Hyperkinetic Impulse Disorder in Children's Behavior Problems

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IT HAS long been recognized and accepted that a persistent disturbance of behavior of a characteristic kind may be noted after severe head injury, epidemic encephalitis and communicable disease encephalopathies, such as measles, in children. It has often been observed that a behavior pattern of a similar nature may be found in children who present no clear-cut history of any of the classical causes mentioned.

This pattern will henceforth be referred to as *hyperkinetic impulse disorder*. In brief summary, hyperactivity is the most striking item. This may be noted from early infancy on or not become prominent until five or six years of age. There are also a short attention span and poor powers of concentration, which are particularly noticeable under school conditions. Variability also is frequent, with the child being described as quite unpredictable and with wide fluctuations in performance. The child is impulsive and does things "on the spur of the moment," without apparent premeditation. Outstandingly also these children seem unable to tolerate any delay in gratification of their needs and demands. They are irritable and explosive, with low frustration tolerance.

Poor school work is frequently quite prominent. The previously described behavioral items in themselves create a pattern which makes it very difficult for the child to participate in the work of a school room. In addition there is often visual-motor difficulty which, combined with the other difficulties described above, makes for poor work in arithmetic and reading. In writing and reading, "reversals" are frequent and the handwriting is often crabbed and irregular.

The salient characteristics of the behavior pattern described are strikingly similar to those in cases with clear-cut organic causation studied by Rosenfeld and Bradley,[1] and in which the present authors have been interested for over 10 years.

They also resemble the behavior pattern described by Frosch and Wortis.[2] On clinical grounds, Frosch and Wortis expressed the belief that this behavior disturbance is in some way related to the diencephalon, perhaps particularly to dysfunction of the hypothalamus. They grouped these behavior patterns under the heading of "the impulse disorders."

It is recognized that none of the items described as characteristic of hyperkinetic impulse disorder is diagnostic and that each of these or any combination of these may also have a purely emotional origin. Hypermotility

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in particular may serve the mechanisms of defense, may have an aggressive and sadistic component, and may have many other dynamic implications. In this paper, however, reference is made to a total symptom complex which, at the time of its origin at least, does not seem to be related to any particular psychological precipitant, though it may have concomitant psychological effects and sequelae. Details of differential diagnosis, psychological test findings, electroencephalographic observation, and so on, will be dealt with in a separate paper.

Experimental Studies

The present paper reports on some experimental studies designed to test the thesis that dysfunction of the diencephalon is in some way connected with the hyperkinetic syndrome of behavior. The studies are based upon the work of Gastaut and his photo-Metrazol technic.[3] Gastaut summarized the potential value of this test as follows: "This is a clinical neurophysiological test which provides a method for the exploration of certain subcortical structures, among which the most

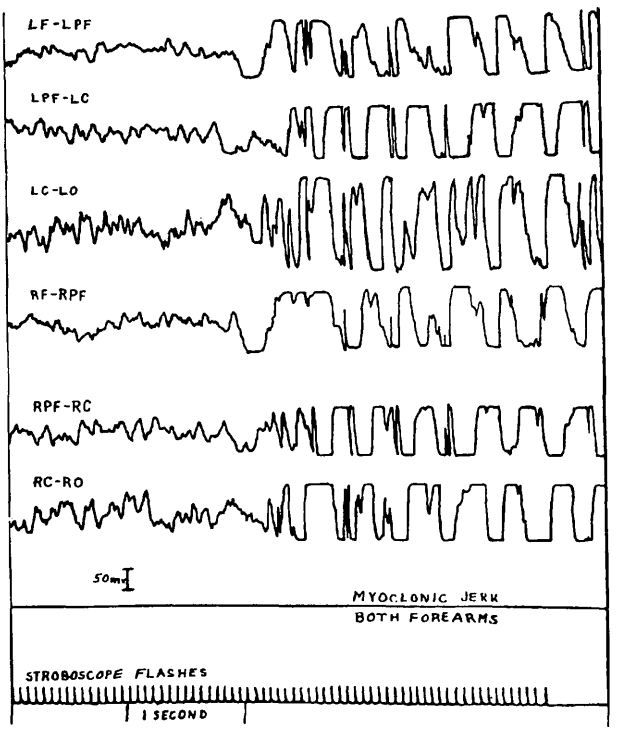

Fig. 1. End point of photo-Metrazol test.

important are those of diencephalon and most especially of the thalamus." The results of this test were given in quantitative form, as the number of milligrams of Metrazol per kilogram of body weight required to obtain a specified type of response clinically and in the EEG when the patient was exposed to the flickering of a stroboscope light within a certain range of frequencies.

