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Recent Evidence of Physical Abnormality in Children with Learning Disabilities

The idea that the difficulties of "learning-disabled" children have a physical explanation rather than a purely psychological one has been in prolonged abeyance, but has recently experienced a powerful resurgence. Pihl reviews recent findings from a number of fields in physiology, and adds to them an account of his own discovery that these disabilities appear to be strongly related to the presence in the body of above-normal levels of certain elements — not only lead, but also lithium and others. These developments point to the clear prospect of our developing forms of intervention by nutrition and other physical means that may bring success where purely educational interventions have faced apparently intractable problems.

While definition remains elusive — baffling the bureaucrats, confounding researchers, and mystifying diagnoses — explanatory models of learning disabilities proliferate. A veteran model now in the midst of a revival is the physical abnormality position. Simply put, the basis for a learning disability is seen by this position as a malfunctioning of some aspect of the nervous system. This condition can be the result of damage, slow development, and/or biochemical abnormality. Early writers in the area of learning disabilities (Orton, 1937; Strauss & Lehtinen, 1947; Johnson & Myklebust, 1965) were quick to postulate and assume this general position. Evidence to support their theorizing was drawn primarily from observations concerning the high incidence among learning disabled children of complicated pregnancies, birth trauma, viral encephalitis, and head injury with loss of consciousness. Of course, the obvious importance of particular brain areas to behaviour and learning, which became apparent from research in physiological psychology and clinical work with brain damaged patients, did not go unnoticed by these writers.

However, belief in physical etiology, although popular, languished until relatively recently for a number of reasons. First, incontrovertible neurological evidence was not immediately forthcoming to support the position. Consequent-

ly, such tautological postulates as the idea of minimal brain damage came and went, leaving an aftertaste of suspicion among the non-converted. Second. viable neurological interventions failed to develop. Obtuse and theoretically ill-conceived notions, such as the specifics of the brain disorganization thesis of the Doman-Delacato viewpoint, achieved some notoriety, mostly negative. Fundamentally, interventions with learning disabled children remained within the schools, without thought of affecting any neurological mechanisms. Finally, a plethora of factor analytic studies appeared in the literature and demonstrated an absence of symptomatic commonality among learning disabled children, thus suggesting the existence of a highly diverse etiology. Consequently, a form of definitional depression overcame researchers, who were enamoured of the need for descriptive specificity.

Cart before the horse?

To advocate explanatory models before some definitional agreement has been achieved does not only seem paradoxical but is also doomed to produce confusion instead of enlightenment. Unfortunately, even when legislated (by the U.S. Education for All Handicapped Children Act, 1975), the definition-first approach has respectively raised in various individuals apathy, antipathy, and apoplexy. The exceedingly broad characterization of a learning disability as an academic performance problem fundamentally unrelated to intellectual, motivational, or emotional deficits will often produce a consensual nod. However, because this is a definition by exclusion, the diagnostic difficulties with this basic definition are considerable, and the outcome is a selection of individuals who hardly form a homogeneous grouping.

The difficulties are two-fold. First, the measurement of what are intellectual, motivational, and emotional deficits varies at present from the precarious to the impossible. Motivational and emotional problems abound in learning-disabled children, seemingly as a concomitant of their learning disorder, although cause and effect have to be intuited and are not measured. Nevertheless, one could argue with good support that at present the most beneficial treatment for these children is that which is specifically aimed at dealing with the motivational and emotional problems. Indeed, a number of approaches amd programs are available to the would-be therapist (Pihl, 1975; Pihl, Parkes & Stevens, 1979).

Second, complex learning is an exceedingly multifaceted event, poorly understood and involving a large number of necessary conditions. There are probably many forms of learning, and it is wrong to speak of a single process. Consequently, the failure of factor analytic studies to show commonality among the characteristics of groups of these children is understandable.

A strategy of obvious heuristic value, and having considerable definitional and treatment potential, is to begin with theoretical notions of etiology and of the existence of separate populations based on measures specific to those no-

tions. Thus, on the presumption of a physical basis for learning disorders, brain dysfunction may be postulated from electroencephalographic, neuropsychological and chemical measures, and from personal history. Each of these areas will now be reviewed here, for it is in these areas that the data for the rebirth of the physical model are incubating.

