Ethology and Stress Diseases

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Many of us have been surprised at the unconventional decision of the Nobel Foundation to award this year's prize for Physiology or Medicine to three men who had until recently been regarded as "mere animal watchers." Since at least Konrad Lorenz and I could not really be described as physiologists, we must conclude that our *scientia amabilis* is now being acknowledged as an integral part of the eminently practical field of medicine. It is for this reason that I have decided to discuss today two concrete examples of how the old method (*1*) of "watching and wondering" about behavior (which incidentally we reviewed rather than invented) can indeed contribute to the relief of human suffering, in particular of suffering caused by stress. It seems to me fitting to do this in a city already renowned for important work on psychosocial stress and psychosomatic diseases (*2*).

My first example concerns some new facts and views on the nature of what is now widely called early childhood autism. This is a set of behavioral aberrations which Leo Kanner first described in 1943 (*3*). To us, that is, my wife Elisabeth and me, it looks as if this set of aberrations is actually on the increase in a number of Western or Westernized societies. From the description of autistic behavior, or Kanner's syndrome (*4*), it is clear, even to those who have not themselves seen these unfortunate children, how crippling this affliction is. In various degrees of severity, it involves, among other things, a total withdrawal from the environment; a failure to acquire, or a regression of, overt speech; a serious lagging behind in the acquisition of numerous other skills; obsessive preoccupation with a limited number of objects; the performance of seemingly senseless and stereotyped movements; and an electroencephalogram (EEG) pattern that indicates high overall arousal. A number of autists recover (some of them spontaneously), but many others end up in mental hospitals, where they are then often diagnosed and treated as schizophrenics.

In spite of a growing volume of research on the subject (*5*), opinions of medical experts on how to recognize autism, on its causation, and therefore on the best treatment vary widely. Let me consider this briefly, point by point.

1) There is disagreement already at the level of diagnosis and labeling. For instance, Rimland compared the diagnoses for 445 children, as given by the doctor who was consulted first, with a "second opinion" (*6*). If the art of diagnosis has any objective basis, there should be a positive correlation between first and second opinions. In fact, as Rimland points out, there is not a trace of such a correlation—the diagnoses are practically random (Table 1). What these doctors have been saying to the parents is little more than, "You are quite right; there is something wrong with your child."

And yet, if we use the term autism in the descriptive sense of "Kanner's syndrome," it does name a relatively well-defined cluster of aberrations.

2) The disagreement about the causation of autism is no less striking. It expresses itself at two levels. First, there is the usual nature-nurture controversy. The majority of experts who have written on autism holds that it is due either to a genetic defect or to equally irreparable "organic" abnormalities, for instance, brain damage such as can be incurred during a difficult delivery. Some of the specialists are certainly emphatic in their assertion that autism is "not caused by the personalities of the parents, nor by their child-rearing practices" (*7*). If this were true, the outlook for a real cure for such children would of course be bleak, for the best one could hope for would be an amelioration of their suffering. But there are also a few experts who are inclined to ascribe at least some cases of autism to damaging environmental causes, either traumatizing events in early childhood or a sustained failure in the parent-infant interaction (*8*). If this were even partially correct, the prospect for a real cure would, of course, be brighter.

The confusion about causation is also evident in the disagreement about the questions of what is primary in the overall syndrome, what is at the root of the trouble, and what are mere symptoms. Some authors hold that autism is primarily either a cognitive or (as is often mentioned in one breath) a speech defect (*9*). Others consider the hyperarousal as primary (*10*). Those who subscribe to the environmental hypothesis think either in terms of too much overall input (*11*), or in terms of failures in the processes of affiliation and of subsequent socialization (*8*).

3) In view of all this it is no wonder that therapies, which are often based on views concerning causation, also differ very widely. Nor is it easy to judge the success rates of any of these therapies, for the numbers of children treated by any individual therapist or institution are small; also, the descriptions of the treatments are inevitably incomplete and often vague. Unless one observes the therapist in action, it is not really possible to judge what he has actually been doing.

In short, as O'Gorman put it not long ago (*4*, p. 124), ". . . our efforts in the past have been largely empirical, and largely ineffectual."

In view of all this uncertainty, any assistance from outside the field of psychiatry could be of value. And it is such assistance that my wife and I have recently tried to offer (*12*). Very soon our work led us to conclusions which went against the majority opinion, and we formulated proposals about therapies which, with few exceptions, had not so far been tried out. And I can already say that, where these treat-

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ments have been applied, they are leading to quite promising results, and we feel that we begin to see a glimmer of hope.

Before giving my arguments for this optimistic prognosis, let me describe how and why we became involved. Our interest in autistic children, aroused initially by what little we had seen of the work that was being done in the Park Hospital in Oxford, remained dormant for a long time. But when, in 1970, we read the statement by Drs. John and Corinne Hutt that ". . . apart from gaze aversion of the face, all other components of the social encounters of these autistic children are those shown by normal nonautistic children . . ." (*13*, p. 147), we suddenly sat up, because we knew from many years of child watching that normal children quite often show all the elements of Kanner's syndrome.

Thinking this over we remembered the commonsense but sound warning of Medawar, namely, that "it is not informative to study variations of behaviour unless we know beforehand the norm from which the variants depart" (*14*, p. 109), and we realized that these words had not really been heeded by psychiatrists. In their literature we had found very little about normal children that could serve as a basis for comparison.

We also realized that, since so many autists do not speak (and are often quite wrongly considered not to understand speech either), a better insight into their illness would have to be based on the study of their nonverbal behavior. And it is just in this sphere that we could apply some of the methods that had already proved their value in studies of animal behavior (*15*).