Gastaut's work indicated that a normal threshold could be established in adults. That is, normal adults exposed to this procedure require a certain consistent amount of Metrazol to evoke the required end point (an EEG spike-wave burst and a myoclonic jerk of the forearms). In the original paper[3] and in an accompanying one with Hunter,[4] the authors presented evidence to show through both human and animal studies that a photo-Metrazol threshold which was lower than normal indicated damage to or dysfunction of the diencephalon.

Results of Gastaut Procedure

In a previous paper the present authors[5] outlined a technic which standardized the Gastaut procedure for use with children. It was shown that the thresholds obtained were reliable and valid. An EEG end point is shown in Fig. 1. With this procedure the photo-Metrazol threshold in children who did not show hyperkinetic impulse disorder and who ranged in age from seven to twelve years was suggested as 6.5 mg. and above. Thresholds of 5.1 to 6.4 mg. were regarded as borderline abnormal and less than 5.1 as clearly abnormal. The individual children, their diagnoses, and photo-Metrazol thresholds are listed.[5]

TABLE 1. EXPERIMENTAL POPULATION

Patient No.	*Age*	*Diagnosis*	*Hyperkinetic*	*Nonhyperkinetic*	*Threshold*
1	10/0	Passive-aggressive personality with anxiety reaction	..	X	7.1
2	10/0	Passive-aggressive personality with anxiety reaction	..	X	7.0
3	10/6	Emotionally unstable personality		X	6.8
4	8/2	Passive-aggressive personality	X	..	2.8
5	9/3	Emotionally unstable personality, convulsive disorder	X+[a]	..	5.2
6	9/3	Passive-aggressive personality		X	8.1
7	9/3	Passive-aggressive personality	X	..	3.9
8	10/1	Passive-aggressive personality	X	..	1.0
9	8/3	Anxiety reaction	X	..	5.0
10	10/6	Anxiety reaction with schizoid personality	X	..	5.7
11	9/1	Schizophrenic reaction childhood type, chronic brain syndrome, convulsive disorder	X+	..	4.8
12	10/1	Passive-aggressive personality	..	X	7.2
13	7/4	Anxiety reaction with compulsive trends	..	X	6.1
14	9/3	Passive-aggressive personality	X	..	1.9
15	10/2	Schizophrenic reaction, childhood type, convulsive disorder	X+	..	6.6
16	9/5	Passive-aggressive personality with anxiety reaction, convulsive disorder, grand mal and petit mal	X+	..	4.4
17	10/6	Passive-aggressive personality	X	..	4.3
18	8/3	Passive-aggressive personality with anxiety reaction	X	..	4.0
19	9/10	Schizophrenic reaction, childhood type, exotropia, periodic	X	..	1.9
20	8/9	Passive-aggressive personality with anxiety reaction	X	..	5.7
21	10/10	Inadequate personality	X	..	5.5
22	10/8	Passive-aggressive personality	X	..	7.3
23	10/8	Passive-aggressive personality	X	..	3.6
24	10/2	Antisocial reaction	X	..	5.5
25	8/8	Inadequate personality	X	..	6.0
26	4/4	Convulsive disorder; adjustment reaction of childhood, conduct disturbance manifested by aggressive behavior and destructiveness	..	X	6.4

TABLE 1. (*Continued*)

