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SUB-CATEGORIZING HYPERACTIVITY - A REPLICATION AND EXTENSION OF THE STONY BROOK SCALE

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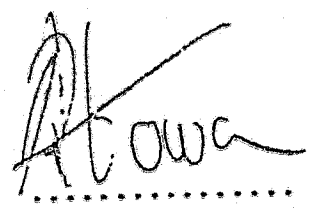
Johannesburg, 1982.

I hereby declare that this dissertation is my own work and that I have not submitted it for a Master's Degree at any other university.



R.A. Cowan

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Professor Julian Barling, my supervisor not only pushed, prompted and advised but also encouraged me, with his continuous stream of good humour. I can only thank him for making me work and helping me laugh. Both were sources of inspiration, for which I am greatly indebted.

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ABSTRACT

The Stony Brook Scale (SBS) is a behaviour rating scale which is claimed to have the ability to sub-categorize highly selected groups of hyperactive children (O'Leary & Steen, 1982). In particular, it has been suggested that this scale can differentiate between purely hyperactive and aggressive children. The present study sought to replicate the research of O'Leary and Steen (1982) and to extend the application of the Stony Brook Scale to parent ratings of child behaviour. Formerly, only teache atings on the scale had been examined.

Three separate factor analyses were conducted on the following samples:

(a) Teacher ratings on the SBS for a specifically selected sample of hyperactive children (n = 90);

(b) Parent ratings on the SBS for the same specifically selected sample of hyperactive children (n = 86);

(c) Teacher SBS ratings for a heterogeneous sample of schoolchildren (n = 90).

Principal component factor analyses with varimax rotation were computed separately for each group. Teacher SBS ratings for

the selected sample, produced three significant factors: the first was labelled "Aggression" (Eigenvalue = 3,34; 40,9% of variance explained); the second "General" (Eigenvalue = 2,56; 31,4% variance explained), and the third "Hyperactivity" (Eigenvalue 1,59 19,5% variance explained). Parent SBS ratings of the same selected sample also yielded three significant factors: the first was labelled "Aggression" (Eigenvalue = 4,39; 58,1% of variance explained); the second was named "General Anxiety" (Eigenvalue = 1,60; 21,2% of variance explained); and the third "Hyperactivity" (Eigenvalue = 1,02; 13,5% variance explained). Finally, in teacher ratings for the heterogenous sample three significant factors again emerged. The first was labelled "Conduct Disorder" (Eigenvalue = 6,33; 69,8% variance explained); the second was called "Uncoordination-Inattention" (Eigenvalue = 1,71; 18,8% of variance explained), and the third factor was named "Anxiety-Depression" (Eigenvalue = 1,03; 11,3% of variance accounted for).

The factorial structure supports the original claims of O'Leary and Steen (1982). Moreover the applicability of the SBS for use with parent ratings is suggested. Further to this, the independence of emergent factors was evaluated by the correlation of factor scores. These results are discussed with special reference to the implications of these findings for theory and clinical practice.

Overactivity in infancy and childhood has been a source of worry and concern to parents, caretakers and teachers for many centuries (Aries, 1962). In contemporary society overactivity continues to rank high on the list of behaviour problems in children (Dielman, Catell & Lepper, 1971). Negative reactions to such overactivity are commonplace in everyday social situations, in both home and school settings. Parents and teachers seem more likely to view passive behaviour favourably and overactive and inhibited behaviour negatively, possibly because of the control and management problems presented by the latter (Chess, 1960). In recent years both parents and teachers have looked increasingly to medical and psychological professions for help in controlling overactive behaviour in children (Schrag & Divoky, 1975). The help offered has varied from stimulant medication on one hand to psychotherapeutic approaches on the other (Knight & Bakker, 1980).

Professional and academic interest in this area dates back to 1902 when a British pediatrician in a series of lectures presented descriptive accounts of children who were hyperactive (Still, 1902). Since then countless clinicians and researchers have attempted to describe and define hyperactivity. They have produced varied descriptions as well as diverse ways and methods of viewing hyperactive behaviour in children (Ross & Ross, 1976).

Werry (1968) for instance has assumed a wholly quantitative description of hyperactivity. Werry defines hyperactivity (p. 587) as -

> a level of daily motor behaviour which is clearly greater (ideally by more than two standard deviations from the mean) than that occurring in children of similar sex, mental age, socio-economic and cultural background.

While some investigators busied themselves in applying a host of advanced technological apparati to the measurement of motor activity (e.g, Wade & Ellis, 1971), others questioned the validity of a purely quantitative approach to hyperactivity (Fish, 1971). Ross and Ross (1976) have drawn attention to the lack of validity in a purely quantitative approach to hyperactivity on two counts. Firstly, they claim there are no activity level norms for children. Due to considerable intra and inter-individual variations, both across and within situations Ross and Ross (1976) maintain that such activity level norms are unlikely to be established.

The second serious flaw in a quantitative concept of hyperactivity noted by Ross and Ross (1976) pertains to its failure to provide information about the normality of the activity level itself. These authors claim that such information essentially lies in the qualitative aspects of the activity itself. Within a broader quantitative and qualitative framework, descriptions of hyperactivity change fundamentally.

Concern with high activity levels has been substantially moderated by specific attention given to more qualitative factors (McMahon, Reem & Greenberg, 1970). Typically these factors involve examination of: instigating social stimuli; situational appropriateness of the behaviour; goal directedness of behaviour; and responsiveness to discipline (Ross & Ross, 1976).

This review will adopt a quantitative - qualitative framework in its broader understanding of hyperactivity. Such a framework (with equal emphasis on both context and behaviour) provides a more flexible conceptual basis for an integrated description of hyperactivity.

