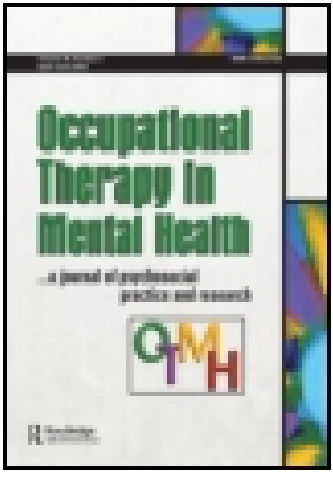

Occupational Therapy in Mental Health

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Occupational Therapy and Neuropsychiatry

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Occupational Therapy and Neuropsychiatry

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ABSTRACT. It is suggested that increasing evidence of neurophysiological etiologies for mental illness must be taken into account in rationalizing the effectiveness of occupational therapy for psychiatric disorders. The role of activity in metabolizing stress hormones is described as a possible mechanism of therapeutic effectiveness. The sensory integrative effects of certain activities are also postulated to contribute to improvement in selected cases.

The post-1974 literature relating to sensory integrative treatment of schizophrenia is reviewed and methodological problems in the research are discussed. The role of vestibular abnormalities in the theoretical formulations regarding schizophrenia is assessed and problems in measuring vestibular processing are enumerated.

Priorities for occupational therapy research are suggested and plans for avoiding methodological pitfalls are described. In conclusion it is emphasized that stress theory and sensory integration theory are compatible and that both can be integrated with current knowledge about neurophysiology.

The revolution in psychiatry which began with the major tranquilizers is far from over. The technologies of the CAT and PETT scans, plus a growing knowledge of the neurotransmitters, the endorphins, the prostaglandins, the hypothalamic releasing factors—all this will probably lay to rest the concept of mental disorders as primarily psychosocial in nature.

The explosion of knowledge in the neurosciences is occurring in a society which is also undergoing a quiet revolution in health care at-

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titudes and practices. This is seen in such areas as the rapid expansion of the holistic health movement, the upsurge of interest in nutrition and the proliferation of sports participation as a health maintenance measure.

All of these changes taken together will inevitably be reflected in the mental health field in different legal formulations and revised treatment strategies. Laws will be changed to give more protection to the incompetent person who will once more be referred to as a patient. More effective drug and nutritional regimens will be developed. And, among these other possibilities, there is a real chance that the 18th-century roots of occupational therapy will be re-discovered—hopefully by occupational therapists.

In the 18th century, the work-a-day world for most people consisted of hard physical labor—most of it closely and obviously connected with survival; growing and processing food, procuring firewood, building shelter. The Tukes family at York Retreat, and the institutions in America which followed their lead, used these same everyday, homely labors as a means of restoring the sanity of their charges, believing that work was healing and holy.

In the 20th century, the work-a-day world for many people consists of sitting at a desk or standing in an assembly line for many hours, this sandwiched between nerve-wracking bouts with city traffic. Hard physical labor, for the most part, is now a leisure activity, called sports. Many people watch it but do not participate. Treatment of the mentally ill generally consists of brief hospitalization for the purpose of instituting a drug regimen, during which time a considerable amount of time is spent sitting in individual or group psychotherapy. If the patient improves, he goes back to his sedentary life. If he does not improve he goes to an even more sedentary life in a boarding house, watching TV or just sitting.

According to Bockoven[1] the treatment methods of 200 years ago—of supervised, assisted, graded work activities—were remarkably successful. They began to fail only when population growth, urbanization and the concomitant rapid increase of organic brain syndromes due to CNS syphilis and alcoholism began to fill the institutions, making it impossible for staff to carry out active work programs.

Many will recognize that the description of the 18th-century work and 20th-century sports can essentially be boiled down to what Rood calls "heavy work patterns"—resisted use of tonic muscle groups, and co-contraction patterns, combined with what Ayres has de-

scribed in terms of vestibular, proprioceptive and tactile feedback mechanisms.

It may be possible that the success of "the work cure" lay in its unwitting adherence to some important physiological principles, in which case it might be worthwhile to try once more the use of physical activities in the treatment of the mentally ill. This could only be justified, however, if current scientific knowledge can be used to rationalize or explain the theoretical benefits. On the other hand, it is hard to justify attempts to confirm scientifically a method of treatment unless it is first demonstrated to be effective. This "which comes first, the chicken or the egg" type of dilemma faces other treatment professions as well as occupational therapy (if that is any comfort).

