

THE PALMAR ANHIDROTIC RESPONSE IN CHRONIC SCHIZOPHRENIA

MICHAEL ORR

M.D. (MALTA), B.A. (OXON.)

Summary

1. The palmar anhidrotic response to self-induced stress was investigated in a group of in-patient schizophrenics, a group of out-patient schizophrenics and a group of normal controls. The findings were then related to scores on clinical rating scales measuring the degree of withdrawal and the degree of morbidity both before and during the illness.

2. The control group and the group of outpatients showed normal palmar anhidrotic responses to the stress; the group of in-patients showed a paradoxical increase in the number of active glands immediately after the stress.

3. The basal level of the palmar sweating index was lower in the in-patient group than in the out-patient group and

that of the out-patient group was lower than that in the group of normal controls.

4. The findings suggested that schizophrenic patients characterized by high chronicity, high withdrawal and a poor pre-morbid personality are also subject to some degree of automatic disorganisation.

Autonomic function in schizophrenia has been the subject of sustained interest for many years and the findings from a considerable number of studies have led to the generally accepted notion that schizophrenics show some disturbance of autonomic function. Investigation of basal levels of indices of autonomic function by Gunderson (1953), Howe (1958), Whatmore and Ellis (1958) and Zahn (1964) and reactivity studies by Dmitriev *et al.* (1968) Zahn *et al.* (1968) and others, have confirmed the notion that schizophrenics are

overaroused and hyporeactive, the hyporeactivity probably arising as a result of the high basal arousal level, according to the law of initial values.

One aspect of autonomic function which has been recently highlighted is the palmar anhidrotic response to stress and its modification in schizophrenia (Mackinnon, 1969); the basis of the palmar anhidrotic response (PAR) lies in the fact that activity in the palmar sweat glands is inhibited in normal subjects placed in a situation characterised by psychological stress. Mackinnon *et al.* (1959) showed a decrease in the number of active sweat glands in mountaineers at high altitude and Harrison *et al.* (1962) showed a decrease in sweat gland activity in patients awaiting surgery. Palmar sweat gland activity in these studies was assessed by counts of active glands obtained from plastic casts of a predetermined area of skin from the palmar aspect of the fingers.

In an attempt to isolate the mechanism mediating the PAR, Harrison (1964) and Harrison and Mackinnon (1966) measured the palmar sweating index (PSI) — the number of active sweat glands over an area of skin 4mm square — under various conditions of induced stress and following the administration of epinephrine; it was seen that, of the many stresses applied only straight-leg raising produced an anhidrosis similar to that seen in patients awaiting surgery; the PAR could also be brought about by the administration of epinephrine and blocked by the administration of phenoxybenzamine; it was therefore concluded that straight-leg raising constituted a self-induced stress capable of activating both the adrenal cortex and the adrenal medulla.

Mackinnon (1969) has investigated the response of the palmar sweat glands in schizophrenics to self-induced stress and has found that both acute and chronic schizophrenics showed a tendency towards a paradoxical rise in the PSI following the stress.

In an attempt to replicate Mackinnon's findings on the PAR in schizophrenia, a group of chronic schizophrenics was selected and the reaction of the palmar

sweat glands to self-induced stress was examined; the findings were then related to clinical state and to the degree of morbidity both before and during the illness, as determined by the appropriate rating scales. Both in-patients and out-patients were tested in order to determine whether the differences in the outcome of the illness were associated with differences in the nature of the sweat gland response.

Methods

Subjects: Forty chronic schizophrenics were tested. The sample was divided into two groups, an in-patient group and an out-patient group; the in-patient group (IPG) consisted of 28 patients of whom 16 were males and 12 were females, the out-patient group consisted of 12 out-patients, of whom 7 were males and 5 were females. A group of ten normal controls, of whom 6 were males and 4 were females, was also tested.

Table 1 gives the mean age, mean chronicity in years and mean drug levels, together with their standard deviations, in the three groups. Mean drug levels were calculated from a survey of all drugs taken by a patient over the three weeks prior to testing; the daily chlorpromazine equivalence was then obtained and the appropriate dosage rating applied (for details see Held *et al.*, 1970).

TABLE 1

	IPG	OPG	NORMALS
Age	43.9	34.6	34.3
S.D.	10.2	6.9	12.2
Chronicity	19.5	9.3	-
S.D.	10.2	5.2	
Drug Level	1.7	1.5	-
S.D.	1.4	0.8	

Measurement of the Palmar Anhidrotic Response

The measurement of the palmar anhidrotic response was carried out by means of a series of estimations of the palmar

sweating index; this was obtained from counts of active sweat glands from plastic casts of the skin over the whorl of the ring finger of the left hand. This method was first described and suggested by Sutarman and Thomson (1952).