Neurometrics

The measurement of brain-wave activity in children with learning problems using the electroencephalogram (EEG) is not new. A flurry of interest in the early 1960s in the technique for diagnosing learning-disabled, behavioral, hyperactive, and intellectual deficiency problems quickly evolved into critical reviews (Ellingson, 1966; Woody, 1967). Ideologically, the isomorphism between the electrical activity of the brain and its cognitive activity, the two products of brain activity, was and is to be questioned. That rather significant empirical question aside, fundamental technological and procedural problems seem to have existed, which are presently being surmounted. The methodology called "neurometrics" is meeting many of the early criticisms. This procedure involves the use of mathematical techniques to identify differential profiles of brain activity by identifying groupings of data points in multidimensional spaces. Actually, the procedure is more complex than just a sophisticated computer analysis of results. A computer is also used to run the procedure for the collecting of the data. Consequently, sufficient amounts of artifact-free data are available for analysis. The neurometric test battery is also complex, involving recording from 57 electrode combinations under a wide variety of conditions so that average evoked responses can be related to various sensory, perceptual and cognitive activities.

The seminal and perhaps even definitive work in this area, was completed by John and his associates (1977). In an extensive research program, these investigators have discriminated learning-disabled fron normal children more successfully with neurometric measures than with psychometric measures. In fact, John and others (1977) demonstrated that the typical psychometric measures, near and dear to the school and clinical psychologist, actually account for only five percent of the variance between groups when corrections are made for socioeconomic status, culture, age, and intelligence. A further finding of this group of researchers was that learning-disabled children thought to be homogeneous are in fact neurometrically heterogeneous. Many distinct patterns were found which occurred with the learning-disabled subjects but not with the normal subjects. For example, many learning-disabled children displayed in a number of regions of the brain dysfunction which varied with age, showing one form of abnormal pattern characteristic of pre-twelve year olds and others for older children. Finally, in another study it was determined that children with defective language but normal arithmetic skills and children with deficits in both these areas reflected left-hemisphere abnormalities, while children with defective arithmetic but normal language skills displayed right-hemisphere abnormalities.

In addition to the diagnostic work with learning-disabled children reflected in neurometric studies, recent research relating EEG responses to short-term memory (Chapman, McCrary & Chapman, 1978) and sensory discrimination (Ritter, Simon, Vaughan & Friedman, 1979) have been reported. These types of findings suggest that localization and specification of cognitive functioning is a distinct possibility. Methodological problems still remain — for example, performance-related factors are perhaps being measured and interpreted as cognitive events — but the research die is cast. The foundry for that die has been certain preparatory studies in the area of neuropsychology.

Neuropsychology

Understanding the relationship between brain and behaviour has been the raison d'être of neuropsychology. Typically the performances on psychological tests of individuals with substantiated brain dysfuntion are assessed. In the case of children, two notable studies present in the literature (Klonoff, Robinson & Thompson, 1969; Reitan, 1974) clearly reflect the deleterious effect of brain disorders on sensory-motor, intellectual, and academic tasks. Somewhat similar results have been found by Tsushima and Towne (1977), who investigated children with assumed but not demonstrated brain disorders. These children included those with histories of perinatal disorders, high fever illnesses including febrile convulsions, and acute convulsions. Particularly noteworthy were deficits in motor and visual-motor abilities.

"Learning disability" by usual definition excludes children with verifiable brain injury. Unfortunately for this definition, as we saw in the case of neurometrics, sophistication in measurement technique is now beginning to verify brain disorders in these children. The work of Rourke (1975, 1976) is exemplary in carrying the neuropsychological approach to learning-disabled children: in summarizing his own research, dealing with the question of whether some deficits typically seen in learning-disabled children are the result of cerebral impairment, Rourke (1976) concludes that "cerebral dysfunction is at least one crucial factor which can limit the satisfactory adaptation of children with learning disabilities" (p. 90). This type of conclusion is derived from the distinctive performance often produced by learning-disabled children on psychological measures previously demonstrated to be related to specific cortical functioning.