Since the problem must be attacked first at one end or the other, let us start with theory. It seems that on the basis of current knowledge there are at least two plausible explanations for the fact that activities embodying heavy work patterns, co-contraction and vestibular, proprioceptive and tactile feedback, seem to benefit some psychiatric patients. These two explanations are by no means mutually exclusive. I refer to stress theory and sensory integration theory. First a brief discussion of stress as it relates to schizophrenia and to physical activity.

The discussion must start with the neurotransmitters. There is a voluminous literature summarized by Meltzer[2] linking dopamine and norepinephrine to the schizophrenic psychoses.

The connection was noted initially with the discovery that it was the amount of anticholinergic action of the various phenothiazines that coincided with their anti-psychotic activity. More recent research, summarized by Hartmann,[3] indicates that the depletion of norepinephrine, accompanied by increased dopamine and/or the blocking of normal dopamine metabolism could account for a variety of schizophrenic symptoms.

Dopamine appears to be connected with generalized arousal as opposed to the focused alertness associated with norepinephrine. Kety[4] also suggests that an imbalance, too little norepinephrine, too much dopamine, could be involved in the postulated biologic vulnerability to stress. He speculates that gonadal and adrenocortical steroids may modify the activity of dopamine betahydroxylase which would help to explain the onset of schizophrenia after puberty and its aggravation by stress. The adrenocortico steroids are, of course, the stress hormones first elucidated by Hans Selye.[5]

Corbett[6] has suggested that an inherited vulnerability to stress expressed in poor modulation of synaptic function is an important predisposing factor.

In short, the supposition is that just as some individuals are predisposed to react to acute or chronic stress with stomach ulcers, and others with hypertension, so there are those who react with disturbances in neurotransmitter function.

The literature contains numerous linkages of schizophrenia with stress. Acute psychotic episodes frequently are triggered by stress such as life change and crises (Arieti).[7] Bleuler[8] has also noted the connections between the stress of physical illness or hormonal imbalance and the onset of schizophrenic psychoses. The research on process or early onset schizophrenia suggests high levels of arousal consistent with chronic stress states.

Activity, as it impinges on the effects of stress, was discussed by King[9] citing the work of Gal and Lazarus[10] and others. It is not necessary to repeat that material here. Suffice it to say that the well known "fight or flight" response to stress involved strenuous physical activity, which, in addition to securing the organism's survival, also resulted in the rapid metabolism of stress hormones, thus preventing secondary damage to the system.

Thus the true efficacy of the hard physical labor characteristic of "moral treatment" could be conceptualized as its tendency to normalize neurotransmitter balance which has been deranged by stress. This may well be the mechanism operating in the successful use of running by some psychiatrists as a treatment for depression.[11, 12]

An alternate rationale for the efficacy of gross motor activities is found in sensory integration theory. Some opposition has been voiced to the use of sensory integration with adults on the grounds that it was developed with and for learning disabled children. However, Ayres[13] says:

> It always comes as a surprise to me when the theory of sensory integrative dysfunction and its treatment is identified primarily with learning disorders. It surprises me because I see this theory as a way of looking at one aspect of development in human beings and some of the malfunctions that befall those beings. Most of the concepts derive from basic research on the vertebrate brain, a science that knows virtually nothing of educational handicaps . . .

My interest in perceptual disorders arose when treating the overtly brain damaged such as stroke and cerebral palsied patients. It then extended to children with "brain damage" (a term used freely at that time) but without upper motor neuron disorder, and then on to the child with minimal brain dysfunction.

There is increasing evidence to support the idea that at least some schizophrenics are brain damaged. Smythies in a recent lecture[14] cited CATT scan studies showing marked changes in schizophrenic brains compared to normals. There were also interesting differences between acute or reactive patients and the typical process-chronic patient. It has been a riddle to researchers for years that there were almost no tests which would distinguish reliably between chronic schizophrenics and organic brain syndrome patients. Now Golden[15] points out that the fault is probably not with the tests but with the stubborn assumption that chronic schizophrenics are not neurologically impaired.

Thus there seems as much reason to apply Ayres work—originally developed with a brain dysfunctional population to the chronic schizophrenic as to other brain injured patients—such as the victims of stroke or trauma.

To those who cling to the idea that the brain is incapable of development after puberty, Angelo's[16] study of low achieving college students should provide food for thought.