Patient No.	*Age*	*Diagnosis*	*Hyperkinetic*	*Nonhyperkinetic*	*Threshold*
27	10/0	Passive-aggressive personality	X	..	6.5
28	11/4	Convulsive disorder; passive aggressive personality with anxiety reaction and schizoid features	X	..	2.3
29	9/8	Schizophrenic reaction, childhood type	..	X	7.6
30	7/9	Passive-aggressive personality	X	..	3.7
31	9/4	Grand and petit mal; chronic brain syndrome, emotionally unstable and passive-aggressive personality	X+	..	1.8
32	11/9	Passive-aggressive personality	X	..	2.3
33	9/10	Passive-aggressive personality	..	X	5.8
34	8/9	Passive-aggressive personality with anxiety reaction	..	X	6.7
35	9/2	Passive-aggressive personality with anxiety reaction	..	X	7.1
36	9/8	Passive-aggressive personality with anxiety reaction	X	..	6.0
37	10/11	Passive-aggressive personality	..	X	6.9
38	8/1	Anxiety reaction, moderate	..	X	1.8
39	11/2	Obsessive-compulsive reaction	..	X	6.6
40	10/7	Emotionally unstable personality	..	X	7.6
41	8/8	Passive-aggressive personality	X	..	6.1
42	9/10	Passive-aggressive personality with anxiety reaction	X	..	3.7
43	7/8	Passive-aggressive personality	X	..	4.8
44	10/10	Chronic brain syndrome with passive-aggressive personality; convulsive disorder	X+	..	2.5
45	10/9	Childhood schizophrenia	..	X	1.9
46	9/9	Passive-aggressive personality with anxiety reaction	X	..	4.0
47	9/7	Passive-aggressive personality	..	X	6.1
48	11/0	Passive-aggressive personality	..	X	7.5
49	10/0	Anxiety reaction	X	..	7.4
50	10/7	Passive-aggressive personality; mental deficiency; Schizoid personality	X	..	7.1

[a] X+ is hyperkinetic syndrome of behavior with epilepsy.

Photo-Metrazol Thresholds and Clinical Picture

In an expansion of this earlier study, two distinct categories were established from among patients at the Bradley Home, which is a psychiatric hospital for emotionally disturbed children with an age range from five to twelve years. Included among them are children with psychoses, neuroses, and behavior disorders.

From this population, 50 reasonably cooperative children were selected and two different groups were established. These were selected without regard to diagnoses, and solely on the basis of whether they did or did not clinically manifest the total picture of behavior which has been described and categorized as "hyperkinetic impulse disorder."

There are 32 children who presented the symptom picture of hyperkinetic impulse disorder. Of these, 11 (34 per cent) had a clear-cut history of commonly accepted factors capable of causing brain damage, such as head injury, encephalitis, or meningitis early in life.

This compared strikingly with the complete absence of such historical data in the 18 children comprising the group without evidence of hyperkinetic impulse disorder.

Taking this another way, the total number of clear-cut postnatal factors generally regarded as significant etiologically for brain-damage syndromes were calculated for both groups. These factors specifically included convulsions associated with pertussis and/or pneumonia; head injury; encephalitis or meningitis; and early epilepsy of an organic type. There were 24 such factors in the hyperkinetic group of 32 and 7 such factors in the nonhyperkinetic group of 18. For the hyperkinetic group the mean was 0.75 and the standard deviation (S.D.) was 0.51. For the

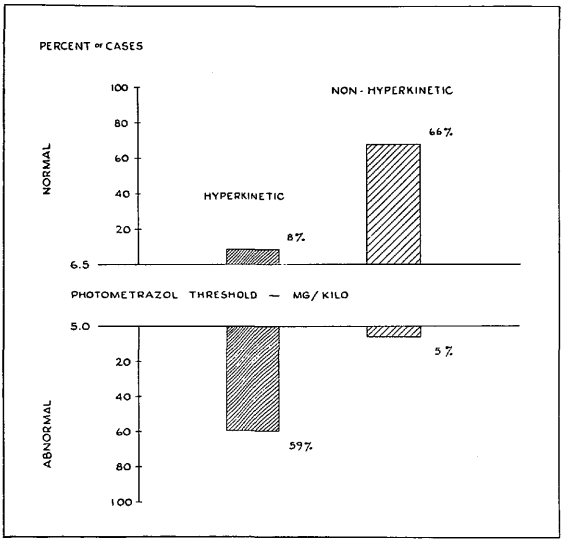

Fig. 2. Correlation of symptom picture with photo-Metrazol thresholds.