While studies such as Rourke's have dealt primarily with clinical populations, information resulting from studies concerned with hemispheric involvement in learning and performance *per se* have also generated enthusiasm for the physical model. The functional asymmetry of the cerebral hemispheres, with language skills basically in the dominant hemisphere and visual-imaginal, spatial, and musical skills in the subdominant hemisphere, has been demonstrated in "split brain" treatments and experiments (Sperry, 1974), and in numerous perceptual and memory experiments (Corballis & Beale, 1976). Predicated on these studies, models of learning disabilities have evolved. Sechzer

(1977), for example, proposes that a lack of functional neuronal processes exists in these children, showing abnormalities much like those seen in split-brain kittens. Similarly, data exist which suggest that development of a left-right sense results from maturation in the asymmetry of the brain, and that those discordant with the left-brained bias of Western education find themselves predictably learning-disabled. Witelson (1977), for example, presents data which support the notion that developmental dyslexia results from a lack of appropriate asymmetry of functioning between spatial and linguistic functions.

Individual health histories and behavioral teratology

Early proselytizers for the physical model of learning disabilities relied heavily upon pointing to relationships between medical problems during early development and subsequent learning problems. These initial observations, which were tempered by negative evidence (Minde, Webb & Sykes, 1968), have been reaffirmed by recent investigations, although the cause-effect relationship is more complex than originally thought. Wright (1978), for example, suggests that in the case of sequelae from childhood meningitis, factors such as occurrence and controllability of seizures, the length of illness, age at illness, and concentration of bacteria in the cerebrospinal fluid are all predictive. In a similar vein Gottfried (1973) illustrates how inconsistent findings relative to the effect of perinatal anoxia result from methodological differences and from the failure to account for age at the time of assessment of effect. In another study, Towbin (1978) clearly illustrates how the sequelae of perinatal anoxia are related to age at time of exposure, and suggests that the severity of anoxia with learning-disabled children reflects minor neurologic abnormalities. Finally, in a longitudinal study which followed the development of children who showed such neurological dysfunction in the newborn period, Francis-Williams (1976) reports that at ages eight to nine and a half, of 42 index children 11 were nonreaders and 13 additional children were at least one year or more behind in reading. Comparatively, of 43 control children none were nonreaders and only 5 were one year or more behind their grade level. Only 9 index children were reading at or above their age level.

The study of the large and growing number of substances which *in utero* or at birth affect the future behavioral development of the individual is called behavioral teratology. We are tragically well acquainted with the ramifications that thalidomide had when taken by some pregnant women. Lead and other heavy metals have been implicated as having similar negative effects. More recently added to the list of potential teratogens are narcotics, alcohol, tobacco, obstetrical medication, and synthetic sex hormones, et cetera. The list in fact is growing with such rapidity and is becoming so general that it will soon rival the U.S. Federal Drug Administration's list of carcinogenic substances.

A relatively well-researched situation is the case of maternal narcotic addiction. Offspring of these women often display a drug-abstinence syndrome consisting of irritability, tremors, and disturbed sleep. Significantly, even after

medical attention has been completed, it has been observed (Kolata, 1978) that these children display a pattern of behaviour much like the hyperactivity syndrome, and consequently it is difficult if not impossible to place them in stable foster homes. Interestingly, teratogens can also act through the male. In a number of studies (also in Kolata, 1978) male animals fed diets containing thalidomide, lead, narcotics, alcohol, and caffeine produced significantly more physically and behaviorally deficient offspring than did control animals. While some of these results are explained by alterations in the male genetic material, it is the case that the female already pregnant can also be affected, as some drugs are excreted in the semen and can and do cross the placenta barrier.

It is logical to think of teratogenic effects as occurring on a continuum from mild to severe. Perhaps with time and more research we shall identify initially undetected sources of damage. One line of research which is finding teratogenic effects consistent with the problems seen in learning-disabled children is the study of the effects on children's behaviour of obstetric medication given to women during delivery. Brackbill and Broman (quoted in Kolata, 1978), in a well-controlled study, have found changes in behaviour up to seven years of age as a result of obstetric medication. Obviously, effects are drug- and dose-related, but what are affected most are cognitive and motor abilities. Specifically, developmental milestones are reached late, the children tend to be impulsive, and language skills lag.