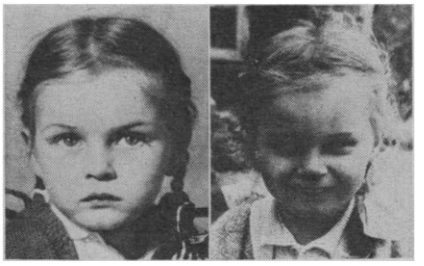

Fig. 1. Two photographs of a girl (aged 6 years) taken in the same spring: (left) taken by a school photographer; (right) taken by her elder sister. These illustrate some nonverbal expressions as used in motivational analysis. [From (*12*); courtesy of Verlag Paul Parey, Berlin, Supplement 10 to *Journal of Comparative Ethology*]

Therefore we began to compare our knowledge of the nonverbal behavior that normal children show only occasionally, with that of true autists, which we had not only found described in the literature but also began to observe more closely at firsthand.

The types of behavior to which we soon turned our attention included such things as the child's keeping its distance from a strange person or situation, details of its facial expressions, its bodily stance, and its consistent avoidance of making eye contact—an extremely rich set of expressions that are all correlated with overt avoidance (Figs. 1 and 2). The work of professional child ethologists is beginning to show us how immensely rich and subtle the repertoire is of such nonverbal expressions (*16*).

But, apart from observing these behaviors themselves, we also collected evidence about the circumstances in which normal children reverted to bouts of autistic behavior. What emerged from this dual approach was quite clear. Such passing attacks of autistic behavior appear in a normal child when it finds itself in a situation that creates a conflict between two incompatible motivations. On the one hand, the situation evokes fear (a tendency to withdraw, physically and mentally); on the other hand, it also elicits social, and often exploratory, behavior, but the fear prevents the child from venturing out into the world. And, not unexpectedly, it is naturally timid children (by nature or by nurture, or both) that show this conflict behavior more readily than more resilient, confident children do. But my point is that they all respond to the environment.

Once we had arrived at this interpretation, we tested it in some simple experiments. In fact, we realized that in our years of interaction with children we had already been experimenting a great deal. Such experiments had not been aiming at the elicitation of autistic behavior, but rather at its opposite: its elimination. As we have written before, each of these experiments was in reality a subtly modulated series of experiments. For a description of what we actually did, I quote from our original publication. We wrote (*12*, pp. 29–30):

> What we invariably do when visiting, or [are] being visited by a family with young children is, after a very brief friendly glance, [to] ignore the child(ren) completely, at the same time eliciting, during our early conversations, friendly responses from the parent(s). One can see a great deal of the behaviour of the child out of the corner of one's eye, and can monitor a surprising amount of the behaviour that reveals the child's state. Usually such a child will start by simply looking intently at the stranger, studying him guardedly. One may already at this stage judge it safe to now and then look briefly at the child and assess more ac-

Table 1. Comparison of first and second opinions about 445 children showing severe behavior disorders. [From Rimland (*6*)]

First opinion	Second opinion								
	Autistic	Infantile autism or early infantile autism	Childhood schizophrenia	Emotionally disturbed or mentally ill	Brain or neurological damage	Retarded	Psychotic	Deaf or partly deaf	Total
Autistic	**33**	5	53	18	23	51	10	7	200
Infantile autism or early infantile autism	1	**10**	6	0	4	6	0	2	29
Childhood schizophrenia	17	3	**1**	2	8	1	0	0	32
Emotionally disturbed or mentally ill	12	2	4	**2**	9	13	3	0	45
Brain or neurological damage	14	3	2	5	**4**	15	0	1	44
Retarded	21	2	6	18	16	**5**	2	2	72
Psychotic	4	0	1	1	2	2	**0**	0	10
Deaf or partly deaf	4	1	0	2	0	5	1	**0**	13
Total	106	26	73	48	66	98	16	12	445

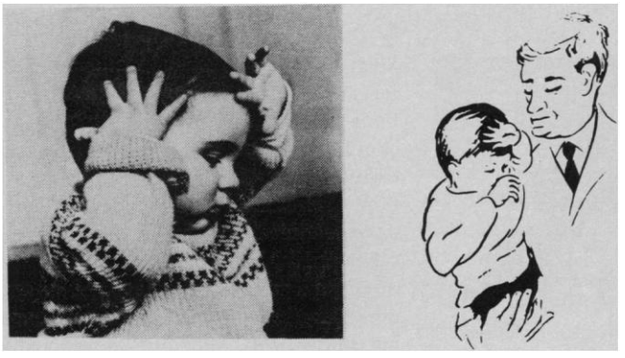

Fig. 2. An example of temporary and permanent autistic behavior. (Left) Typical slight rejection by a 12-month-old normal boy, photographed in his own home in the presence of his mother, who was smiling at him, and faced by him, from a distance of approximately 4 meters. The photographer, who was his (rarely met) grandfather, was approximately 1½ meters away from the child. [From (*12*); courtesy of Verlag Paul Parey, Berlin, Supplement 10 to *Journal of Comparative Ethology*] (Right) Response of an autistic child to repeated attempts of adult to make eye-to-eye contact (drawn from 8-mm motion picture film) [From (*13*); courtesy of Charles C Thomas, Publisher]