TABLE 2. CLINICAL GROUPS CORRELATED WITH HIGH THRESHOLD

Group	Total Number	6.5 mg./kg. and above	
		Number	%
Hyperkinetic	32	6	18
Nonhyperkinetic	18	12	66

TABLE 3. CLINICAL GROUPS CORRELATED WITH LOW THRESHOLD

Group	Total Number	5.0 mg./kg. and below	
		Number	%
Hyperkinetic	32	19	59
Nonhyperkinetic	18	1	5

nonhyperkinetic group the mean was 0.39 and the S.D. was 0.50. This difference between the two groups is statistically significant as shown by $t = 2.43$ and $p < .02$.

The photo-Metrazol thresholds previously recorded for each child were then collected and studied for each of the two groups. The individual children, their diagnoses classification, etc., are listed in Table 1.

In the group of 32 patients who clinically presented the hyperkinetic syndrome, the mean photo-Metrazol threshold was 4.54 mg./kg., with a range from 1.0 to 8.1 and an S.D. of 1.87. In the other group of 18 patients, with a wide range of diagnoses but no clinical evidence of hyperkinetic impulse disorder the mean photo-Metrazol threshold was 6.35 mg./kg. with a range from 1.8 to 8.1 and an S.D. of 0.74. Statistically, the difference between the means for the two groups is highly significant. ($t = 3.43$, $p - < .01$)

If a threshold of 6.5 mg. is taken as the lower limit of normal and 5.0 mg. as the upper limit of abnormal, inspection also indicates a striking difference between the two groups (Tables 2 and 3; Fig. 2).

These results suggest that children with the hyperkinetic impulse disorder, regardless of whether or not their history contains clear-cut evidence of any agent causing injury to the central nervous system, as a group have a photo-Metrazol threshold significantly lower

than do children of comparable age without the syndrome.

Amphetamine and Photo-Metrazol Threshold

Previous work by Bradley had suggested that the amphetamines may have a favorable influence upon behavior disorders in children.[6] Continuing observations suggested that this favorable influence was primarily noted in children presenting the hyperkinetic impulse disorder. For this reason an experiment was executed to determine the effect of amphetamine upon the photo-Metrazol thresholds, previously shown to be low, in children who present clinically the hyperkinetic impulse disorder pattern. A total of 13 subjects from within this previously described group was used, comprising twelve boys and one girl. The age range was eight years and two months to eleven years and two months, average nine years and ten months.

Repeat determinations of threshold were carried out in each of the cases. Seven of the children were tested the first time while receiving medication (that is, either racemic amphetamine, from 10 to 25 mg., or D-amphetamine, from 5 to 15 mg.). The second time medication was discontinued. For the other six children, on the first test medication was withheld, but it was given prior to the time of the second photo-Metrazol determination.

Under these circumstances, the mean of the thresholds for these children who were not receiving amphetamine in one form or the other at the time of the test was 4.8 mg./kg. The mean for the same subjects when receiving amphetamine in clinically efficaceous doses was 6.7 mg. Thus the administration of amphetamine in this group resulted in a rise of the mean photo-Metrazol threshold to the level previously noted as characteristic for the children who do not present this clinical syndrome.

The difference between these two means is statistically significant beyond chance expectancy. ($t = 5.38$, $p < .001$) There was no reliable relationship between the order of testing (whether on medication or off medication for the first test) and the change in threshold.

These findings indicate that the abnormally low threshold in this syndrome is raised by amphetamine and lowered again when amphetamine is removed. An illustrative example is given by the results of testing the child in the following case report.

Case Report

This child was nine years of age when the project began; the hyperkinetic syndrome was quite marked. He was admitted at eight years of age with chief complaints of being hyperactive, destructive, and having short attention span since the age of two to three years. He was the product of a three-day, difficult labor, with difficult delivery and cyanosis of unknown duration after birth. In the course of his hyperactivity he had a number of falls and other traumatic incidents. This accentuated the ambivalence in his mother who had never come to terms with her feelings about her own femininity and with the significant males in her life. The father was a passive, insecure man who was jealous of his own children.

On April 8, 1952, a photo-Metrazol threshold was determined, without medication, and found to be 2.8 mg./kg. This was repeated on May 13, 1952, again without medication, and the threshold was 3.4 mg./kg., both falling within the range of experimental error.