Given this basis, a socio-historical account of academic interest in childhood hyperactivity from Still's (1902) initial publication to current scientific literature is presented. An attempt will be made to illustrate various etiological hypotheses postulated, in relation to hyperactivity. Attention will be drawn to the failure of such hypotheses to generate means of sub-categorizing hyperactive children and the problems this has posed for clinical assessment and diagnosis. Finally, empirical evidence for a descriptive-behavioural methodology for sub-categorizing hyperactive children will be presented. This will provide a theoretical and empirical context within which this research may be viewed.

80 Years of Hyperactivity - an Overview

The accumulation of knowledge about hyperactive children began with a series of lectures published in 'Lancet' by Still (1902). This author presented detailed descriptions of two main categories of hyperactive children.

The first of these groups of children presented with hyperactivity as the result of some physical impairment. Gross brain lesions, diseases, and other conditions producing brain damage were some of the impairments demonstrably linked to hyperactive behaviour. The second group of children also presented with similarly hyperactive behaviour. In this group, however, there was no evidence of brain damage; no impairment of intelligence and no hint of neurological or other physical disease.

Still (1902), reflecting the societal values of his era, diagnosed the latter group of hyperactive children as having "defects in moral control". Ross and Ross (1976) draw a parallel between Still's (1902) children with "defects in moral control" and contemporary children diagnosed as hyperactive. Indeed, descriptive accounts of children with "defect in moral control" seem to justify the comparison.

In Still's (1902) second sample of hyperactive children he noted that the children seemed remarkably resistant to punishment and often the hyperactivity seemed unrelated to the child's training and environment. He further determined that onset of hyperactive behaviour began in the early pre-school years and noted that the incidence was far greater amongst boys than girls.

Etiologically Still (1902) linked hyperactivity to numerous factors including child-rearing and genetic transmission. Despite this he recommended medication in treating hyperactive behaviour, irrespective of the presence or absence of discernible brain damage. It is also noted that at this point, hyperactive children were viewed socially as disobedient and morally defective (Ross & Ross, 1976). During this early part of the century society was only just beginning to acknowledge childhood problems such as retardation, learning difficulties and epilepsy (Safer & Allen, 1976). Social awareness of less severe childhood disorders was at a low ebb.

The view that hyperactive behaviour was linked to demonstrable brain damage was destined to be encouraged by the encephalitis epidemic of 1918 (Ross & Ross, 1976). In describing children who had recovered from encephalitis Strecker and Ebaugh (1924) document a lack of cognitive impairment, but also noted "catastrophic personality changes". Previously normal children

became hyperactive, distractable, antisocial and unmanageable. Studies of behavioural correlates of other brain disorders such as epilepsy, and in particular anoxia during birth, provided further evidence linking hyperactivity to brain damage (Tredgold, 1908).

The question of anoxia raised by Tredgold (1908) was interesting, as it posed the possibility of undemonstrable brain damage. This can be seen as the first step towards the foundation of the concept of minimal brain damage (Doll, Phelps & Melcher, 1926). These authors claimed that in some instances even if brain damage could not be directly observed, it may be presumed present inferentially on the basis of overt behaviour.

Childers (1935) in a particularly articulate paper, however, commented that only a small proportion of hyperactive children appeared to have discernable brain damage. It is thus seen that even at this early stage of professional interest in hyperactivity, etiological disparity and conflict existed. These characteristics seemed to determine the nature of research and debate in this area for many years to come. The Depression and Second World War, however, stifled academic concern in this area until the early nineteen-fifties. There was one notable exception. In a children's home on Rhode Island, Dr. Charles Bradley experimented with the use of stimulant medication (Benzedrine) with young children displaying

behaviour problems (Walker, 1977). Bradley (1937) claimed dramatic behaviour changes as a result of medication, characterised by increased interest in school work, better work habits and a marked reduction in disruptive and antisocial behaviour.

The Post War Period

The disruption of research during this period stopped with the end of the war in the mid nineteen-forties. Once again the emphasis of academic interest in hyperactivity was focused on the relationship between hyperactive behaviour and brain damage (Werry, 1979).

Strauss and Lehtinen (1947) attempted retrospectively to infer a diagnosis of brain damage amongst a group of institutionalised children, on the basis of teacher behaviour ratings. In comparing groups of demonstrably brain injured and unimpaired children these authors identified a number of behaviours that differentiated the two groups (i.e. hyperactivity, distractability and impulsivity). Their etiological conclusions were that the presence of hyperactive behaviour was in itself sufficient to make a diagnosis of brain damage, even in the absence of neurological evidence. The diagnostic status attributed to hyperactivity was now that of a hard neurological sign (Rie & Rie, 1980).

Sarason (1949) provided a comprehensive critique of Strauss and Lehtinen's (1947) research. He criticised their methods of diagnosis, which he claimed were of poor validity and reliability. He also noted biases in their behaviour rating scale. In summary Sarason (1949) discredited both the methodology and conclusions of the Strauss and Lehtinen (1947) study. Sarason (1949) concluded that the hyperactive behaviour described by Strauss and Lehtinen (1947) cannot be said to be the result of brain damage, nor can it be said that such behaviour is characteristic of every brain damaged child.

Despite this, the work of Strauss and Lehtinen received widespread support on both a social and academic level (Ross & Ross, 1976). The special education and teaching methods advocated by Strauss and his colleagues (Strauss & Lehtinen, 1947; Strauss & Kephart, 1955) were widely adopted despite poor empirically established efficacy (Cruikshank, Bentzen, Ratzeburg & Tannhauser, 1961). This tended to reinforce both conceptually and practically the hypothesized link between hyperactivity and brain damage, in the minds of researchers and educators alike (Ross & Ross, 1976).