curately the state it is in. If, on doing so, one sees the child avert its glance, eye contact must at once be broken off. Very soon the child will stop studying one. It will approach gingerly, and it will soon reveal its strong bonding tendency by touching one—for instance by putting its hand tentatively on one's knee. This is often a crucial moment: one must *not* respond by looking at the child (which may set it back considerably) but by cautiously touching the child's hand with one's own. Again, playing this "game" by if necessary stopping, or going one step back in the process, according to the child's response, one can soon give a mildly reassuring signal *by touch*, for instance by gently pressing its hand, or by touching it quickly, and withdrawing again. If, as is often the case, the child laughs at this, one can laugh oneself, but still without looking at the child. Soon it will become more daring, and the continuation of contact by touch and by indirect vocalisation, will begin to cement a bond. One can then switch to the first, tentative eye contact. This again must be done with caution, and step by step; certainly with a smile, and for brief moments at first. We find that first covering one's face with one's hands, then turning towards the child (perhaps saying "where's Andrew?" or whatever the child's name) and then briefly showing one's eyes and covering them up at once, is very likely to elicit a smile, or even a laugh. For this, incidentally, a child often takes the initiative [see e.g. Stroh and Buick (*11*)]. Very soon the child will then begin to solicit this; it will rapidly tolerate increasingly long periods of direct eye contact and join one. If this is played further, with continuous awareness of and adjustments to slight reverses to a more negative attitude, one will soon find the child literally clamouring for intense play contact. Throughout this process the vast variety of expressions of the child must be *understood* in order to monitor it correctly, and one must oneself *apply* an equally large repertoire in order to give, at any moment, the best signal. The "bag of tricks" one has to have at one's disposal must be used to the full, and the "trick" selected must whenever possible be adjusted to the child's individual tastes. Once established, the bond can be maintained by surprisingly slight signals; a child coming to show proudly a drawing it has made is often completely happy with just a "how nice dear" and will then return to its own play. Even simpler vocal contacts can work; analogous to the vocal contact calls of birds (which the famous Swedish writer Selma Lagerloef correctly described in "Nils Holgersson" as, "I am here, where are you?") many children develop an individual contact call, to which one has merely to answer in the same language.

The results of this procedure have been found to be surprisingly rapid, and also consistent *if one adjusts oneself to the monitoring results*. Different children may require different starting levels, and different tempos of stepping-up. One may even have to start by staying away from the child's favourite room. It is also of great significance how familiar to the child the physical environment is. Many children take more than one day; with such it is important to remember that one has to start at a lower level in the morning than where one left off the previous evening. We have the impression that the process is on the whole completed sooner if one continually holds back until one senses the child longing for a more intense contact.

With all these experiences with normal children in mind, we began to reconsider the evidence about permanently autistic children—again using our own observations as well as the reports we found in the literature. And two things became clear almost at once. Neither for genetic abnormalities nor for gross brain damage was there any convincing, direct evidence; all we found were inferences, or arguments that do not hold water.

The main argument for a genetic abnormality is the statement (and one hears it time and again) "these children have been odd from birth." And we also found that, for various reasons, neither the specialists nor the parents are very willing to consider environmental influences. But in view of what we know about the effects of nongenetic agents that act in utero, of which the recently indicated effect of rubella contracted by pregnant women is only one (*17*), the "odd-from-birth" argument is of course irrelevant. And at least two cases are known of identical twins of whom only one developed Kanner's syndrome (*18*).

Equally unconvincing are the arguments in favor of gross brain damage, and this idea too is based mainly on inference.

On the other hand, the body of positive evidence that points to environmental causes is growing. For instance, many workers report that the incidence of autism is not random. Relatively many autists are firstborn children (*19*). There is also a pretty widespread conviction that the parents of autists are somehow different; for instance, many of them are very serious people, or people who are themselves under some sort of strain. And to a trained observer it is also very obvious that autists respond to conditions, which to them are frightening or intrusive, by an intensification of all their symptoms. Conversely, we have tried out our "taming procedure," as described for normal children, on some severely autistic children, and succeeded in drawing them out of their shells, in making them snuggle up to us, and even in making them join us in, for instance, touch games. I cannot possibly go into all the evidence, but there are several good indications: first, that many autists are potentially normal children, whose affiliation and subsequent socialization processes have gone wrong in one way or another; and second, this can often be traced back to something in the early environment—on occasion a frightening accident, but most often something in the behavior of the parents, in particular the mothers. Let me hasten to add that in saying this we are not blaming these unfortunate parents. Very often they seem to have been either simply inexperienced (hence perhaps the high incidence among firstborns); or overapprehensive; or overefficient and intrusive; or, perhaps most often, they are people who are themselves under stress. For this and many other reasons, the parents of autists deserve

as much compassion, and may be as much in need of help, as the autists themselves.

Now, if we are only partially right in assuming that at least a large proportion of autists are victims of some kind of environmental stress, whose basic trouble is of an emotional nature, then one would expect that those therapies that aim at reducing anxiety, by allowing spontaneous socialization and exploration whenever it occurs, would be more successful than those that aim at the teaching of specific skills. Unfortunately, as I have already said, it is hardly possible to judge from published reports what treatment has actually been applied. For instance, one speech therapist may behave rather intrusively and turn a child into a mere "trained monkey," leaving all the other symptoms as they were, or even making them worse. Another speech therapist may have success simply by having proceeded in a very gentle, motherly way. One has to go by those instances where one has either been involved oneself or where one knows pretty precisely how the therapist has in fact proceeded. It is with this in mind that I will now mention briefly three examples of treatments that seem to hold great promise.

First, even before we published our first paper, the Australian therapist Helen Clancy had been treating autistic children and their families along lines that are very similar to, and in fact are more sophisticated than, those recommended by us in 1972.

The gist of Clancy's method is as follows (*8*). (i) Since she considers the restoration of initially defective affiliation with the mother as the first goal of treatment of autism, she treats both mother and child, and the family as well. She does this by provoking in the mother an increase in maternal, protective behavior. (ii) She uses a form of operant conditioning for speeding up the child's response to this change in the mother. In other words, she tries to elicit a mutual emotional bond between mother and child, and refrains, at least at first, from the piecemeal teaching of particular skills.

With those mothers who were willing to cooperate, Clancy has achieved highly encouraging success, although of course a few families (4 out of approximately 50 treated over a period of 14 years) have failed to benefit.

Second, after the first public discussion of our work, my wife received invitations to visit some schools for autists and to observe what was being done. She found in one of them, a small day school which already had an impressive record of recoveries, that the treatment was likewise aimed at the restoration of emotional security, and teaching as such, including some gentle speech therapy, was never started until a child had reached a socially positive attitude. Much to our dismay, this school has since been incorporated into a school for maladjusted children—the experiment has been discontinued.