Since the specific rationale for using sensory integrative treatment with schizophrenics has been set forth in some detail by King, details of those studies should be clarified, since they have been misunderstood by some. Although King mentioned an initial group of 15 highly visible chronic schizophrenics, this was by no means the only group treated or on which conclusions were based. Over the course of 6 years upwards of 150 patients were included in the gross motor treatment groups. Since it was never felt possible to refuse treatment to patients, King worked with both sexes and all ages and diagnoses. When King reported in 1974 that gross motor activities seemed to benefit chronic process schizophrenics and did not seem to benefit paranoids, the statement was based on experience, not theory.

Another ambiguity which has emerged is in the definition of chronicity. No one in our original sample had been hospitalized for less than 10 years. Screening out those who were not oriented as to

time, place and person would have eliminated two-thirds of the original group, while requiring that they appear to understand directions would have eliminated the rest.

As experience increased it seemed that the criterion was not so much chronicity measured by years of hospitalization as pre-morbid history, combined with posture and movement characteristics. Thus improvement was seen in young, first-hospitalization clients who fitted the pattern.

In this connection, attention should be called to a recent study by Cantor, Pearce and Evans[18] called "the Group of Hypotonic Schizophrenias." The subjects were schizophrenic children and the striking similarity in posture to the "S" curve posture which we described in 1974 is very interesting. Muscle fiber abnormalities identical to those described earlier by Melzer[19] were also found.

The studies since 1974 of sensory integration and schizophrenia can roughly be divided into those attempting to replicate treatment results and those exploring the hypothesized theoretical base. A survey of the treatment studies reveals that they range from a single subject study by Leveille[20] to an N of 33 including controls, reported by Crist.[21] There were informal pilot studies as reported by Levine, Jorstad et al.,[23] Rider's[24] study where the patient was his own control, and formal experimental and control group studies such as Bailey's,[25] Crist's and King's study.[26]

Behaviors measured included verbalization (Bailey) NOSIE scores (Rider, King), person drawing scores (Crist, King, Rider, Levine), posture and autonomic response to exercise (Rider), speed of response test (King '77), general behavioral improvements (Leveille, Levine, Jorstad).

Patient selection was in general limited to chronic schizophrenics, though it was not clear in some studies (Rider) whether paranoids were included. Three of her 5 subjects were found to have mild congenital defects.

In some studies (Crist) the age discrepancy between experimental group and control group was wide enough to cast doubt on the results.

Results have varied widely. Bailey found statistically significant improvement in relevance of verbalization but not in speed of verbal response or quantity of verbalization. King (27) saw improvement in NOSIE scores and person drawing scores, but not in speed of response. Crist failed to get significant improvement in person drawing scores, while Rider's subjects improved on the person

drawing during treatment but failed to maintain gains six weeks after treatment was ended. Rider's subjects became more active and less irritable according to NOSIE scores, but on other factors gains either were not maintained or did not occur. Posture did not change in her subjecs but gait (which was not a target behavior) improved in all 5 subjects. Leveille reports slow but steady improvement in behavior in her subject which was important in his overall adjustment and Levine and Jorstead also reported important improvement in behavior.

While the overall results are not particularly convincing, neither are they totally without significance. Since this subject group generally had failed to respond to other treatment strategies over a long period of time, any improvements are worth noting.

Attempts to examine the theoretical base for sensory integration techniques in the treatment of schizophrenia have ranged from reviews of the literature to formal research protocols.

Saffir[28] studied the literature on the physiological effects of chlorpromazine and related this material to the hypothesized effects of sensory integration. She concluded that the two treatment strategies were compatible and could be mutually reinforcing.

Endler and Eiman[29] contrasted a paranoid and non-paranoid and a normal population on measures of reflex integration and bilateral integration. There were significant differences between both clinical populations and the normal group, but no significant differences between the two patient groups. However, their "chronic" paranoids averaged less than a year of hospitalization and their chronic non-paranoid group only a little over three years in hospital. This definition of chronic departs widely from that of other studies.

Huddleston[30] used the generally accepted Phillips scale, which assesses pre-morbid adjustment, to differentiate the process and reactive subjects for her study. The study showed significant differences between process and reactive schizophrenics on vestibular reactivity as measured by post rotary nystagmus. Differences between grip strength and posture were in the predicted direction, but did not reach statistical significance.