In an attempt to provide some etiological clarity the Oxford International Study Group on Child Neurology recommended the term minimal brain damage be replaced by minimal brain dysfunction. The rationale behind this change was that brain

damage should not be inferred from behaviour alone (Bax & MacKeith, 1963). This term was later adopted by a United States Public Health Service task force, constituted to investigate terminology and symptomatology in this area (Clements, 1966). Thus, by the mid nineteen-sixties hyperactivity came to be widely associated with a heterogeneous group of children diagnosed minimal brain dysfunction (Ross & Ross, 1976). The euphemistic nature of this term is noted, as is its tendency to perpetuate the link between hyperactivity and underlying organic factors, demonstrable or not (Conrad, 1976).

Whilst clinicians and researchers vigourously debated etiological issues, practical developments in the treatment of hyperactive children began to emerge. Silverman and Lee (1974) document a surge in research and advertising amongst United States drug companies since 1930, reaching a peak in the early nineteen-sixties. Not surprisingly perhaps, at this point a resurgence of interest emerged in the research of Bradley (1937). Researchers at Johns Hopkin University began to publish encouraging articles about the use of stimulant medication with hyperactive children (e.g. Connors & Eisenberg, 1963). By the mid nineteen-sixties stimulant medication in general and methylphenidate (Ritalin) in particular were established as the treatment of choice for hyperactive children (Barkley, 1977). The overall portrait of hyperactivity by the mid nineteen-sixties was one of a brain damage syndrome, to be

primarily treated with stimulant medication, and possibly additionally with educational programmes suggested by Strauss and Kephart (1955). Psychotherapy was not a widely recommended intervention (Ross & Ross, 1976).

On a societal level changes had also occurred. Hyperactivity and minimal brain damage were now familiar terms to both parents and teachers. Government and private agencies geared themselves towards increasing public awareness of what had now become a medical problem that was to reach near epidemic proportions. The former child of "defective morals" was now diagnosed as minimal brain dysfunction (Conrad, 1976; Schrag & Divoky, 1975) and viewed primarily as a medical problem.

1965 Onwards: Proliferation, Change and Confusion

In the current period, research findings and conceptual formulations of hyperactivity have changed quite dramatically (Ross & Ross, 1976).

While continued research on the occurrence of minimal brain damage accumulated (e.g. Towbin, 1970), the concept of minimal brain damage itself came to be seriously questioned (Ross & Ross, 1976). The main thrust of Ross and Ross' (1976) argument refers to the overextended diagnostic nature of minimal brain dysfunction in that it is often still freely inferred

solely from the presence of behavioural symptoms. On a more empirical level, following an extensive survey of relevant research, Dubey (1976) concludes that there is little evidence to support the viewpoint that organic factors play a significant role in the behaviour of hyperactive children.

On the level of treatment changes have also occurred. Certainly while psychopharmacological treatments have had some documented efficacy (Barkley, 1977), they have also been the target of pertinent criticism. Schrag and Divoky (1975) in a penetrating text have critically evaluated some of the social implications of over-prescription of stimulant medication. Further to this, some contemporary researchers view the large majority of drug-effect studies to be methodologically inadequate (e.g. Sroufe, 1975), rendering their results dubious. In addition, it is noted that some advances have occurred in the psychotherapeutic treatment of hyperactivity (e.g. O'Leary & O'Leary, 1980), mainly in the sphere of behaviour therapy. This has led to considerable debate with regard to the treatment of choice for hyperactive children. O'Leary (1980) outlines some key issues in the pharmacotherapy - behaviour therapy debate, and illustrates some positive short term effects of behavioural approaches. O'Leary (1980) concludes, however, that further research is warranted in evaluating the long term efficacy of behavioural treatment, and also the long term effects of medication.

The etiological and treatment models of hyperactive children, which gained widespread acceptance during the early nineteen-sixties have thus been challenged. Both conceptually and empirically hyperactivity has by-and-large become separated from brain damage or dysfunction. What perhaps had previously been seen as a medical problem has more recently been conceptualised as a behaviour problem (Lahey, Hobbs, Kupfer & Delamater, 1979). This has been reflected in the major classification systems of the World Health Organization and American Psychiatric Association (Ross & Ross, 1976). These bodies have respectively classified the complex spectra of behaviour viewed as hyperactive under the terms "hyperkinetic syndrome" and "hyperkinetic reaction of childhood".

These developments have formalized the distinction between hyperactivity and hypothesized organic dysfunction. Further to this they represent a broad agreement with regard to the wider symptomatology of hyperactivity. It however is noted that a causal etiology of hyperactivity has not been forthcoming. What has occurred is a proliferation of research efforts postulating a host of different causes of hyperactive behaviour (Ross and Ross, 1976). These include

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(a) Genetic factors (Cantwell, 1972),

(b) Organic factors (Towbin, 1970),

(c) Psychogenic factors (Bettleheim, 1973), and

(d) Environmental factors (e.g. Feingold, 1973).

In order to evaluate the current etiological status of hyperactivity, this review will briefly examine each of the above-mentioned factors.

Genetic Factors

Numerous studies have illustrated the prevelance of psychopathology amongst parents of hyperactive children (e.g. Satterfield, Cantwell, Lesser & Podosin, 1974). Cantwell (1972), in a systematic research programme, with a matched control group, confirmed a tendency towards psychiatric diagnosis amongst parents of hyperactive children. More specifically, Cantwell (1972) found a greater incidence of alcoholism, hysteria, and sociopathy amongst parents of hyperactive children. These studies lend empirical evidence to the common clinical observation of a high incidence of psychiatric illness amongst the parents and families of hyperactive children. They do not, however, specify the nature of the relationship between hyperactivity and adult psychiatric disorders, nor do they distinguish between environmental and genetic modes of transmitting hyperactivity (Cantwell, 1975; Ross & Ross, 1976).