Third, a regional psychiatrist invited us a year ago to act as advisers in a fascinating experiment which she too had begun well before she had heard of our work. Three boys, who are now 9, 9½, and 11½ years of age, and who had all been professionally diagnosed as severely autistic, are now being gently integrated into a normal primary school. This involves a part-time home tutor for each boy, a sympathetic headmaster, and willingness of the parents to cooperate. The results are already little short of spectacular. In fact, a specialist on autistic children who visited the school recently said to us: "Had the records not shown that these three children were still severely autistic a couple of years ago, I would not now believe it." This experiment, which is also run along lines that are consistent with our ideas, is being carefully documented.

It is this type of evidence, together with that provided by a number of already published case histories (*20*), that has by now convinced us that many autists can attain a full recovery, if only we act on the assumption that they have been traumatized rather than genetically or organically damaged. I cannot go into further details here, but I can sum up in a few sentences the gist of what the ethological approach to early childhood autism has produced so far.

1) There are strong indications that many autists suffer primarily from an emotional disturbance, from a form of anxiety neurosis, which prevents or retards normal affiliation and subsequent socialization, and this in its turn hampers or suppresses the development of overt speech, of reading, of exploration, and of other learning processes, based on these three behaviors.

2) More often than has so far been assumed these aberrations are not due to either genetic abnormalities or to gross brain damage, but to early environmental influences. The majority of autists, as well as their parents, seem to be genuine victims of environmental stress. And our work on normal children has convinced us not only that this type of stress disease is actually on the increase in Western and Westernized countries, but also that very many children must be regarded as semi-autistic, and even more as being seriously at risk.

3) Those therapies that aim at the reduction of anxiety and at a restarting of proper socialization seem to be far more effective than, for instance, speech therapy per se and enforced social instruction, which seem to be at best symptom treatments, and to have only limited success. Time and again treatment at the emotional level has produced an explosive emergence of speech and other skills.

If I now try to assess the implications of what I have said, I feel at the same time alarmed and hopeful. We are alarmed because we found this corner of psychiatry in a state of disarray, and because we discovered that many of the established experts—doctors, teachers, and therapists—are so little open to new ideas and even facts. Another cause for alarm is our conviction that the officially recognized autists are only a fraction of a much larger number of children who obviously suffer to some degree from this form of social stress.

We feel hopeful because attempts at curing such children at the emotional level, while still in the experimental stage, are already leading to positive results. And another encouraging sign is that, among the young psychiatrists, we have found many who are sympathetic to our views, or even share them, and have begun to act on them.

In the interest of these thousands of unfortunate children we appeal to all concerned to give the stress view of autism at least the benefit of the doubt, and to try out the forms of therapy that I have mentioned.

My second example of the usefulness of an ethological approach to medicine has quite a different history. It concerns the work of a very remarkable man, the late F. M. Alexander (*21*). His research started some 50 years before the revival of ethology, for which we are now being honored, yet his procedure was very similar to modern observational methods, and we believe that his achievements and those of his pupils deserve close attention.

Alexander, who was born in 1869 in Tasmania, became at an early age a "reciter of dramatic and humorous pieces." Very soon he developed serious vocal trouble and he came very near to losing

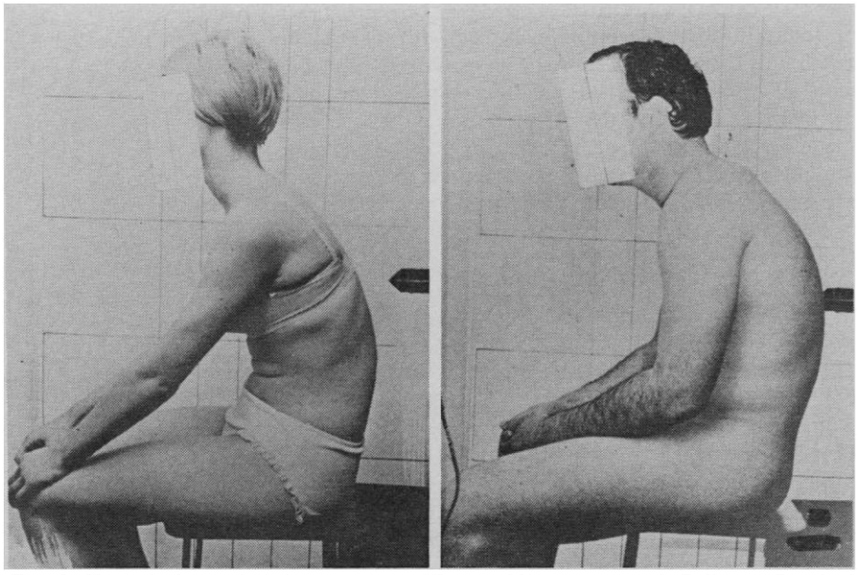

Fig. 3. Typical slumped sitting positions. [From (*23*); courtesy of Gollancz, London]

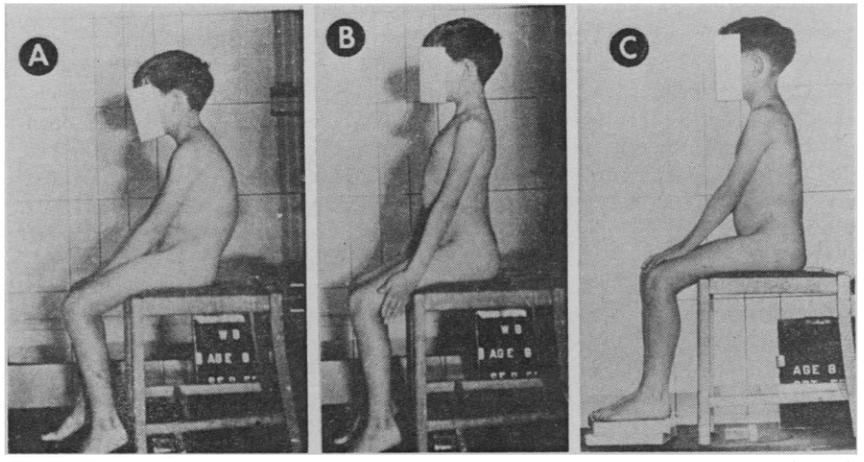

Fig. 4. Three sitting positions: (a) slumping; (b) sitting too straight; and (c) balanced. [From (*23*); courtesy of Gollancz, London]