Lindquist[31] examined the differences between schizophrenic, manic depressive, spinal pain patients, and normal controls on direct measures of vestibular function such as post rotary nystagmus and standing balance, and on a number of indirect measures such as diadokokinesis and serial finger-thumb opposition. He also attempted to relate general activity level to the other variables.

There was no correlation found between Lindquist's so-called indirect measures of vestibular function and the direct ones, i.e., PRN and standing balance. One would be tempted to conclude that these measures are so indirect as to be useless in assessing vestibular function. They are, however, highly correlated with neurological integrity, which is worth noting.

Lindquist's rationale in choosing a group of manic-depressives as medication control group is not entirely clear, since the medications usually given manic depressives are quite different in their effects and mode of acting upon the nervous system from the drugs usually given schizophrenics.

Also, half of the spinal pain group were on medication—one on pain medication, the other 4 on unknown drugs. It would have been useful if Lindquist had separated the scores for the spinal pain group who were medicated.

Lindquist suggests that the reduced scores of the schizophrenic and spinal pain experimental groups on post rotary nystagmus and standing balance might be due to the effects of inactivity rather than any innate defect in vestibular processing. This is, of course, possible; however, his data is not necessarily suggestive of this. It is quite possible that lowered vestibular responsiveness could result in abnormal postural reflexes, tone, and movement patterns which could result in increased susceptibility to spinal injuries, pain and hence, reduced activity. Sorting cause from effect will require considerably more research.

A number of scholars have focused on the question of vestibular reactivity in schizophrenia, seeming to feel that this is the linchpin of the whole theoretical structure of sensory integration and schizophrenia. Putting aside for the moment the question of its importance to the theory, let us look at the controversy as to the evidence. The questions deal with the techniques of measuring nystagmus, both caloric and post rotary.

Among the most significant details are the effects of states of alertness, and controlled fixation in determining duration and amplitude. The effects of medication are an important question, as is the question of the importance of dysrhythmia and the masking effects it may have on duration and amplitude. By all odds the most painstaking and detailed study is Holzman, Levy and Proctor's 1978 report "Vestibular Responses in Schizophrenia."[32]

On the subject of effects of medication they report "these results suggest that heightened dysrhythmia occurs independently of medi-

eation status." They warn, however, that this conclusion needs replication.

While Holzman's group did not replicate earlier findings of hyporesponsiveness in chronic schizophrenics, they did note, "chronic deteriorated schizophrenics and recent schizophrenics have significantly greater dysrhythmic responses."

An interesting facet of the Holzman study is that results were very different depending on whether hospital diagnosis or psychological testing was used to determine diagnostic category. For example, "in an analysis based on hospital diagnosis we compared 29 recent schizophrenics with 25 chronic deteriorated schizophrenics. Multivariate tests produced significant overall differences between these two groups ($p < .0001$). Specifically, the groups differ on 4 out of 5 of the dependent variables." Chronic schizophrenics had less intense—and more dysrhythmic responses as well as later culmination times than did the recent schizophrenics. The chronic deteriorated schizophrenics had longer durations, however.

When psychological test diagnosis was used, results differed. When recently hospitalized acute schizophrenics were compared with recently hospitalized chronics, the results did not show any differences.

The Levy, Holzman, Proctor group divided their groups in so many ways and got so many differing results as to make drawing any conclusions very difficult. What emerges fairly clearly, however, is very significant dysrhythmic patterns in both chronic and recent schizophrenics.

One would have no quarrel with their statement that their findings are not consistent with peripheral vestibular disease in schizophrenics. Sensory integration theory does not postulate peripheral vestibular disease as being the problem, but rather central processing mechanisms. Levy, Holzman and Proctor conclude their abstract by saying that their results may "reflect state-related phenomena consistent with disturbances in alertness, which are not necessarily voluntary or motivational in origin."

Levy et al. do not deal at all with the fact that Angyal and his Worcester group used a behavioral measure of vestibular responsiveness, the "Stepping Test," and found under-responsiveness in their schizophrenic subjects. The Stepping Test, of course, is not subject to the criticisms made of techniques for measuring post rotary nystagmus.

They mention, but do not deal with, Fish's[33] findings of vestibular

anomalies in at-risk infants who later are identified as schizophrenic, nor do they comment on the findings of Ornitz[34] and Ritvo of decreased responsiveness of autistic children in sleep as well as waking patterns of response.

Lindquist states that the early studies, both of nystagmus and postural-ocular reactions, did not control for "the possible influences of medication, length of illness, age, sex, activity level and subtype of schizophrenia."