Mineral elements in the body

Certain chemical elements, particularly lead, have been demonstrated to be teratogenic, and others are currently under suspicion. Elements comprise five percent of the molecular composition of our bodies and serve a wide range of functions. They may be crucial for life, or necessary for good health, or unwanted and toxic. Additionally, there are many elements of hypothesized yet undemonstrated value or detriment. In spite of the obvious significance of elements to our physical survival, with the possible exception of lead the relative paucity of research dealing with the effects of element levels on behaviour is noteworthy.

Certain initial studies that we have completed in this area suggest a significant role for mineral elements in learning and behaviour. In one study (Pihl & Parkes, 1977) a group of third and fourth grade learning-disabled children were compared to non-learning-disabled controls matched for age, grade, sex, and socioéconomic level. Table 1 presents the mean parts per million of fourteen elements for these two groups, as well as the results of analyses of variance of individual elements across groups. The relative elevations in the levels of the toxic elements of lead and cadmium, and the low level of lithium, are noteworthy and consistent with our own and some other studies.

While the neurological consequences of high dosages of lead are well known, the subtle effects of low dosages suggested above have only recently

Table 1

MEAN ELEMENT SCORES FOR TWO GROUPS OF CHILDREN, AND ANALYSIS OF VARIANCE FOR EACH ELEMENT ACROSS GROUPS.

Element	Learning-disabled (ppm)	Control (ppm)	F ratio	Level of significance
Calcium	397	344	1.75	N.S.
Magnesium	35	37	0.28	N.S.
Potassium	1359	1240	0.21	N.S.
Sodium	1637	919	5.10	P< .02
Cadmium	1.72	1.08	84.52	P< .001
Cobalt	0.16	0.23	35.00	P< .001
Copper	12	17	0.72	N.S.
Iron	23	22	0.82	N.S.
Lead	23	4	28.32	P< .001
Manganese	0.83	0.58	15.21	P< .001
Zinc	139	140	0.10	N.S.
Chromium	0.25	0.09	8.49	P< .01
Lithium	0.22	0.40	7.29	P< .01
Mercury	14	15	0.52	N.S.

ppm: parts per million
N.S.: not significant
d.f. = 1,47
(From Pihl & Parkes 1977)

been reported. Perino and Ernhart (1973), David (1974), and Needleman and others (1979) all report studies supporting this view. Perino and Ernhart found that blood-lead levels of three and six year old children who showed no indications of acute lead intoxication correlated negatively with measures of cognitive, verbal, and perceptual-motor abilities. David has demonstrated how body-lead levels were significantly higher in hyperactive children than in control children, and was able in another study (David, Hoffman, Sverd, Clark & Voeller, 1976) to diminish the symptoms of hyperactivity by the use of lead-chelating medications. Needleman and others (1979) compared children with high and low dentine lead levels on an intelligence scale, a teacher's questionnaire, learning disability tests, and a large number of control variables. The children studied came essentially from the same background and were selected from all the children in the first and second grade in two suburban school systems, and thus the impetus of the study was to discern the effect of low levels of lead. The results showed that the relatively high lead group did significantly more poorly than the control group on the IQ test and all subtests of the Wechsler Intelligence Scale for Children; on three measures of auditory and verbal processing; and on most teacher ratings. These children not only had more trouble learning, but were seen as more distractable, more impulsive, more frustrated, and functioning at a lower level than the children with low dentine lead level.

The learning-disabled children in the Pihl and Parkes (1977) study also were assessed with teacher, parent, and self-ratings of behaviour. Table 2 presents the

significant correlations between chemical elements and the teacher ratings on the Myklebust Pupil Rating Scale. For the total score on this scale as well as for each of the five subtests there is a marked consistency of significant negative correlations between the scale and the elements of lead, cadmium, sodium, manganese, and potassium. Significant positive correlations were determined for lithium, cobalt, and copper. Parents' ratings on the Connors Scale (Connors, 1970) essentially presented a random scattering of significant findings and are uninterpretable, with the exception that the three correlations computed between the element iron and the factor perfectionism were significant. An artifactual explanation for this finding might involve the speculation that compulsive parents produce compulsive offspring and are also likely to provide their children religiously with dietary supplements, hence the high iron score.