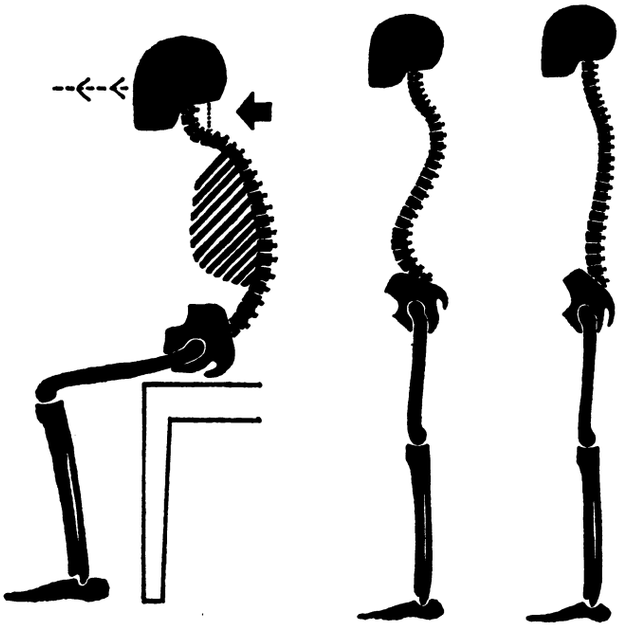

Fig. 5 (left). Position of pelvis, back, neck and head in slumping position. [From (*23*); courtesy of Gollancz, London] Fig. 6 (right). Standing in hunched position (left) and well balanced (right) [From (*23*); courtesy of Gollancz, London]

his voice altogether. When no doctor could help him, he took matters into his own hands. He began to observe himself in front of a mirror, and then he noticed that his voice was at its worst when he adopted the stances which to him felt appropriate and right for what he was reciting. Without any outside help he worked out, during a series of agonizing years, how to improve what is now called the "use" of his body musculature in all his postures and movements. And, the remarkable outcome was that he regained control of his voice. This story, of perceptiveness, of intelligence, and of persistence, shown by a man without medical training, is one of the true epics of medical research and practice (*22*).

Once Alexander had become aware of the misuse of his own body, he began to observe his fellowmen, and he found that, at least in modern Western society, the majority of people stand, sit, and move in an equally defective manner.

Encouraged by a doctor in Sydney, he now became a kind of missionary. He set out to teach—first actors, then a variety of people—how to restore the proper use of their musculature. Gradually he discovered that he could in this way alleviate an astonishing variety of somatic and mental illnesses. He also wrote extensively on the subject. And finally he taught a number of his pupils to become teachers in their turn, and to achieve the same results with their patients. Whereas it had taken him years to work out the technique and to apply it to his own body, a successful course became a matter of months, with occasional refresher sessions afterward. Admittedly, the training of a good Alexander teacher takes a few years.

For scores of years a small but dedicated number of pupils have continued his work. Their combined successes have recently been described by Barlow (*23*). I must admit that his physiological explanations of how the treatment could be supposed to work (and also a touch of hero worship in his book) made me initially a little doubtful and even skeptical. But the claims made, first by Alexander, and reiterated and extended by Barlow, sounded so extraordinary that I felt I ought to give the method at least the benefit of the doubt. And so, arguing that medical practice often goes by the sound empirical principle of "the proof of the pudding is in the eating," my wife, one of our daughters, and I decided to undergo treatment ourselves, and also

to use the opportunity for observing its effects as critically as we could. For obvious reasons, each of us went to a different Alexander teacher.

We discovered that the therapy is based on exceptionally sophisticated observation, not only by means of vision but also to a surprising extent by using the sense of touch. It consists in essence of no more than a very gentle, first exploratory, and then corrective manipulation of the entire muscular system. This starts with the head and neck, then very soon the shoulders and chest are involved, and finally the pelvis, legs, and feet, until the whole body is under scrutiny and treatment. As in our own observations of children, the therapist is continuously monitoring the body, and adjusting his procedure all the time. What is actually done varies from one patient to another, depending on what kind of misuse the diagnostic exploration reveals. And naturally, it affects different people in different ways. But between the three of us, we already notice, with growing amazement, very striking improvements in such diverse things as high blood pressure, breathing, depth of sleep, overall cheerfulness and mental alertness, resilience against outside pressures, and also in such a refined skill as playing a stringed instrument.

So from personal experience we can already confirm some of the seemingly fantastic claims made by Alexander and his followers, namely, that many types of underperformance and even ailments, both mental and physical, can be alleviated, sometimes to a surprising extent, by teaching the body musculature to function differently. And although we have by no means finished our course, the evidence given and documented by Alexander and Barlow of beneficial effects on a variety of vital functions no longer sounds so astonishing to us. Their long list includes first of all what Barlow calls the "rag bag" of rheumatism, including various forms of arthritis, then respiratory troubles, and even potentially lethal asthma; following in their wake, circulation defects, which may lead to high blood pressure and also to some dangerous heart conditions; gastrointestinal disorders of many types; various gynecological conditions; sexual failures; migraines and depressive states that often lead to suicide; in short, a very wide spectrum of diseases, both somatic and mental, that are not caused by identifiable parasites.

Although no one would claim that the Alexander treatment is a cure-all in every case, there can be no doubt that it often does have profound and beneficial effects; and, I repeat once more, both in the mental and somatic sphere.

