis in the lower social classes is due both to poor conditioning and poor environmental experiences. So far, the excess of enuretics in abnormal populations such as delinquents (Stein and Susser 1966) has been inadequately explained, though the speculative suggestion has been advanced that it may be related to social influence. However, the pertinent question in relation to the hypothesis under scrutiny is whether primary and secondary enuretics have different social class distributions. One of the few research groups to look into this question (Kolvin *et al.* 1972) report no differences, but the size of their sample is too small to allow any firm conclusions.

Familial Evidence

The main questions to be asked are: (1) is there a genetic basis for enuresis? and (2) are there differences between the primary and secondary forms in respect of the aetiological significance of genetic factors?

The first question is tackled by Bakwin (this volume Chapter 9) who, in his brief paper, reports that monozygotic twins were significantly more likely to be concordant for enuresis (*i.e.* both to have enuresis) than dizygotic twins. This is a crucial study, because it attempts to make the necessary distinction between familial transmission and genetic inheritance. However, it is not clear whether Bakwin has differentiated between primary and secondary nocturnal enuresis, and therefore this work makes no contribution to our 'dual theory' hypothesis.

Other genetic/familial evidence appears contradictory. Gunnarson and Melin (1951), Hallgren (1957) and White (1968) provide evidence in favour of a *significantly* higher familial incidence in primary than in secondary nocturnal enuresis. In contrast,

Barbour *et al.* (1963), from a of small series cases, give figures which show only a slight trend for primary enuretics to have a higher familial incidence. Likewise, the importance of familial factors when considering the prognosis, though emphasised by some researchers (Kolvin *et al.*, this volume Chapter 26) is denied by others (Barbour *et al.* 1963).

Over-all, however, the balance of evidence is in favour of the view that genetic/familial factors are of greater importance in primary nocturnal enuresis than in secondary nocturnal enuresis.

Gender Differences

Nocturnal enuresis is more common in boys (Bakwin 1961, de Jonge 1969, Kolvin *et al.* 1972), but the gender differences are not as clear-cut as those usually found in other developmental disorders (Rutter *et al.* this volume Chapter 17).

De Jonge (1969) found a smaller male/female ratio amongst secondary enuretics in the six to twelve years age range, than amongst primary enuretics of the same age, though the difference was not significant. Hallgren (1957) reports finding a higher proportion of girls than boys with acquired nocturnal enuresis, and in his series the difference between the primary and secondary enuretics does achieve statistical significance ($p < 0{\cdot}05$). These findings are reasonably consistent with the view that primary enuresis is a developmental disorder, and provide important evidence in support of the hypothesis that primary and secondary enuresis are separate and distinct.

Discussion

In this paper, we have advanced a simple theory which proposes that nocturnal enuresis consists of two separate disorders, *i.e. primary* or '*continuous*' enuresis and *secondary* or '*onset*' enuresis.

It is hypothesised that primary enuresis has a mainly biological basis, possibly with some minor psychological determinants, while secondary enuresis has a mainly psychogenic basis, possibly with some minor biological determinants.

A survey of the literature and current research provides only limited evidence to support the various aspects of the hypothesis. Nevertheless, there is sufficient evidence to suggest that nocturnal enuretics do consist of at least two different populations of children. However, there are many areas in which these two populations overlap, and this leads to the crucial question of whether they are fundamentally distinct and separate, or whether one is a variant of the other. It is possible that secondary enuresis may result from interference by anxiety or other factors with poorly learned processes associated with bladder control. The interference may be the result of acute stresses, as described by Hallgren (1957) and Paulett and Tuckman (1958), or some long-standing emotional disorder (Rutter *et al.* this volume Chapter 17). On the other hand, there is evidence that earlier transient psychological stresses are operative in primary enuretics (Douglas this volume Chapter 15). It is also clear that the theory of primary enuresis being a disorder of maturation is quite compatible with the possibility that important psychogenic influences may prevent the acquisition of continence in an individual whose potential for learning continence is already impaired. Familial

factors are certainly important in primary nocturnal enuretics but they also appear to be operative, albeit at a lower level, in secondary enuretics.

While it appears likely that primary nocturnal enuresis is a developmental disorder, the status of secondary nocturnal enuresis remains less certain. It may be that secondary enuretics are a less homogeneous group, consisting: (a) of cases in which continence has been poorly learned (due to a combination of neurophysiological immaturity and early adverse psychological influences), or in which there is an underlying deficiency of bladder sensation with the enuresis remaining latent (as hypothesised by Yeates—this volume Chapter 18); and (b) of cases in which control has been well learned, but in which there is later a breakdown of control due to acute or chronic stress.

It would, of course, be naive to expect such a simple theory to explain anything more than the major aspects of primary and secondary enuresis, as the part played by many other factors has to be taken into account. At present, it must be concluded that the proposed explanatory theory has only moderate validity.

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