He was then placed on a dose of amphetamine previously found to be efficacious clinically and on June 10, 1952, the determination was repeated. This time his photo-Metrazol threshold showed a tremendous rise, to 7.3 mg./kg. Then, after eight months of successful control of the hyperkinetic syndrome by the same medication, for testing purposes the amphetamine was removed and the test repeated on Februry 4, 1953. At this time (simultaneously with clinical reappearance of the syndrome), the threshold dropped to 3.3 mg., a level essentially identical with that existing prior to the use of medication.

Electroencephalograms persistently showed diffuse, moderately slow activity to a greater extent than normal for the boy's age. On one occasion there were some paroxysmal 4/sec. spike-wave complexes which were generalized.

On admission, a psychological test battery showed a Full Scale I.Q. of 111 on the Wechsler Intelligence Scale for Children, with evidence of a capacity for superior functioning. He was

noted to have conflict around sexual feelings and a fear of harm from the male figure, and anxiety thus aroused was repressed and inhibited. Two and a half years later the entire test battery was repeated. At this time his Full Scale I.Q. was found to be 122. His personality was much more integrated and he was more efficient intellectually. Ego strengths were obviously greater. This was the first time he had actually obtained a superior rating, with the demonstration of an ability to handle abstract concepts.

He received D-amphetamine sulfate in the long-acting form, a total dose of 15 mg. daily, throughout his stay except for repeated periods of removal either for testing or attempts at a clinical trial to see whether the medication was still needed. It was possible to arrange a total of only 30 psychotherapeutic interviews and out of the 30 interviews, 17 had to be devoted to handling his feelings around eye operations required for his strabismus. The striking thing about these interviews was the rapidity of relationship formation, the development of some insight into his hostility, a better acceptance of his masculine identification and good critical judgment. In other words the amphetamine did not interfere with the operation of the psychotherapeutic process and the fostering of a basic inner change.

He has been followed for eight months after his discharge, representing a total of three and a half years on this daily dosage of amphetamine. Even at the present time attempts at removal or even reduction of dosage of the amphetamine are quickly followed by a return of the hyperactivity and short attention span which are immediately noted by teachers and neighbors, though they are not aware of the manipulation in medication.

It is expected that, as with most children seen here, the manifestations of the hyperkinetic syndrome will disappear, with medication no longer needed, by the end of adolescence, at least and often before this. The actual time for a given child, however, cannot be predicted.

Discussion

Experimental facts have been presented to support two points: (a) children with the hyperkinetic impulse disorder type of behavior have a low photo-Metrazol threshold (and therefore possibly some disturbance of function of the diencephalon, regardless of whether their history is indicative of trauma to the central nervous system or not) and (b) that this behavior and the altered photo-Metrazol threshold may both be changed by the use of amphetamine. The photo-Metrazol studies in themselves do not give the explanation for hyperkinesis. It must be recognized that interpretations are largely speculative, as neurophysiological research concerning diencephalic and cortical relations in the human is still in such an amorphous state.

The first problem is why there should be children who present the hyperkinetic impulse disorder without having any of the classic etiologic traumatic or infectious factors in their historical backgrounds. This is a problem not unique to this field, as in cerebral palsy, a known organic central-nervous-system condition, definite clear-cut causative factors are lacking in at least 40 per cent of cases carefully studied.[7] In that field as in this, these cases may be due to inaccurate or incomplete histories, a succession or combination of minor insults, none of which seem significant enough in themselves, minor infections during the mother's third trimester of pregnancy and minor infections in the baby in the first few months of life, and lastly the hazards of normal birth.

Accidents at Delivery

In addition, emphasis must be given the possibility of cerebral hemorrhage occurring during apparently normal vertex deliveries and symptomless at the time of delivery. An explanation for this may come from the thesis of Schwartz.[8] He pointed out that in normal vertex presentations, following the rupture of the fetal membranes, the presenting vertex is exposed to atmospheric pressure, with the remainder of the body subjected to intrauterine pressure. Unlike the normal hemodynamics, this pressure is exerted equally upon the venous and arterial sides of the circulation and upon the pooled blood contained in the placenta, the viscera, etc., with a resulting forcing of the blood into the poorly protected cerebral

vessels. There may be a "water hammer" effect upon the end loops of the capillaries and thus the possibility of perivascular hemorrhages and eventual organization into glial scars. This possibility gains some support from the work of Arey and Dent,[9] showing the frequency of ring-shaped hemorrhages around the terminal vein of the thalamus in newborn infants dying for often inexplicable reasons.