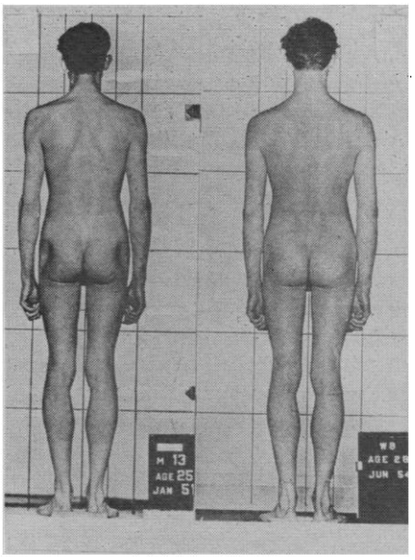

Fig. 7. Posture before (left) and after (right) Alexander treatment. The photograph on the left shows muscle contractions at the back of the neck; raised shoulders and tightened buttocks. After treatment these tensions had disappeared and the patient was overall taller. [From (*23*); courtesy of Gollancz, London]

The importance of the treatment has been stressed by many prominent people, for instance, John Dewey (*24*), Aldous Huxley (*25*), and, perhaps more convincing to us, by scientists of renown, such as Coghill (*26*), Dart (*27*), and the great neurophysiologist Sherrington (*28*). Yet, with few exceptions, the medical profession has largely ignored Alexander, perhaps under the impression that he was the center of some kind of cult, and also because the effects seemed difficult to explain. And this brings me to my next point.

Once one knows that an empirically developed therapy has demonstrable effects, one likes to know how it could work—what its physiological explanation could be. And here some recent discoveries in the borderline field between neurophysiology and ethology can make some aspects of the Alexander therapy more understandable and more plausible than they could have been in Sherrington's time.

One of these new discoveries concerns the key concept of reafference (*29*). There are many strong indications that, at various levels of integration, from single muscle units up to complex behavior, the correct performance of many movements is continuously checked by the brain. It does this by comparing a feedback report that says "orders carried out" with the feedback expectation for which, with the initiation of each movement, the brain has been alerted. Only when the expected feedback and the actual feedback match does the brain stop sending out commands for corrective action. Already the discoverers of this principle, von Holst and Mittelstaedt, knew that the functioning of this complex mechanism could vary from moment to moment with the internal state of the subject—the "target value" or *Sollwert* of the expected feedback changes with the motor commands that are given. But what Alexander has discovered beyond this is that a lifelong misuse of the body muscles (such as caused by, for instance, too much sitting and too little walking) can make the entire system go wrong. As a consequence, reports that "all is correct" are received by the brain (or perhaps interpreted as correct) when in fact all is very wrong. A person can feel at ease, for example, when slouching in front of a television set, when in fact he is grossly abusing his body. I can show you only a few examples, but they will be familiar to all of you (Figs. 3 to 7).

It is still an open question exactly where in this complex mechanism the matching procedure goes wrong under the influence of consistent misuse. But the modern ethologist feels inclined, with Alexander and Barlow, to blame phenotypic rather than genetic causes for misuse. It is highly unlikely that in their very long evolutionary history of walking upright, the hominids have not had time to evolve the correct mechanisms for bipedal locomotion. This conclusion receives support from the surprising, but indubitable fact that even after 40 to 50 years of obvious misuse one's body can (one might say) snap back into proper, and in many respects more healthy, use as a result of a short series of half-hourly sessions. Proper stance and movement are obviously genetically old, environment-resistant behaviors (*30*). Misuse, with all its psychosomatic, or rather somatopsychic, consequences must therefore be considered a result of modern living conditions of a culturally determined stress. I might add here that I am not merely thinking of too much sitting, but just as much of the cowed posture that one assumes when one feels that one is not quite up to one's work, when one feels insecure.

Second, it need not cause surprise

that a mere gentle handling of body muscles can have such profound effects on both body and mind. The more that is being discovered about psychosomatic diseases, and in general about the extremely complex two-way traffic between the brain and the rest of the body, the more obvious it has become that too rigid a distinction between mind and body is of only limited use to medical science, in fact can be a hindrance to its advance.

A third biologically interesting aspect of the Alexander therapy is that every session clearly demonstrates that the innumerable muscles of the body are continuously operating as an intricately linked web. Whenever a gentle pressure is used to make a slight change in leg posture, the neck muscles react immediately. Conversely, when the therapist helps one to release the neck muscles, it is amazing to see quite pronounced movements, for instance of the toes, even when one is lying on a couch.

In this short sketch, I can do no more than characterize, and recommend, the Alexander treatment as an extremely sophisticated form of rehabilitation, or rather of redeployment, of the entire muscular equipment, and through that of many other organs. Compared with this, many types of physiotherapy which are now in general use look surprisingly crude and restricted in their effect, and sometimes even harmful to the rest of the body.

What then is the upshot of these few brief remarks about early childhood autism and about the Alexander treatment? What have these two examples in common? First of all they stress the importance for medical science of open-minded observation—of "watching and wondering." This basic scientific method is still too often looked down on by those blinded by the glamor of apparatus, by the prestige of tests, and by the temptation to turn to drugs. But it is by using this old method of observation that both autism and general misuse of the body can be seen in a new light; to a much larger extent than is now realized both could very well be due to modern stressful conditions.

But beyond this I feel that my two excursions into the field of medical research have much wider implications. Medical science and practice meet with a growing sense of unease and of lack of confidence from the side of the general public. The causes of this are complex, but at least in one respect the situation could be improved: a little more open-mindedness (*31*), a little more collaboration with other biological sciences, and a little more attention to the body as a whole and to the unity of body and mind could substantially enrich the field of medical research. I therefore appeal to our medical colleagues to recognize that the study of animals—in particular "plain" observation—can make useful contributions to human biology not only in the field of somatic malfunctioning, but also in that of behavioral disturbances, and ultimately help us to understand what psychosocial stress is doing to us. It is stress in the widest sense, the inadequacy of our adjustability, that will become perhaps the most important disruptive influence in our society.

If I have today emphasized the applicability of animal behavior research I do not want to be misunderstood. As in all sciences, applications come in the wake of research motivated by sheer intellectual curiosity. What this occasion enables me to emphasize is that biologically oriented research into animal behavior, which has been done so far with very modest budgets, deserves encouragement, whatever the motivation and whatever the ultimate aims of the researcher. And we ethologists must be prepared to respond to the challenge if and when it comes.

