Brit. J. Psychiat. (1973), 122, 687–8

ABSTRACT

A Physiological Difference Between Hallucinosis and Schizophrenia

By RUSSELL MEARES and THOMAS HORVATH

Boulton (1971), in an authoritative review, has pointed out that biochemical research into schizophrenia has depended upon the assumption that hallucinogens, such as mescaline and LSD produce a state similar to schizophrenia, and that such research has therefore principally been concerned with attempts to find hallucinogens, or substances closely related to them, in the bodily fluids of schizophrenics. Since this model has been criticized on phenomenological grounds, we decided to make a comparison between states of visual hallucinosis and of schizophrenia with thought disorder, using a measure which tests a common hypothesis concerning the origin of schizophrenic symptoms. This hypothesis suggests that the symptoms of schizophrenia are manifestations of failure of a hypothetical sensory 'filter' (Broadbent, 1958). This mechanism determines the individual's 'selective inattention' to the irrelevancies of his environment. Its function can be assessed by a study of habituation, which is the process whereby a randomly repeated stimulus fails in time to elicit an orienting response.

Method

Habituation rates were determined in 10 schizophrenics, whom three psychiatrists independently considered to be suffering from schizophrenic thought disorder. They were compared with those of 15 normals, and also with those of another 11 patients who suffered from visual hallucinosis as part of an acute brain syndrome. LSD was the cause of hallucinosis in two cases. All patients were alert when tested, and none were taking psycho-active medication. The ages were 36·2 years (s.e. 4·0) for the schizophrenics, 42·4 years (s.e. 4·7) for the normals, and 47·1 years (s.e. 5·7) for those with visual hallucinosis. None of these differences reach significance when the t-test is applied.

The data were analysed according to standard practice (Lader, Wing, 1966), so that changes in skin resistance were calculated as change in log skin conductance. The mean change in log conductance for each stimulus was plotted against the log stimulus number, and a regression line was drawn, using the method of least squares.

Results

The regression lines displayed in Fig. 1 show that the schizophrenics had no tendency to habituate, while on the other hand the subjects with visual hallucinosis habituated significantly more rapidly than normals. The mean changes in log skin conductance for each stimulus point are displayed in a table, which has been omitted for reasons of space, but can be supplied on request.

Using Student's t-test, the first significant difference between the schizophrenics' and the normals' response is at the fourth stimulus point, while the first significant difference between those with visual hallucinosis and normals is at the fifth stimulus point.

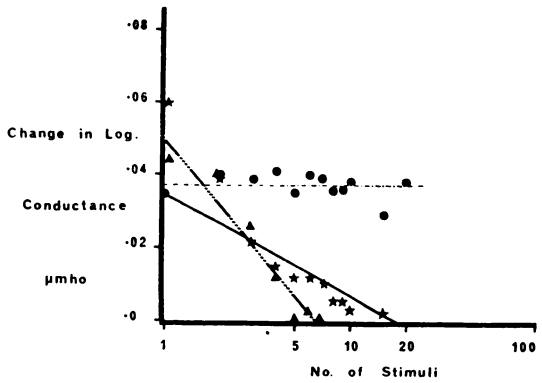


FIG. 1.—Log/linear regression lines of stimulus number against log skin conductance change. For schizophrenia (●) r = −·2559, b = ·0364, m = −·0028, and y = ·036−·003X. For those with visual hallucinosis (▲), r = −·9424, b = ·051, m = −·063, and y = ·051−·063X. For normals (★) r = −·9334, b = ·035, m = −·028, and y = ·035−·028X.

Discussion

This study suggests that schizophrenia and visual hallucinosis of toxic origin diverge from normal in opposing directions when the parameter is habituation rate, and that they may be therefore quite dissimilar in physiological terms. Conclusions must be tentative, since the subject numbers are small and the bases for the acute brain syndromes disparate. Nevertheless, the

https://doi.org/10.1192/bjp.122.6.687 Published online by Cambridge University Press

results are consistent with evidence that non-paranoid schizophrenia is a high 'arousal' state (Venables, 1967), and also, in contrast, that 'LSD and other catechol and indoleamine hallucinogens have some effects which are pharmacologically "depressant"' (Halasz, Formanek, Marrazzi, 1969).

Since these findings do not support the assumption that schizophrenia is comparable to states of visual hallucinosis induced by LSD or other toxins, they have implications concerning the validity of the 'model psychoses' as starting points in the search for the genesis of schizophrenia.

ACKNOWLEDGEMENT

This investigation was supported by a grant from the N.H. & M.R.C. of Australia.

REFERENCES

BOULTON, A. A. (1971). 'Biochemical research in schizophrenia.' *Nature*, **231**, 22–8.

BROADBENT, D. E. (1958). *Perception and Communication*. N.Y.: Pergamon.

HALASZ, M. F., FORMANEK, J., and MARRAZZI, A. S. (1969). 'Hallucinogen-tranquillizer interaction: its nature'. *Science*, **164**, 569–71.

LADER, M. H., and WING, L. (1966). '*Physiological Measures, Sedative Drugs and Morbid Anxiety*'. (Maudsley Monograph). London: O.U.P.

MEARES, R., and HORVATH, T. B. (1972). ' "Acute" and "chronic hysteria".' *Brit. J. Psychiat.*, **121**, 653–7.

VENABLES, P. H. (1967). 'Partial failure of cortical-subcortical integration as a factor underlying schizophrenic behaviour', in *Origins of Schizophrenia* (ed. J. Romano), p. 42–53. Amsterdam: Excerpta Medica.

Russell Meares, M.D., M.A.N.Z.C.P., M.R.C.Psych., *First Assistant, Departments of Psychiatry and Medicine, University of Melbourne,*

Thomas Horvath, M.B., B.S., M.R.A.C.P., *N.H. and M.R.C. Research Scholar, University of Melbourne,*

Austin Hospital, Heidelberg, Victoria 3084, Australia.

(*Received 19 June 1972*)

https://doi.org/10.1192/bjp.122.6.687 Published online by Cambridge University Press