In order to examine the following hypotheses:

(a) The hyperactive child syndrome is genetically transmitted from generation to generation;

(b) That a genetic relationship exists between the hyperactive child syndrome and psychiatric disorders of adulthood,

Cantwell (1972) and Morris and Stewart (1973) used an adoption study method. In a sample of adopted hyperactive children, these authors conducted systematic psychiatric examinations of both non-biologic parents. They also examined the biological parents in a second group of clinically diagnosed hyperactive children. The results of both these studies are weakened by the fact that the biological and adoptive parents did not share the same hyperactive offspring (Ross & Ross, 1976). Nevertheless, both these studies report

(a) higher prevalence of hyperactivity amongst first and second degree biological relatives as opposed to adoptive relatives; and

(b) evidence to suggest that adult psychopathology, in particular alcoholism, hysteria and sociopathy occurred to a greater extent amongst the biological families of hyperactive children than it did amongst the adoptive families.

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In sum despite methodological difficulties (Cantwell, 1975) these studies tend to support some genetic etiology to hyperactive behaviour. This is further confirmed by some encouraging results from studies comparing monozygotic and dyzygotic twins on incidence of hyperactivity (Omenn, 1973) and activity levels (Willerman, 1973). It is once again noted that a host of methodological problems surround genetic studies of both the adoption type and the twin type (Mittler, 1971); Cantwell, 1975). It nonetheless appears valid to assume that some genetic factors might be implicated in the etiology of hyperactivity. In this regard Cantwell (1975) notes that

> ...if it is likely that there is a genetic component to the syndrome, it is operating in the subgroup of these children or there may be several genetically distinct subgroups. Further genetic investigations should concentrate on various subgroups of hyperactive children - p.102.

Organic Factors

The current status of an etiological link between hyperactivity and minimal brain dysfunction (M.B.D.) has been discussed previously in this study. The conclusions of Dubey (1973) that there is little evidence to support such an etiological link remains. However, some interesting theoretical propositions are elucidated by Wender (1971). It is noted that the thrust of Wender's proposal differs from previous accounts, linking

organic dysfunction and hyperactivity in that it assumes a biochemical rather than a physical nature (Ritvo, 1975).

It is beyond the scope of this study to provide an overview of neurobiochemical research to date. What is possible, however, is to provide an outline of the theoretical formulations proposed by Wender (1971).

Wender proposes a direct link between hyperactive or M.B.D. children (the terms are used interchangeably by Wender, (1971)) and a metabolic imbalance of monoamines. In particular Wender (1971) proposes a deficit in the production of cortical noradrenaline. This is seen to produce a deficiency in the cortical inhibitory abilities, leading to hyperactive or uninhibited behaviour. In this theory, activity level is assumed to be a function of a balance between excitatory and inhibitory systems which are both thought to be monaminergic. Wender (1971) postulates that the low noradrenaline level in the hyperactive child decreases the efficacy of the inhibitory system, leading to over-excitation and hyperactive behaviour.

This theory also addresses the question of the so-called "paradoxical effect" of stimulant drugs, in particular amphetamines, in hyperactive children. Wender (1971) acknowledges that both the inhibitory and excitatory systems are sensitive to amphetamines, which are chemically similar to noradrenaline. How-

over, it is claimed that in hyperactive children the sensitivity level of the inhibitory system is chemically heightened due to the low noradrenalin level. As a result of this the net effect of amphetamine is to boost the inhibitory system, hence reducing behavioural output.

A further notion, which has been inferred from Wender's (1971) proposal, suggests that hyperactive behaviour decreases with maturation. Wender (1971) extrapolates from studies with rats which indicate an increase in monoamine production with maturation. If the same changes occur with human maturation Wender (1971) may have at least provided a hypothesis to explain Bradley and Bakwin's (1957) clinical observation that hyperactivity decreases with age.

Despite the comprehensive picture provided by Wender (1971), little confirmatory empirical evidence has been produced in support of his formulations (Ross & Ross, 1976). Further to this, contradictory empirical evidence has been offered by Shaffer (1975) and Ferguson and Pappus (1979). In relation to Wender's (1971) hypotheses it is noted that not all hyperactive children respond favourably to amphetamines (Barclay, 1979; Fish, 1975) and that not all hyperactive children outgrow their hyperactivity (Maletzky, 1974). Thus at best if Wender's (1971) propositions are accurate they may apply to a small proportion of the heterogeneous population (Fish, 1975) of hyperactive children.

Other biochemical approaches to hyperactivity have appeared in recent years (e.g. Coleman, 1971; Shaywitz, Yager & Klopper, 1976). Despite producing some interesting correlational results, these studies have as yet contributed little towards formulating specific etiological causes (Ferguson & Pappas, 1979).

The overall picture of a biochemical etiology to hyperactivity remains equivocal. As illustrated above, the limited empirical validity of current propositions offers little security in biochemical accounts of hyperactivity. Further to this biochemical etiological causes of hyperactivity are considered to apply only to sub-categories of all children diagnosed hyperactive (Fish, 1975). Despite this the biochemical propositions themselves have failed to produce adequate methods for subgrouping these children (Ferguson & Pappas, 1979). This factor has further weakened the validity of the biochemical approach.

Psychogenic Factors

Noting the pre-paradigmatic nature of psychology as a discipline (Kuhn, 1962), it is not surprising that psychological perspectives on hyperactivity are fragmented along classical theoretical lines. Bettleheim (1973) has postulated on etiological basis for hyperactivity, primarily based on the characteristics of the mother - child relationship. Bettleheim

(1973) draws broadly on the psychoanalytic conceptions of Bowlby (1969). Learning theorists have also postulated etiological accounts of hyperactive behaviour (Ross and Ross, 1976; Zentall, 1975). Typically, such accounts have focused on subtle reinforcement contingencies operating in the child's environment from infancy (Ross & Ross, 1976). Finally, more recently, some psychologists have attempted to understand hyperactivity in systems terms, focusing on interactional themes in the child's family (Hetherington & Martin, 1979; Hartsough & Lambert, 1982).