References and Notes

1. I call the method old because it must already have been highly developed by our ancestral hunter-gatherers, as it still is in non-Westernized hunting-gathering tribes such as the Bushmen, the Eskimo, and the Australian Aborigines. As a scientific method applied to man it could be said to have been revived first by Charles Darwin in 1872 in *The Expression of the Emotions in Man and the Animals* (John Murray, London, 1872).
2. L. Levi, Ed., *Society, Stress, and Disease*, volume 1, *The Psychosocial Environment and Psychosomatic Diseases* (Oxford Univ. Press, London, 1971).
3. L. Kanner, "Autistic disturbances of affective contact," *Nerv. Child* **2**, 217 (1943). Recently, Kanner has published a selection of his papers [*Childhood Psychosis* (Winston, Washington, D.C., 1973) (distributed by Wiley)].
4. When I speak of Kanner's syndrome, I refer to the largely descriptive list of symptoms given by G. O'Gorman [*The Nature of Childhood Autism* (Butterworth, London, 1970)]. This is a slightly modified version of the description given by M. Creak ["The schizophrenic syndrome in childhood: Progress report of a working party," *Br. Med. J.* **2**, 889 (1961). Many other definitions of autism in its various forms are mixtures of observed behavioral deviations and interpretations. For a discussion of the confusion surrounding the word "autism" see Tinbergen and Tinbergen (*12*, pp. 45–46).
5. For the purpose of finding one's way in this literature we can refer to M. Rutter [*Infantile Autism* (Methuen, London, 1965)] and to the quarterly, started in 1971 (*Journal of Autism and Childhood Schizophrenia*, published by Winston, Washington, D.C.), which prints original articles as well as reviews. The most recent and most exhaustive review is by E. M. Ornitz [*Calif. Med.* **118**, 21 (1973)]. Throughout the literature (not only on autism but on many other psychiatric issues as well) one finds one fundamental error in scientific reasoning. Time and again we receive the comment that we overlook the "hard" evidence of internal malfunctioning in autists as well as in other categories of the mentally ill. I assure my readers that we do not *overlook* such evidence (such as that on blood platelets, on lead contents, and on electronencephalogram patterns). The erroneous assumption underlying most of the arguments in which such facts are used for the purpose of throwing light on the causation of the behavioral deviation is almost invariably due to *the confusion between correlations and cause-effect relations*. With some exceptions (such as the deleterious effect of lead) the physiological or biochemical evidence is considered, without any ground whatsoever, to indicate causes, whereas the correlations found could just as well point to consequences or side effects. It is just as nonsensical to say that retarded bone growth, or abnormalities in the blood platelet picture (or for that matter speech defects, or high overall arousal) are causes of autism as it is to say that a high temperature is the cause of typhoid or pneumonia. Unless there is evidence, clinical and ultimately experimental, indicating what is cause and what is effect the opinions based on hard evidence are in fact worthless. Our experimental evidence discussed on pages 21 and 22 is hard, whereas evidence on correlations—however impressive the techniques might be by which they are found—are scientifically useless until an attempt is made to place it into cause-effect context. This is what I mean in my final paragraphs by "the glamor of apparatus"—the idolization of techniques, coupled with the failure to think about the meaning of evidence, is a serious disease of medical research.
6. B. Rimland, *J. Autism Child. Schizophrenia* **1**, 161 (1971).
7. L. Wing, *Br. J. Hosp. Med.* (1970), p. 381; see also E. A. Tinbergen and N. Tinbergen (*12*, p. 51).
8. One of the most prominent exponents of this view is B. Bettelheim [*The Empty Fortress: Infantile Autism and the Birth of Self* (Collier-Macmillan, London, 1967)]; see also H. Clancy and G. McBride, *J. Child Psychol. Psychiatry Appl. Discip.* **10**, 233 (1969).
9. M. Rutter, L. Bertak, S. Newman, in *Infantile Autism: Concepts, Characteristics, and Treatment*, M. Rutter, Ed. (Churchill, London, (1971).
10. C. Hutt, S. J. Hutt, D. Lee, C. Ounsted, *Nature (Lond.)* **204**, 908 (1964); S. J. Hutt and C. Hutt, Eds., *Behaviour Studies in Psychiatry* (Pergamon Press, Oxford, 1970).
11. G. Stroh and D. Buick, in *Behaviour Studies in Psychiatry*, S. J. Hutt and C. Hutt, Eds. (Pergamon Press, Oxford, 1970), pp. 161–174.
12. E. A. Tinbergen and N. Tinbergen, *Adv. Ethol.* **10**, 1 (1972).
13. S. J. Hutt and C. Hutt, *Direct Observation and Measurement of Behavior* (Thomas, Springfield, Ill., 1970).
14. P. B. Medawar, *The Art of the Soluble* (Methuen, London, 1967).
15. For a recent review about the analysis of nonverbal signs of mixed motivation, or motivational conflicts, see, for example, A. Manning [*An Introduction to Animal Behaviour* (Arnold, London, 1972), chap. 5] and R. A. Hinde [*Animal Behavior* (McGraw-Hill, New York, 1970), chap. 17]; both books give further references.
16. See N. G. Blurton Jones, Ed., *Ethological Studies of Infant Behaviour* (Cambridge Univ. Press, London, 1972).
17. S. Chess, *J. Autism Child. Schizophrenia* **1**, 33 (1971). The point I want to make with this brief reference is that, while one should call rubella an early environmental influence and therefore not congenital in the sense of genetic, it might well be correct to call it organic, even though rubella could well create a state of anxiety already during pregnancy in mothers who have heard about other damaging effects of the disease. And this in itself could well cause a complex psychosomatic state.
18. N. L. J. Kamp, *Psychiatr. Neurol. Neurochir.* **67**, 143 (1964); G. E. Vaillant *Arch. Gen. Psychiatry* **9**, 163 (1963). While I do not of course intend to underrate the possibility of