This possibility is perhaps made more likely to occur by the present-day obstetrical tendency to induce labor by rupture of the membranes. This deprives the fetus, from the earliest stage of labor, of the protection given by the amniotic fluid which by Pascal's law provides an equal distribution upon the fetus of intrauterine pressure. As a result, too, the fetal head sustains more pressure in opening the maternal passages, particularly since it does not have the help of the amniotic fluid and membranes acting as a hydrostatic dilator. In our experience this syndrome has been particularly apt to occur in precipitous labors, especially where for one reason or another the head was held back.

Emotional Cause

Another possibility that cannot be dismissed is that there may be a purely emotional cause for the behavior syndrome as described and that in some of these cases the apparent abnormal function of the central nervous system as depicted by the photo-Metrazol test might have been purely secondary to the emotional disturbance. Such a possibility definitely exists, but as yet we have no data on it.

Behavioral Mechanism

The next major question is by just what means or mechanism, trauma to or dysfunction of the central nervous system may bring about the kind of behavior characterized as the hyperkinetic impulse disorder. One striking point is that the characteristics which have been described are to some extent normally found in the course of development of children. That is, as compared with adults, children are hyperkinetic, have a short attention span and poor powers of concentration, and are impulsive (ruled by the "pleasure principle"). In the course of their development they outgrow this mode of behavior and actually, in the course of time, so do most of the children with the hyperkinetic syndrome. Therefore, somehow the processes of maturation must be involved in this. This may be connected with the interrelationship between the diencephalon (which is probably essentially fully formed and in full functional integrity at birth) and the cortex, which is by no means fully formed at birth but continues to grow at least beyond the fifth year of life. One set of theories has suggested a reverberating feedback type of circuit between cortex and diencephalon. This postulates that as the cortex grows in mass it is eventually able to exert greater influence and allow for the more organized, localized and discriminating responses characteristic of growth, and for the ability to inhibit which is one characteristic of maturation. Other theories have stressed the role of the diencephalon as the rostral component of the reticular activating system of Magoun[10] and its possible role as a first-stage sorting, routing, and patterning mechanism for impulses coming in from sensory receptors to the various higher levels of the central neuraxis.[11]

Postulated Theory

At the present time, the most satisfactory, though admittedly fragmentary hypothesis is based upon the concept that stimuli, constantly coming in from sensory and visceral receptors pass through the diencephalon on the way to cortical areas and that the diencephalon serves to pattern, route and give valence to these stimuli. It is postulated that injury to or dysfunction of diencephalon would alter resistance at synapses. This would allow incoming impulses to spread out of usual pathways and irradiate large cortical areas.

Freud[12] and subsequent investigators have emphasized that in the first few months after birth the newborn infant operates under a homeostatic principle which induces great discomfort when any tension accumulates, with a great need to discharge this tension and return to the previously undisturbed state. It is

our concept that underlying the hyperkinetic syndrome is a dysfunction of the diencephalon which by a mechanism as described could make the individual unusually sensitive to stimuli flooding in from both peripheral receptors and viscera. The possibility of such a mechanism is supported by a recent study. Watson and Denny-Brown[13] have recently advanced a hypothesis that there can be a breakdown of synaptic coordination channels associated with an instability of synaptic resistance (to transmission of impulses across them) due to diffuse neuronal disease, particularly in subcortical structures (with especial emphasis upon the thalamus). This could well make a young infant respond with greater than usual urgency to what might otherwise be regarded as a normal amount of tension coming from the usual organic sources, such as hunger, from a full bladder, etc. As a result, these infants are often hypertonic, querulous, and irritable.