This review will now turn to a more detailed exposition of each of the above proposed psychogenic factors contributing to hyperactive behaviour.

Psychodynamic Views. Bettleheim (1973) has postulated that certain children are constitutionally predisposed to hyperactivity. As such these children are likely to react with hyperactive behaviour when placed under environmental pressures which exceeed their tolerance. It is Bettleheim's (1973) view that potentially normal infants may become restless and overactive in reaction to an impatient and irritable mother. This may lead to a spiralling of tension and conflict between mother and child. The infant, in response to the mother's irritability, becomes increasingly restless, which in turn exacerbates the mother's impatience and anxiety (Bettleheim, 1973).

With a constant fear of disapproval Bettleheim (1973) sees such a child developing with a poor self concept, unable to conform and adapt to the demands of the school situation.

Some empirical support for Bettleheim's propositions has emerged. Dahlin, Engelsing and Henderson (1975) concur with Bettleheim's (1973) view that the mother can be the source of the hyperactive child's difficulty. In a pilot study designed to modify early mother - child avoidance patterns, through an increase in physical contact Dahlin, Engelsing and Henderson (1975) witnessed an improvement in infants and elementary school childrens' behaviour.

Bettleheim (1973) suggests warmth, acceptance and flexibility on the part of women in the child's environment, as a measure to normalize the child's behaviour. Gelfend (1973) has produced some evidence to support Bettleheim's suggestion. Measuring behavioural performance on an experimental task Gelfend (1973) showed hyperactive children to perform better in the presence of a warm caring mother figure, than in the presence of their own mothers. It is noted however that Gelfend's (1973) hyperactive children were chosen on the basis of a negative mothering situation. Of course, this need not be reflective of all hyperactive children.

While question marks may arise regarding the validity of supporting empirical evidence, Bettleheim's (1973) views have unquestionably contributed to contemporary conceptions of hyperactivity.

Ross and Ross (1976) highlight that Bettleheim's (1973) linkage of parental; affect, attitude and behaviour, to behavioural disturbances in children, has assumed the status of a psychological fact. Whereas Bettleheim (1973) views this relationship as uni-directional Ross and Ross (1976) prefer to conceptualize a bi-directional relationship between the child and his environment. This is a view consistent with current perspectives in child development (Lerner, 1976).

In this sense not only is the environment a determinant of the child's behaviour, but the child's behaviour to some extent may also determine the quality of his social environment. Thus the impatient mother may be as much a reaction to her constitutionally restless child as the child's restlessness is a reaction to maternal impatience and irritability.

This extension of Bettleheim's (1973) views is conceptually significant. While Bettleheim proposes a broad psychodynamic etiological basis for hyperactivity in children, Ross and Ross (1976) fundamentally alter this. With their bi-directional

view Ross and Ross (1976) seem to offer an interactional understanding of hyperactivity which assumes a more descriptive than etiological nature.

This is seen to cast doubts upon the validity of Bettleheim's (1973) uni-directional view, and upon his etiological emphasis on the quality of the child's mothering experience.

Behavioural Perspectives

> There is considerable theoretical and empirical support for the early acquisition of hyperactivity as a function of direct reinforcement or through observational learning processes. (Ross & Ross, 1976 p. 78).

While the above statement by Ross and Ross (1976) certainly has some validity, there nonetheless exists various different arguments, regarding the nature of learned "hyperactive responses".

Zentall (1975) has proposed an Optimal Stimulation Theory of hyperactivity. He claims an increasing body of data to support the suggestion that hyperactive behaviour functions to optimize stimulation. The increase in activity typically associated with the hyperactive child in Zentall's view may well be, an attempt to compensate for insufficient environmental stimulation from infancy. Such behaviour may be reinforcing in two ways. Wasserman, Asch and Snyder (1972) have

shown that increased activity can heighten awareness of environmental stimuli, creating a responsivity to a new source of stimulation. Secondly, increased activity itself may provide a source of behavioural and proprioceptive feedback to the infant (Zentall, 1975).

In these terms hyperactivity is viewed as an adaptive response to inadequate environmental stimulation. However, this adaptability is not permanent.

While an infant is likely to receive social reinforcement for high activity levels from adults and peers, this most probably terminates abruptly at school entry (Ross & Ross, 1976). In the school and pre-school situation, demands for conformity are high amongst both teachers and peers. The hyperactive child is likely to encounter disapproval from both quarters (Ross & Ross, 1976; Campbell & Paulauskas, 1979).

Despite the growing maladaptability of the child's hyperactive behaviour (Zentall, 1975) it remains a highly reinforced response. It is likely that the child in a desperate bid for social reinforcement will use hyperactivity as a means of attaining the approval behaviour produced in the past (Ross & Ross, 1976). This behaviour is likely to be reinforced by negative attention in the classroom situation (Ross & Ross, 1976) and is likely to impede and interfere with the learning process and social integration (Zentall, 1975).

Zentall (1975) draws on various areas of research for supporting empirical evidence (e.g. Pope, 1970; Douglas, 1974). Further to this he weaves his argument carefully into the fabric of more social-learning perspectives such as that of Ross and Ross (1976). The social learning view thus appears to be encompassed by the Optimal Stimulation Theory. This has seemingly provided a more holistic etiological account of childhood hyperactivity, encompassing early environmental aspects and later social implications. It is noted that most of Zentall's (1975) evidence is of an inferential and collateral nature. Zentall (1975) provides little direct diagnostic and empirical validity for his propositions.

