Neuropsychology of Psychosis
Overview and Summary of the Symposium
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The problem of the relation between organic and functional brain disorders or structure vs. function which this symposium deals with, is an old one. Schizophrenia for example, was once regarded as having no organic basis occurring in a clear sensorium and the presence of organic disorder had to be ruled out before a diagnosis of schizophrenia could be entertained. Early attempts at finding differences in sensory thresholds and reflex responses failed and had to wait for more sophisticated methods of experimental psychology for the discovery of deviations in schizophrenic responses (viz information processing techniques, signal detection methods and similar developments in reaction time studies, and sensory and perceptual thresholds). Today the evidence seems to indicate that there may be certain deviations in brain functioning and even in brain anatomy in schizophrenia. However, my attempt at integrating the two sets of papers along the organic - functional dimensions did not succeed, so that each paper will be treated as an entity in itself.

Turning to Dr. Mirsky's paper, we can safely assume that there is no evidence for believing that the proportion of schizophrenic genotypes in the kibbutz and in the town groups were different. However, there were twice as many phenotypes in the former. It is clear, however, that considering the small size of the sample, the standard error of the expected proportion of genotypes in the offspring, and the standard error of the level of penetrance, that the results may lie well within the expected chance range. However, the proportion of depressions in the kibbutz offspring presents a problem. Is this further evidence that there is a common underlying psychosis to the schizophrenic and the affective disorders and that the ecological niche, premorbid personality and social network (including family influences) determine the direction the psychosis takes? Or is the source of the excess of affective disorder due to the "other parent", the non-schizophrenic parent, whose diagnostic status is not specified? In a personal communication, Professor Kugelmass, one of the members of the original research team, intimated that perhaps a parallel study of offspring of affective parents is called for in which the diagnosis of both parents is determined and only those in whom no schizophrenic taint occurs are included. On the other hand, it may be possible that though the kibbutz and town samples were equally vulnerable, the psychosocial milieu of the kibbutz with its open intimacy may have proved more stressful and effective in eliciting phenotypes. The major thrust of Mirsky's paper is focused on the deficit syndrome developed at age 11 in those who subsequently developed schizophrenia spectrum disorders. This deficit exhibited itself in cognitive, affective and perceptual performances as well as in psychosocial factors - lonely, anxious, socially detached. One wonders whether the cognitive, attentional and perceptual deviations were the building blocks on which the psychosocial deviation developed. Assuming that the former are less dependent on the external environment than on internal maturational and genetic loadings, is it possible that these presumably behaviorally non-noxious deviations in themselves, produced the appearance of a maverick in their possessor. Once the maverick character of the person developed, his peers may have begun to treat him in a manner which produced the psychosocial deviations as a reaction formation. One would have thought that the peer influence would have been different in the closely knit intimate milieu of the Kibbutz, but according to Silberman and Tassone [1] no such difference developed.
Professor Cohen's ingenious experiments on reaction time are a fundamental contribution to unravelling some of the problems in this field. He had accepted the challenge that had been made in the Schizophrenia Bulletin [2] symposium on reaction time suggesting that the cross-over techniques may reflect faulty time judgment in schizophrenics rather than basic deviation in reaction time or set. Ingeniously, he provided an hour-glass contraption by which the subject could monitor the passage of time and found that even under these conditions the cross-over effect persisted. However, the cross-over effect under cued time and under no-cued time showed no relationship or even a slightly negative one [-.36]. Furthermore, the cross over effect under cued time correlated .76 with the average speed of response for schizophrenics. One wonders whether the cross-over effect, when freed from time judgment variance, is merely a function of speed of response. Since speed is so highly dependent on the general deficit syndrome, and is amenable to change under training, the value of the cross-over effect has to be reevaluated. Professor Cohen also investigated the cross-modality effect in reaction time. One problem still facing the results he reports is the difference between light and sound in the cross-modality effect. Perhaps equating the light and sound stimuli for their energies might explain the difference.

Dr. Goldberg provides a rationale for the occurrence of schizophrenia-like behavior as due not to the classical hypothesis of genetic transmission, but to environmental factors producing diffuse brain damage. Furthermore, the diffuse brain dysfunction can have the functional appearance of a focal disease, selectively implicating prefrontal cortex. These observations may have implication for the claim that prefrontal cortex dysfunction is related to schizophrenia. Brain damage of a very subtle variety may in part be responsible for the final pathways of schizophrenic behavior even though the location, time of occurrence and sequences of noxious events may differ. This may be the source of sporadic phenocopies. Whether common loci of injury will be found is debatable. The peculiarly complex functioning of neurons and intricate interaction of neurotransmitters make for a pathless jungle as Axelrod [3] indicated in his plenary address at this conference. One aspect of Goldberg's contribution is his suggestion that iatrogenic factors may be responsible for the frontal lobe syndrome features in schizophrenia. He does not consider the possibility that some of the apparent frontal lobe syndrome features may be ecogenically or nosocomially produced by psychosocial factors of isolation, deprivation, frustration and hopelessness. Now one would investigate this possibility is, of course, a very involved problem. Another problem requiring some attention is the question whether the poorer performances on the Wisconsin Sorting Test and Trail Marking Tests reflect specific deficits, or are they merely a reflection of the general deficit characterizing schizophrenic attitudes, interests and motivation. Chapman [4] has provided techniques for assessing this possibility but I am not aware that it has ever been applied to the above mentioned techniques.

Dr. Goldstein has been interested in studying neuropsychological test batteries and has come to the conclusion that they fail to discriminate between brain damaged patients and schizophrenics, even though no brain damage can be detected in schizophrenics. One possible explanation may be that performance due to brain lesions on these tests is mimicked by the performance due to the general deficit syndrome which characterizes schizophrenics. Furthermore, certain laboratory techniques based on the paradigm of information processing have found consistent differences between schizophrenics and normal controls. These findings may reflect differences in the processing of information in intact channels. Somehow, these functional deviations have not been captured by formal tests yet. Perhaps the formal neuropsychological test batteries
of the future will contain such techniques as evoked potentials, pupillography, heart rate in the psychophysiological sphere and continuous performance, span of apprehension, cross modality reaction time, dichotic listening with distraction in the behavioral sphere. It is interesting to note that the results of the clustering techniques emerged with level rather than profile difference across the tests. Perhaps methods directed at profile analysis such as Penrose's [5] method for dividing the total variance of the distances between profiles into level and shape components, might serve better than more general clustering techniques.

In considering the contrast between brain damaged patients and patients in a schizophrenic episode, a possibility arises that brain damage may partake more of the nature of a permanent "trait" while a schizophrenic episode is more like a "state". The reason why neuropsychological tests fail to tap the schizophrenic state is because they are geared to respond to permanent trait aspects and not to the transient dysfunctional characteristics that apply to schizophrenic episodes. While it is true that there are certain permanent personality traits that are frequently found in schizophrenics, these traits may be independent of the schizophrenic syndrome and hence not be of value in identifying the episode. Thus, the distinction between brain damage and schizophrenia parallels the difference between structure and function. Sherrington has somewhere pointed out that the distinction between function and structure is temporal. The question then arises when does a dysfunction become structural? If the psychosis lasts long enough does it partake of a structural change in the brain, or does it ever remain on a functional level? The sudden recovery of function after a long psychosis would argue for the latter.

REFERENCES