Psychological Difficulties

Thus, in an infant with the hyperkinetic syndrome, a perfectly normal amount of mothering may be inadequate. Many new mothers have deep concern about their worth and adequacy as females, wives and mothers. An infant behaving the way just described may to them seem to offer clear proof of their unfitness to be mothers, leading to further tension in the mother and increasingly frantic efforts to mold the child into a more acceptable pattern. As this fails, the development of unconscious hostility toward the child, to which in turn he responds, may set off a train of secondary emotional disturbance. Of course this is even more likely to occur if there have been pathogenic factors in the mother's or father's emotional development, their relationship to each other, and other important areas.

Caplan[14] has pointed out:

> Motility which is unduly delayed or precipitous or which, after being delayed, becomes markedly hyperkinetic and uncontrolled, results in an anxiety in the child and most certainly an anxiety in the parents which must be dealt with early. . . . Parents react to the psychic aggression revealed in the motor impulse by counter-aggression and we have another example of the vicious cycle of "snowballing" anxiety with reinforcement of pathology in the primary motor defect.

In addition, the various components of the hyperkinetic syndrome later in the child's development make it hard for him to fit into any scheme of family activity and it is difficult to provide for him satisfactorily. Also the impulsive and explosive responses which these children have are particularly intolerable to many parents who find it difficult to accept from their children any behavior which has an aggressive aspect. In this connection it has been interesting to observe that a number of mothers who themselves have had a strong drive toward both masculinity and dominance have seemed to derive great satisfaction from their hyperkinetic infants, though the same amount of hyperkinetic activity seemed to cause great distress to other mothers.

In the crowded classrooms of today, the teacher often becomes hostile toward a child who, despite seemingly good intelligence, can not sit still, can not keep his mind on his work, hardly ever finishes the assigned task and yet unpredictably may turn in a perfect paper. Added to this, the specific learning difficulties, particularly in reading and arithmetic, combine with the child's increasing unhappiness to worsen the situation. The child frequently fails to gain a proper foundation for the fundamentals of schooling so that each successive year he falls further behind.

To these stresses are added the general unhappy situation at home, with more and more open expression of their feelings by parents who can not understand why they should have been afflicted with so difficult a child. The net result is extreme unhappiness in the child with a marked ego disturbance, whose image of himself is certainly a distorted and unfavorable one and who in turn may react with almost any kind of acting-out behavior, running the gamut of the so-called habit or conduct disturbances. While the resulting symptom picture is generally not of a neurotic type, the presence of the hyperkinetic syndrome does not offer any immunity against the development of neuroses. It is possible

that the presence of the hyperkinetic syndrome and associated ego disturbances may make the individual more susceptible to emotional stresses and perhaps thus to the development of a neurosis. It is perfectly possible to have a hyperkinetic syndrome and a neurotic state in the same individual.

Frosch and Wortis[2] and Greenacre[15] have also offered stimulating and closely reasoned hypotheses as to how organically determined hyperkinesis may mold the personality structure. Bellak has recently suggested[16] that childhood schizophrenia can best be understood in terms of very early injury to or defect of the ego from a number of possible causes, including unrecognized brain damage. While the hypothesis here described fits very neatly into his scheme, the number of childhood schizophrenics studied with this method are too few and the results too equivocal for any definite statement as yet.

Action of the Amphetamines

The final major remaining question is that of the mode of action of the amphetamines in this picture. Marrazzi[17] has reported that amphetamine inhibits or lowers the level of synaptic transmission at all synapses studied, including the lateral geniculate. Bradley of Birmingham[18] has presented evidence from work on cats to suggest that "the site of action of amphetamine may be related to the brain stem reticular activating system of Magoun."[10] These observations, together with our own, suggest that amphetamine may in some way, perhaps by raising the level of synaptic resistance, alter the functions of the diencephalon in such a way that it once more can keep the cortex from being flooded by streams of unmodulated impulses coming in through sensory receptors, stimulating the reticular activating system, etc. In this regard it is of interest that the children with the hyperkinetic syndrome give the impression of constant and excessive activity on the part of the reticular activating system; that Thorazine, which has been reported[19, 20, 21] as having a favorable effect upon this syndrome, is thought to have its locus of action in the reticular activating formation of Magoun.[22] Freud [23] has spoken of the *Reizschutz*, or protective barrier, which is differentiated from the Ego and serves to protect it from being overwhelmed by sensory stimuli beyond its power to assimilate. What has been described here may be an example of a defective *Reizschutz* altered favorably by pharmacological means. He also said, prophetically:

> The future may teach us how to exercise direct influence, by means of particular chemical substance, upon the amounts of energy and their distribution in the apparatus of the mind. It may be that there are other undreamt of possibilities of therapy.[24]

In contradiction to the "all or none" type of thinking prevalent until recently, this work has suggested the need for continual consideration of both neurophysiological and psychological factors. Clinically it has been seen that when the hyperkinetic syndrome exists alone and has been recognized early, there may be only minimal associated emotional disturbance. However, even in these cases—although the favorable effect of the amphetamine upon the behavior often induces an era of good behavior which has its own beneficial effect—a definite part of the treatment consists of work with the parents, who frequently have feelings of guilt and also hostility. If there is something of an organic factor existing, which has played a significant role, and it can be demonstrated to the parents, in some cases it may relieve to some extent their feelings of guilt so that they can be more readily worked with.

In the majority of cases there are, by the time the child is seen, psychological difficulties of greater magnitude and persistence. In these cases there is a definite need for psychotherapy for the child and work directly with the parents, and it is important not to allow the parents to place sole emphasis upon an organic factor as a means of dismissing any responsibility for making changes in the situation. It is also possible that symptomatic control of behavior and relief of anxiety by medication, by its "magical" effect, may interfere with the course of psychotherapy and may also make the child feel that he has no re-

sponsibility for his conduct and no difficulties which need further work. Parenthetically, there have been a number of cases where psychotherapy was indicated but was unable to proceed unless the child was receiving amphetamine and enabled to settle down enough to participate in the therapeutic process.

The illustrative case previously cited indicates that amphetamine may prove clinically efficacious without either interfering with the psychotherapy, with the maturation of the child's own inner controls, or with his ability to learn. This is shown quite strikingly by the comparative psychological test findings given as well as by the clinical results observed.

It is necessary to emphasize that the hyperkinetic syndrome is a very specific entity. It does not account for the majority of children's behavior disturbances, and differential diagnosis continues to be just as much needed as heretofore. Disturbed behavior in children should not be treated indiscriminately with amphetamine, chlorpromazine, or reserpine, and children who need specific psychiatric care should not be deprived of it because of knowledge of existence of this syndrome. Moreover, hyperactivity as such may reflect an emotional disturbance entirely and not anything of an organic nature. Nevertheless, the entity characterized as the hyperkinetic impulse disorder does seem to exist, and where it is found amphetamine and some of the newer medications such as chlorpromazine may be helpful for it. No significant adverse physical effect has been seen from use of amphetamine for up to five years.

Summary

A very common cause of children's behavior disorder disturbance is an entity described as the *hyperkinetic impulse disorder*. This is characterized by hyperactivity; short attention span and poor powers of concentration; irritability; impulsiveness; variability; and poor school work. The existence of this complex may lead to many psychological problems, due to the extremely irritating effect it has upon parents and teachers.

Often the history reveals some clear-cut organic insult to the central nervous system, before birth, during birth, or during the first five years of life, which presumably may result in dysfunction of the diencephalon. In many cases, however, though clinically they seem identical, the history shows no such factor. Such dysfunction of the diencephalon exposes the cortex to unusually intense storms of stimuli from peripheral receptors coming through the diencephalon and the reticular activating system and may interfere with the function of the cortex and its relationships with diencephalon. The photo-Metrazol determination done in children showing this syndrome is significantly different from those without the syndrome.

This situation is in time overcome by the operation of normal maturational processes. Until such takes place, apparently amphetamine and possibly other medications have a very specific ameliorating effect upon this syndrome by means of a direct action upon the diencephalon. This suggestion is supported by the data given on the effect of amphetamine in raising the photo-Metrazol threshold in such children.

This situation has many psychological and psychodynamic implications.

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AAAS Socio-Psychological Prize

Through the generosity of an anonymous donor, the American Association for the Advancement of Science offers an annual prize of $1000 for a meritorious essay in socio-psychological inquiry. The conditions of competition for the prize to be awarded at the 1956 annual meeting, New York, December 26–31, have been published in *Science* for March 30, 1956 (Vol. 123).