Further support for a learned acquisition of hyperactive behaviour is provided by modelling theorists such as Bandura (1969). While it is possible that hyperactivity may be a modelled response based upon parental behaviour (Daniels, 1973; O'Leary & Emery, in print) studies have not excluded genetic transmission as a possibility. Further definitive research thus seems necessary in the clarification of these hypotheses.

Systems Theory Approaches. A third model offering an etiological account of hyperactivity is the social system model (Conrad, 1976). While this is not a purely psychological model, it seems worthy of consideration.

The social system model of hyperactivity (Conrad, 1976) provides a framework to view wider sociological factors in defining and "creating" hyperactive behaviour (Hartsough & Lambert, 1982). This perspective views the role of institutions, primarily the family and the school in labelling a child "hyperactive". This labelling process is seen to be a source of social control (Conrad, 1976). It is noted however, that social systems theory is presented at a broad abstract level. These conceptions have generated little empirical support and as yet have not facilitated an improvement in diagnostic abilities.

In an effort to apply some of these conceptions practically, attempts have been made to view hyperactivity as a consequence of family interactional patterns (Hetherington & Martin, 1979). Hetherington and Martin (1979) quote a core list of disciplinary practices, which are prevalent in families with hyperactive and conduct disordered children. Hartsough and Lambert (1982) attempt to specify some family variables highly associated with hyperactivity. These authors portrayed families of hyperactive children to be comparatively disciplinarian, often viewing their child's academic competence in a pessimistic-negativistic manner. On an index of instability the families of hyperactive children ranked significantly higher than a normal control group (Hartsough & Lambert, 1982). Whether such patterns are the cause of the result of having a hyperactive child still however, remains unclear.

Despite this research no composite picture emerges of the hyperactive child's family as is the case with other childhood disorders e.g. anorexia nervosa (Minuchin Baker, Rosman, Liebman, Milman & Todd, 1975).

Nonetheless, familial and other social aspects of hyperactivity are currently being acknowledged as important to the overall diagnostic picture (Anderson, Williamson & Rushing, 1981). Etiological specificity and further empirical validation of some of the primary suggestions presented here are seen as necessary (Hartsough & Lambert, 1982).

The overall contribution of psychogenic determinants of hyperactive behaviour is clearly both relevant and important. Much literature in this area, however, deals with these aspects in broad and often theoretical terms. The diagnostic evaluation and measurement of specific, empirically validated variables is wanting. Although the heterogeneity of hyperactive children as a group, has been noted (eg Fish, 1975), psychological theory and research has largely failed in delineating etiological different sub-groups of hyperactive children.

This review, in examining possible etiological determinants of hyperactivity will now turn briefly to some factors in the non-social environments of these children. Various hypotheses have emerged linking hyperactivity to factors such as lead

poisoning (Sibbergeld & Goldberg, 1973), dietary factors (Feingold, 1975), and environmental constraints (McNamara, 1972). A more detailed expose of these non-social environmental factors will conclude this study's overview of possible factors etiologically relevant to hyperactivity.

Non-Social Environmental Factors

Lead Poisoning. Ross and Ross (1976) document that the toxic significance of lead has been known for many centuries, and has been long recognized as an industrial disease. In contemporary urban society, environmental exposure to lead is almost inevitable, even for the human fetus (Lin-Fu, 1972). Needleman (1973) has commented that lead poisoning amongst children in the United States has reached almost epidemic proportions.

Ross and Ross (1976) comment that most cases of lead poisoning amongst infant children in the United States occurs from the ingestion of lead based paints. Typically these paints were used thirty to forty years ago, and often still coat the outside walls of deteriorating urban areas. A further source of lead is documented by Ross and Ross (1976). Quoting a 1972 Environmental Protection Agency Report, these authors cite that in the United States over 200 000 tons of lead are emitted annually into the atmosphere as the result of gasoline combustion.

In a recent research programme, David, Clark and Voeller (1972) expose the relevance of the effects of lead on hyperactivity. These authors have established that children with an elevated "body lead burden" may exhibit hyperactive patterns of behaviour - what is extremely pertinent, however, is the claim that the toxicity levels needed to produce such symptoms are well below clinical toxicity levels (David, et al, 1972). As such these children would not readily be identified as "lead poisoned" in general practice.

In a controlled study, David, et al (1972) have elucidated that hyperactive children seem to have significantly higher lead stores than a non-hyperactive control group. Further to this, these authors comment that most of these children had been identified as hyperactive, but due to the subclinical level of their toxicity they had not been considered lead poisoned. Further to this it is noted that David, et al (1972) recognize that lead presence seems to occur more frequently in children with vague etiological reasons for their hyperactivity, than in children who have good etiological causes for their hyperactive behaviour.

Animal studies (Silbergeld & Goldberg, 1973) in which mice were given lead-laced drinking water, have produced results that tend to support David, et al's (1972) hypothesis. Silbergeld and Goldberg (1973) have documented increased activity levels in mice with increased lead levels.

While some of the evidence on lead toxicity amongst hyperactive children seem compelling, it would be premature to assume a direct causal link between lead and hyperactivity. Ross and Ross (1976) comment that it is not yet known whether the involvement of lead in hyperactivity is primary, contributory, or incidental. Should a causal link be firmly established between lead and hyperactive behaviour, it is noted that it would, once again, apply to a subgroup of these children. It would pertain in particular, to those hyperactive children who live in cities, and who have no clear etiological causes for their behaviour. A possible doubt regarding lead pollution as a determinant of hyperactivity is evidenced by Anderson Williamson and Rushing (1981). These authors supply tentative rural statistics, suggesting a comparable level of hyperactivity between populations of rural and urban children. It is clear that further definitive research is necessary before a clear etiological link is drawn between lead toxicity and childhood hyperactivity.