Table 2

SIGNIFICANT CORRELATIONS (P • .05) BETWEEN MYKLEBUST PUPIL RATING SCALE AND ELEMENTS, FOR LEARNING-DISABLED AND NORMAL CHILDREN (N = 46)

	Total Score	Auditory Comprehension	Spoken Language	Orientation	Behaviour	Motor
CA						
MG						
K	—.24	—.29	—.24	—.33		
NA	—.41	—.45	—.39	—.40	—.26	—.25
CD	—.65	—.62	—.47	—.49	—.65	—.38
CO	.47	.49	.34	.46	.38	.28
CU	.30	.25			.33	
FE						
PB	—.60	—.53	—.36	—.47	—.49	—.39
MN	—.40	—.42	—.24	—.30	—.38	
ZN						
CR	—.25	—.29				
LI	.60	.61	.49	.47	.51	.36
HG						

The self-rating scale was the Piers-Harris (1969), the results of which were significant in showing a relationship between high cadmium levels and the child's self-rating as one who displays bad behaviour, cries easily, is often afraid, is unhappy, and dislikes himself or herself.

Two additional groups were analyzed in order to compare them to the element levels found in the learning-disabled and normal groups. These groups were 31 institutionalized, mentally-retarded children and 9 of the siblings of the learning-disabled children. Elements were determined by hair analysis, which

has been the procedure we have used throughout our studies (Strain, Pories, Flynn & Hill, 1972; Maugh, 1978). We found these four groups could be separated from each other with 97.5% accuracy using a discriminant function analysis with the elements cadmium, cobalt, manganese, chromium, and lithium. Siblings were like their learning-disabled brothers and sisters in being higher in cadmium (although lower than learning-disabled) and lower in cobalt than the other groups, but that was the only similarity. The retarded children were significantly higher than all groups in calcium, perhaps reflecting an institutional milk-rich diet, and they were also like the learning-disabled children in being relatively high in lead.

Table 3

CORRELATIONS OF OTHER ELEMENTS WITH CADMIUM IN TWO GROUPS OF CHILDREN

Element	Toxicology Literature	Learning-disabled and Mentally retarded	Normal and Siblings
Zinc	Neg.	—.64	+.15
Copper	Neg.	—.42	+.49
Calcium	Neg.	—.54	+.14
Iron	Neg.	—.20	+.53
Lithium	—	—.62	+.29
Cobalt	—	—.26	—.68
Chromium	—	—.53	+.89

The interrelationships between elements were examined for these four groups, as it is well known that elements interact and consequently the absolute level of an element may not be as important as the interaction. Table 3 presents for cadmium only the significant correlations with other elements for the learning-disabled and retarded children and for the normal and sibling controls, as well as a prediction from the toxicology literature concerning the direction of the correlation. What of course is obvious and notable in this table, and reflects our findings in general in this area, is that the relationships among elements for the learning-disabled and retarded correspond to predictions one would produce from the toxicology literature, while the opposite is true for the normals and siblings. This dramatic discrepancy may mean that what we have tended to view as the normal relationship between some elements, as derived from toxicological research, probably represents a special if not abnormal situation. Pertinent to our interests, this special situation is reflected in children who have learning and behavioral problems.