genetic predisposition, the hypothesis of a purely genetic deviation conflicts with this type of observation. At the same time we know that even when twins grow up in the same family, their experiences can never be identical.
19. See, for example, L. Wing, *Autistic Children* (Constable, London, 1971), p. 8.
20. Although not all the authors of the following books label their subject as "autistic," I mention them because the descriptions of the initial behavior conform in whole or in part to Kanner's syndrome; and, as I have said, I consider such descriptions the only acceptable starting points: R. d'Ambrosio, *No Language but a Cry* (Cassell, London, 1971); V. Axline, *Dibs—In Search of Self* (reprint of 1964 publication, Penguin Books, Harmondsworth, England, 1971); J. Copeland and J. Hodges, *For the Love of Ann* (Arrow Original, London, 1973); J. M. Hundley, *The Small Outsider* (reprint of 1971 publication, Angus and Robertson, Sydney, 1973); C. C. Park, *The Siege* (reprint of 1967 publication, Penguin Books, Harmondsworth, England, 1972); S. S. Wexler, *The Story of Sandy* (reprint of 1955 publication, Signet, New York, 1971); G. Thieme, *Leben mit unserem autistischen Kind* (Hilfe für das Autistische Kind e.V., Lüdenscheid, West Germany, 1971). No two of these seven children received the same treatment, but on the whole one can say that those who were treated primarily at the emotional level rather than at the level of specific skills showed the most striking improvement.
21. The clearest introduction is: F. M. Alexander, *The Use of Self* (Chaterston, London, 1932); but a great deal of interest can also be found in: F. M. Alexander, *Man's Supreme Inheritance* (Chaterston, London, 1910) and *The Universal Constant in Living* (Chaterston, London, 1942).
22. The history of medical science is full of such examples of breakthroughs due to a reorientation of attention. Compare, for example, Jenner's discovery that milkmaids did not contract smallpox; Goldberger's observation that the staff of a "lunatic asylum" did not develop pellagra; Fleming's wondering about empty areas around the *Penicillium* in his cultures
23. W. Barlow, *The Alexander Principle* (Gollancz, London, 1973).
24. J. Dewey, see, for example, introduction to F. M. Alexander, *The Use of Self* (Chaterston, London, 1932).
25. A. Huxley, *Ends and Means* (Chatto and Windus, London, 1937); "End-gaining and means whereby," *Alexander J.* **4**, 19 (1965).
26. G. E. Coghill, "Appreciation: The educational methods of F. Mathias Alexander," in F. M. Alexander, *The Universal Constant in Living* (Dutton, New York, 1941).
27. R. A. Dart, *S. Afr. Med. J.* **21**, 74 (1947); *An Anatomist's Tribute to F. M. Alexander* (Sheildrake Press, London, 1970).
28. C. S. Sherrington, *The Endeavour of Jean Fernel* (Cambridge Univ. Press, London, 1946); *Man on His Nature* (Cambridge Univ. Press, London, 1951).
29. E. von Holst and H. Mittelstaedt, *Naturwissenschaften* **37**, 464 (1950).
30. N. Tinbergen, *Proc. Roy. Soc. Lond. Ser. B* **182**, 385 (1973).
31. This plea is nowadays heard more often: see L. Kanner, *J. Autism Child Schizophrenia* **1**, 453 (1971); see also p. 457.

Resource Partitioning in Ecological Communities

Research on how similar species divide resources helps reveal the natural regulation of species diversity.

Thomas W. Schoener

Biologists have long been intrigued by differences in morphology and habit among closely related species, for to comprehend the manner and extent of such differences is to comprehend much of the natural control of organic diversity. Ecologists especially have concentrated on differences in the way species in the same community utilize resources. Studies of this resource partitioning are currently enjoying great popularity. In fact, in the 12 years since Hutchinson (*1*) posed his celebrated riddle: "Why are there so many kinds of animals?" such studies have grown exponentially at a rate four times that typical of scientific works (*2*).

The major purpose of resource-partitioning studies is to analyze the limits interspecific competition place on the number of species that can stably coexist. That such limits exist was suggested by the mathematical models of two early 20th-century theoreticians, Lotka and Volterra (*3*). The idea was supported by experiments of Gause (*4*) and later workers on simple organisms in laboratory containers, in which similar species tended to cause one another's extinction. The infusion of models and data crystallized into the Gause principle, one version of which states that species cannot coexist for long if they too similarly use the same kinds of resources. The application of this idea to natural communities, however, was begun primarily in the 1940's during the time of the New Systematics. Although at first interested in differences between species that might ensure reproductive isolation, evolutionists quickly seized upon the idea of reducing competition as an alternative rationale for those differences (*5*). So rapidly, in fact, did this idea take hold that David Lack, eventually its principal proselytizer, was placed by a publication lag in the awkward position of having two quite different explanations for bill differences in Darwin's finches appear simultaneously (*6*)!

Hutchinson's (*7*) reformulation of the concept of ecological niche provided a precise language for the description of resource partitioning. In essence, he proposed that a species' population could be characterized by its position along each of a set of dimensions ordering environmental variables such as ambient temperature, prey size, and so on. Ideally, these dimensions would be few and independent. Hutchinson originally conceived of the niche as comprising intervals of population survival along each dimension. Now, however, many ecologists consider the frequency distribution of utilization or occurrence along dimensions as the niche.

What evidence demonstrates that the pattern of resource utilization among species results from competition? Mere presence of differences is not enough, for even if niches were arranged randomly with respect to one another, differences would exist. Hence a sufficiently precise search for differences would be bound to result in their detection, a state of affairs that led Slobodkin (*8*) to rephrase the Gause principle as a "rule of ecological procedure" rather than a verified or even verifiable proposition.

Ecologists now follow two approaches in their attempts to make a case for the importance of competition in nature. One approach is experimental, the other is observational.

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