Dietary Factors. The food additive hypothesis originated with the work of Feingold (1973, 1975). From initial clinical observations Feingold (1973) suggested that the chemicals that add colour and flavour to food may be a causative factor in some cases of hyperactivity and learning disabilities. Feingold (1973) noted that these hyperactive reactions did not constitute an allergy in the classical immunological sense, but

seems to be provoked by biochemical mechanisms in the central nervous system. Feingold (1973) isolates "natural salicylates" as the chemico-active componet and identified it as an ingredient in countless food colouring and flavouring additives approved by the United States Food and Drug Administration.

Feingold (1975) reports the results of treating one hundred and ninety four hyperactive children on a diet considered to be free of natural salicylates. Feingold's results are seemingly impressive. Of the one hundred and ninety four children placed on the diet Feingold (1975) claims fifty eight of these children responded dramatically and thirty five responded favourably. Approximately half of these children were able to discontinue medication within ten days of starting the diet (Feingold, 1975).

In describing these changes Feingold (1975a) claims that symptoms of hyperactivity, aggression and impulsivity are seen to decrease rapidly in the early stages following implementation of the diet. Further to this, it is claimed that these children experience improved motor co-ordination manifested in improved drawing and writing, improved speech, loss of clumsiness, and in younger children, specific disturbances of perception and cognition are reportedly improved (Feingold, 1975a).

Beneficial results of a salicylate free diet have been reported

from similar studies conducted in Australia (Cook & Woodhill, 1976). These authors describe symptom changes, almost identical to those portrayed by Feingold (1975; 1975a). Further to this, these authors report a recurrence of hyperactive symptoms upon infractions of the diet.

Despite the above evidence, Ross and Ross (1976) comment on the numerous methodological errors prevalent in Feingold's (1973, 1975) research and in subsequent studies. As such these authors advise that confirming evidence for Feingold's hypotheses be treated with some caution.

In a well planned research programme evaluating Feingold's hypothesis Connors (1980) concludes that Feingold's hypothesis remains unsubstantiated and that his original observations of gross and noticeable behavioural reactions seem irreplicable.

Following an extensive review of current research Harley and Mathews (1980) comment that there is little evidence to warrant the adoption of the Feingold diet as a far reaching therapeutic intervention. These authors draw attention however to some important secondary factors associated with the diet. Harley and Mathews (1980) claim the diet may help parents to reduce their own guilt feelings by identifying outside causative agents for their child's disruptive behaviour. Further to

this it is added that as a result of the diet, essential changes may occur in family dynamics. Parents and children may be encouraged to buy groceries together and share in food preparation in a mutually supportive fashion.

The etiological connection between food preservatives and hyperactive behaviour is currently a hotly debated point in the literature. While some very recent studies (e.g. Swanson & Kingsburne, 1980) illustrate a positive link between hyperactive behaviour and food preservatives, others claim negative results (e.g. Connors, 1980). Methodological debates between those researchers who have produced supporting evidence and those that have not, continues. In the light of the above uncertainty, Harley and Mathews (1980) offer relevant comment. These authors suggest that on the basis of Feingold's original data, plus evidence from more recent research, Feingold's (1975, 1975a) hypotheses apply to a small sub-grouping of hyperactive children. In particular this sub-grouping is seen to consist of younger children. Clearly, more research is needed in this area, and in particular the development of diagnostic criteria for delineating such sub-groups is a vital priority. A usable methodology for this process on the basis of dietary factors is still awaited.

The above review has presented a myriad of possible etiological causes of hyperactivity. These hypotheses have largely been

incapable of generating a sound basis on which different sub-groups of hyperactive children may be distinguished. This has created a diagnostic and prognostic problem for clinicians in this field. This review now turns to a synopsis of current diagnostic policy regarding hyperactivity in childhood and will attempt an evaluation of its validity in terms of recent research.

Diagnostic Classification and Syndrome Status

Given the prevalence and severity of hyperactivity and the controvesy surrounding its etiology and treatment (Loney, 1980; Ross & Ross, 1976), the classification and assessment of hyperactive behaviour becomes most important.

The most recent text for the diagnosis and classification of mental disorders (D.S.M. III-American Psychiatric Association 1980) has made some changes in the diagnostic classification of hyperactivity. Children are diagnosed as Attention Deficit Disorder with Hyperactivity if they are considered inattentive, impulsive and hyperactive, over a variety of situations and over a significant period of time (American Psychiatric Association 1980). (It is to this constellation of symptoms that the term "hyperactive" will refer, as used in this study). This change from the former D.S.M. II Classification, Hyperkinetic reaction of childhood (American Psychiatric

Association 1968) is most probably based largely on the research of Douglas (1972; 1975). In these studies Douglas isolates attention deficits as a primary symptom in hyperactive children.

Despite these changes it is noted that "Attention Deficit Disorder with Hyperactivity" is attributed the status of a clinical syndrome in the Multiaxial diagnostic evaluation (American Psychiatric Association 1980). In this regard the new classification system is comparable to the former system.

Ross and Ross (1976), however, comment that assigning syndrome status to hyperactivity is open to criticism and is not purely semantic. Ross and Ross (1976) continue to outline two necessary requirements for syndrome status. These are "that the symptoms form a unitary cluster, and that they have a common cause, or at least have major etiological factors in common." (p. 9).

These authors comment further that there is no evidence to assume that these clusters of characteristics subsumed under the label hyperactivity (Stewart, Pitts, Craig & Pieruf, 1966) meet either of these requirements. While clusters of characteristics such as hyperactivity, impulsivity and distractability are frequently described, (McMahon, Deem & Greenberg, 1970; Stewart et al, 1966), few attempts have been made to

statistically interrelate these characteristics. Further to this, it is clearly evidenced from the above review of possible etiological determinants that no clarity exists in this regard. In the case of hyperactivity, unlike other childhood disorders, no single or common etiological cause is discernible at this stage (Quay, 1979). Instead what has emerged are fragmented and separate etiological hypotheses which are thought to pertain to specific sub-groups of children diagnosed hyperactive (Fish, 1975; Ross & Ross, 1976). It is probable from this state of affairs that children currently diagnosed hyperactive do not form a homogeneous population. They seem rather to form a heterogeneous population, who, despite displaying a broadly common symptomology, seem to be fragmented etiologically.