The lack of differentiation of subtype of schizophrenia is a serious flaw in most studies. Even Levy, Holzman et al. are suspect because their results were so different depending on hospital diagnosis versus psychological test results.

However, it is not accurate to say that medication was not controlled as a variable since all the early studies (prior to 1956) were carried out on unmedicated subjects. Myer's[35] subjects in 1973 had been off medication for over one year. Also Holzman, Proctor and Levy studied the effects of medication as mentioned earlier.

The effects of gender on vestibular response have not been studied systematically, but most of the early studies, also Myer's and Leach's[36] were carried out with male populations, so *that* variable was at least controlled.

Age is a factor which should be controlled, since it is often assumed that responsiveness to vestibular stimulation increases with age. However, there was a difference in mean age of 15 years between Lindquist's group one and group two. Given the difficulty in finding research populations, however, this is understandable, if regrettable.

Taking the evidence on balance, including Levy, Holzman and Proctor's postulation of an attentional state defect, it sems likely that there is some problem with vestibular system function in schizophrenia. Exactly what it is is not yet clear.

Before one worries unnecessarily about whether or not proof of vestibular hypo-responsiveness is absolutely necessary to a treatment theory, it seems that therapists have some higher priority issues to deal with.

The first question to ask about any treatment for any condition is "Does it work?" If the answer to that is negative, one need go no further. If the answer is positive, one then needs to narrow the question to: "For which patients is it most successful?" The answer to that question, by helping to define the characteristics of a certain

group, will assist with answering the third question, "Why does it work?"

The suggestion is, then, that higher priority needs to be given to demonstrating treatment effectiveness than to exploring the theoretical base. (Unfortunately, proving that, in theory, something *should* work, does not guarantee that in practice it *will* work.)

In looking at the treatment outcome studies of the last seven years, one can be pessimistic because the results have been meager, or one can be optimistic because there have been *some* positive results.

Being of an optimistic nature, the author prefers to think that as the profession becomes more sophisticated about research (and more careful), we will begin to see more significant results.

Several steps can be taken which will improve the percentages for success. The first is to define and describe the activities used in detail so that anyone wishing to replicate a study can be more certain that their treatment methods are identical.

A second important step is the selection of appropriate outcome measures. The general consensus is that changes in psychiatric status are the major determinant of the efficacy of a treatment strategy in psychiatry. Therefore, whatever *other* measures may be used, a psychiatric status test, like the NOSIE 30, should be part of the pre and post testing. This is not to minimize the importance of verbalization, ADL scores, and so forth, but to suggest that these measures accompany a psychiatric status test.

A close study of the treatment outcome studies already undertaken might reveal behaviors which anecdotal reports identify as having improved. If there is any concurrence as to what they are, then perhaps ways can be found to quantify them.

Since it is very difficult to find enough subjects to form homogeneous groups for a statistically meaningful research design, it might be wise to adopt other research strategies. The single case research design described by Hacker[37] can lend itself to our purposes. Rider used a variation of the single care design in her study. Leveille's case would have fitted this model very well with minor changes in procedure. A collection of well designed and executed single case experiments might well be more useful than a group design with poor control of variables.

If the time comes that we have demonstrated treatment effectiveness and are ready to move on to exploration of the "why does it

work?'' question, there is another set of ideas that may prove useful. First of all, there is not much point in evaluating vestibular responsiveness with techniques that have either been discredited or never standardized with adults.

A first step in this type of research might be standardizing evaluative measures such as Fukuda's Vertical Writing Test,[38] the Stepping Test[39] and the Romberg which measure behavioral or sensorimotor aspects of vestibular function.

One last comment on this subject is that if therapists can demonstrate that gross motor activities are successful in improving psychiatric status with a certain group or groups of patients, one can be sure that other disciplines will be eager to research why.

I began this discussion by pointing out that the concept of activity as a metabolizer of stress hormones is not at all in conflict with the idea of sensory integrative dysfunction. Build-up of stress hormones and the resulting interference with neurotransmitter function may well be an important mechanism producing breakdown of perceptual constancy. This might be a very important mechanism in the reactive schizophrenic.

If it can be demonstrated that gross motor activities are effective therapy, for whichever reason, we might even dare to dream of the return of the hospital garden and dairy as a means of helping rather than exploiting the patient. It is an exciting time to be working in psychiatry, an exciting time to be an explorer of inner space!

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