Approximately two years after the results presented in Table 1 had been obtained, both learning-disabled and normal subjects who could be located were again assessed for element levels. Previous significant differences did not reoccur, and application of the discriminant function analysis failed to separate the

Table 4

MEAN ELEMENT SCORES, LEARNING-DISABLED AND CONTROLS, FOR 1975 & 1977

	Learning-disabled		Control	
	1975	1977	1975	1977
Cadmium	1.72	1.01	1.08	0.98
Lead	23	5	4	6
Lithium	0.22	0.68	0.40	0.49

Significant decrease in Cadmium
Significant decrease in Lead
Significant increase in Lithium

two populations. Table 4 presents changes occurring in cadmium, lead, and lithium levels in these two groups which may be explanatory. One can see in this table that the learning-disabled children at follow-up sampling were like the normal children in cadmium and lead, but now were significantly higher in lithium level. Lithium, of course, is the treatment of choice in mania, and there are a number of reports of it being used therapeutically with marked success with disturbed children. It is also noteworthy that all parents of the learning-disabled children had received medical and nutritional counselling, although no controls were maintained on the level of compliance. Learning-disabled children had also received support from a child modulator, or advocate, and significant improvements in their behaviour were recorded (Pihl, Parkes & Stevens, 1979). Additionally, many of these subjects had entered adolescence, a time when primary learning disorders are thought by many to wane. Perhaps some natural process occurs at this stage, reversing previous element abnormalities.

We have recently completed an attempt to replicate the Pihl and Parkes (1977) finding with a new sample of children obtained in exactly the same fashion. With this new sample the same discriminant function analysis was only 66% accurate. Nevertheless, significant correlations again occurred for cadmium and lead on ratings of behaviour and learning. Additionally, an extensive battery of psychological tests and tasks was administered to these children and it was apparent that magnesium, calcium, and zinc were positively and highly correlated with tests of associate learning, memory, and skill at generalization, and with performance on IQ verbal subtests. Negative correlations occurred for cadmium and lead with the above measures as well as with IQ performance subtests.

While the perfunctory observation that "more research is required in this area" is clearly true, it is also true that the obvious and well-supported significance of elements for physical functioning seems to hold for learning and behaviour.

Nutrition

One way that the levels of elements in the body may be controlled is

through nutrition. Increasingly, it is becoming apparent that the adage "we are what we eat" is true of some children with learning and behaviour problems. This statement pertains not only to the need for consuming the right balance of elements and for avoiding an excessive intake of sugar, and to the behavioral concomitants of hyper- and hypoglycemia; but it also applies to the allergic reactions which have been postulated as occurring in these children.

While three percent of adults have specific food allergies, the figure for children is much higher, between ten and fifteen percent. It is often notable how, given a chronic allergy, one's physical, psychological and educational disposition is deleteriously affected. Indeed, there is growing evidence that learning-disabled children have a disproportionately high number of specific allergies (Baldwin, Kettler & Ramsay, 1968; Trites, Ferguson & Tryphonas, 1979). These reactions are not only to common food stuffs but also to the chemicals that are being added to our food at a seemingly frantic pace. Feingold's (1975) model of food-coloring-produced hyperactivity has resulted in a growing number of believers and in the generation of active investigation. The supposed definitive study in this area (Harley & Matthews, 1979) has proven to be less than definitive, and present investigations seem bogged down around the mundane but unresolved question of dose. When straight elimination diet studies are used, changes in behaviour are often noted. Unfortunately, these same changes often occur when a placebo diet is used. Consequently, testimonial claims of 50-85% success need to be devalued to a more realistic 20% (Connors, 1979).

Conclusions

Considering the recent findings in neurometrics, neuropsychology, individual health histories, behavioral teratology, mineral elements, and nutrition which are discussed in this paper, the revival of the physical model from its latency period is understandable. It would seem that the model will only grow in persuasiveness and win converts as evidence accumulates and physical interventions develop. Presently, within the model, treatments such as pharmacological and diet supplement and control predominate. However, not much imagination is required to speculate that the near future will allow for more direct manipulation using more pertinent chemicals and perhaps even direct electric stimulation of prescribed brain areas. Blind alleys, of course, abound, with perhaps the most recent being the tempestuous affair with methylphenadate as a specific treatment for hyperactivity. The fact that we are dominated by a biochemical *zeitgeist* can obviously have both positive and negative ramifications.

While it must always be remembered, particularly by medical practitioners, that the physical model and physical treatment can never ignore social and educational positions and approaches, what the work reviewed in this paper significantly demonstrates is that education is not the exclusive purview of the educator. The physical state of the individual is a most important, if not crucial, factor in learning and learning problems.

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