Further empirical developments have created more doubts regarding the validity of hyperactivity as a diagnostic category. Sandberg, Rutter and Taylor (1978) have failed to distinguish between hyperactive and conduct disordered children, concluding that a re-examination of the broad concept of hyperactivity (hyperkinesis) is necessary. Other investigations (Sandberg, Wieselberg & Shaffer, 1980) have produced evidence of large correlations, in empirical comparisons of Hyperactivity and Conduct disorders. On the basis of these results, Sandberg et al (1978) and Sandberg et al (1980) have argued that symptoms of overactivity and distractability are commonly associated

with a variety of children's psychiatric disorders, rather than forming a specific syndrome. This point is reiterated by Quay (1979) who suggests abandoning the diagnostic category of hyperactivity, due to its poor construct and discriminative validity. Some methodological aspects regarding these studies seem noteworthy. Firstly, in both the abovementioned studies as well as in a comparable study of Stewart, Cummings, Singer and de Blois (1982), data was obtained from a variety of sources (e.g. Family History, Medical History, Teacher behaviour ratings, Parent behaviour ratings), but nonetheless analyzed in a unitary fashion. Secondly, it is noted whilst Sandberg et al (1978) and Sandberg et al (1980) produced similar results, the former utilized a referred clinic sample, whilst the latter utilized a London school-going sample. More interestingly, however, Taylor and Sandberg (1980), also using a London school population, failed to produce comparable results. In fact it is reported that in this study distinct dimensions of hyperactivity and conduct disorders were clearly apparent. The ambiguous evidence for Sandberg et al's 1978 propositions may sound a note of caution for those researchers who, perhaps too easily, wish to abandon hyperactivity as a diagnostic category (e.g. Quay, 1979). Perhaps a re-evaluation of research methodologies as suggested by Loney (1980) is more appropriate.

Both the literature involving hyperactivity and current diagnostic practice with hyperactive children seem to reveal more

confusion than clarity. This review will now examine some responses to this state of confusion and will present recent evidence and debate in this regard.

Various suggestions have been made regarding future research. Tryphanos (1979) requests a more precise definition of the hyperactivity syndrome in order to procure a research environment in which more meaningful conclusions may be attained. To have etiological relevance a more precise definition of hyperactivity would need to delineate sub-groups of hyperactive children on the basis of demonstrable causes (Fish, 1975; Ross & Ross, 1976). Cantwell (1975) poses the intended direction of research by asking

> How can children with the (hyperactivity) syndrome be divided into meaningful sub-groups, whose conditions differ in etiology prognosis and response to treatment. (p. 203)

However, such research, in producing well-defined homogeneous sub-groups, has not been easily forthcoming. A number of investigators have used a variety of correlational techniques, most notably factor analysis to identify sub-groups of children diagnosed hyperactive (e.g. Routh & Roberts, 1972; Sandberg, et al, 1978). Such research, however, has proved to be largely disappointing, demonstrating little or no relationship between behavioural, cognitive, neurological and medical history

componets (Loney, Langhorne & Paternite, 1978). These variables have not formed meaningful sub-groups along the lines of etiological specifity, distinctive symptomology or response to treatment. Instead they seem to have clustered as a function of the data source (Loney, Langhorne & Paternite, 1978; Homatidis & Konstantareas, 1981). As such these findings are inconsistent with similar multivariate research on child psychopathology. Such research has typically been capable of delineating major categories of behaviour dysfunction (Quay, 1979).

Langhorne, Loney and Paternite and Bechtoldt (1976) conducted a factor analysis of eleven cited core behavioural symptoms in ninety four hyperkinetic boys. These authors once again found that variables tendered to cluster by source rather than by symptor or cause. Loney et al (1978) suggest that this may be considered as a firm empirical indication of heterogeneity in the wider group of children diagnosed hyperactive. Langhorne et al (1976) offer an interesting explanation as to why such variables should cluster by source. These authors speculate that source factors result due to differences in the behaviour of hyperactive children across different environmental settings. This directly postulates an environment-specific theory of hyperactivity. Such a theory could possibly provide explanations for the lack of agreement often found between parents, school and clinic descriptions of the behaviour of a

single child (Loney et al, 1978). This persists despite widespread cross-professional agreement on the basic components of the syndrome (Prinz, Connor & Wilson, 1981).

Over and above this, both empirical and theoretical validity exists for an environment-specific theory of hyperactivity.

Walter Mischel (1968), in his now classic text on personality outlines an environment specific-theory of personality and behaviour. Mischel (1968) finds poor empirical evidence to substantiate both the trait and state conceptions of personality. Mischel (1968) argues

> ...behaviour depends on stimulus situations and is specific to the situation: response patterns even in highly similar situations often fail to be strongly related. Individuals show far less cross-situational consistency than has been assumed by trait-state-theories. The more dissimilar the evoking situation, the less likely they are to lead to similar or consistent responses from the same individual. Even seemingly trivial situational differences may reduce correlations to zero. Response consistency tend to be greatest within the same response mode ..... (p. 177).

Current research in hyperactivity has produced results consistent with Mischel's (1968) expectations. Langhorne et al (1976) has postulated a situation-specific theory in explanation of his ulti-source results. While acknowledging situational differences in hyperactive children, others (e.g. Schacher, Rutter & Smith, 1981) have called for an abandonment

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