Plastic paint was applied to the chosen area of skin and allowed to dry for a period of 20 to 30 seconds; the thin layer of plastic was then detached from the skin by means of a small strip of adhesive tape. The cast thus obtained was then transferred to a labelled microscope slide and examined under the low power of a light microscope. The plastic paint used consisted of a 2/4% (w/v) solution of polyvinyl formal (Formvar) in ethylene dichloride containing 1% butyl phthalate as plasticizer.

The experimental procedure was as follows: subjects were tested singly or in groups of two; a total of six plastic casts were taken in each subject from the same area of skin. The first cast was taken at the beginning of the experiment; the second cast was taken immediately before a period of self-induced stress, the third cast was taken immediately after the stress and the final three were taken at subsequent five minute intervals. The self-induced stress consisted of straight-leg raising to the point of maximum endurance; the total time the leg was kept in the air was not considered to be relevant as the main object was to make the subjects drive themselves to the limits of endurance and these limits were expected to vary with age, sex and general physical condition.

The series of impressions thus obtained were examined under the low power of a light microscope and the number of active sweat glands over an area of skin 4mm square was counted; active sweat glands were easily differentiated from inactive glands in that the former give rise to distinct round holes in the cast whereas the latter present a crinkled, closed appearance. The number of active sweat glands over this area was taken as the palmar sweating index and the changes in the palmar sweating index as a result of the self-induced stress were taken as a

representation of the palmar anhidrotic response to stress.

The 21 female subjects were tested during the follicular phase of the menstrual cycle; this was done in order to avoid any effects of circulating progesterone on the PSI — it has been shown by Harrison *et al.* (1962) that progesterone tends to lower the level of PSI.

Clinical Rating: In order to obtain a measure of the patient's clinical condition at the time of testing, each subject in the schizophrenic groups was read by the charge nurse or ward sister or, in the case of the out-patients by the experimenter with the cooperation of the families of the patients, on a withdrawal rating scale consisting of items 6, 7, 9 and 10 from the scale devised by Venables (1957) and items 2, 4, 6, 8 from the scale devised by Venables and O'Connor (1959).

In order to obtain an indication of the degree of morbidity both before and during the illness, each patient was rated on items A to F from the Prognostic Rating Scale devised by Phillips (1953); rating was carried out by the experimenter from data obtained from the case notes.

Results

The Palmar Anhidrotic Response: Changes in the PSI were taken as indications of the nature of the PAR; there were differences in the PSI response to stress between the in-patient group and the out-patient group and between the in-patient group and the group of normal controls; there seemed to be no qualitative difference in the PAR between the out-patient group and the control group.

Figure 1 shows the pattern of PSI change before and after the period of self-induced stress; it can be seen that there was a general tendency towards a paradoxical rise in the PSI after stress in the group of in-patients and that the response in the out-patient group was similar to the anhidrotic response shown by the control group.

Figure 2 shows the distribution of the PSI responses in the three groups; it can be seen that in the group of the in-patients

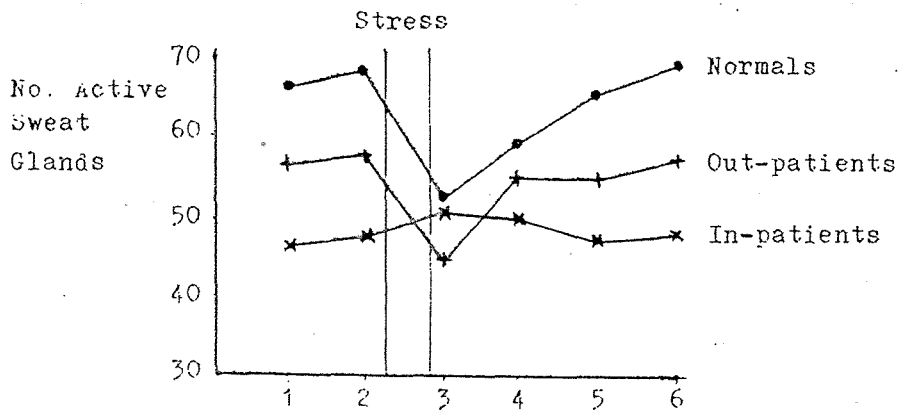


Figure 1 : The pattern of PSI change over time. 1 = Basal, 2 = pre-stress, 3 = post-stress, 4, 5 & 6 = readings taken at 5 min. intervals after stress.

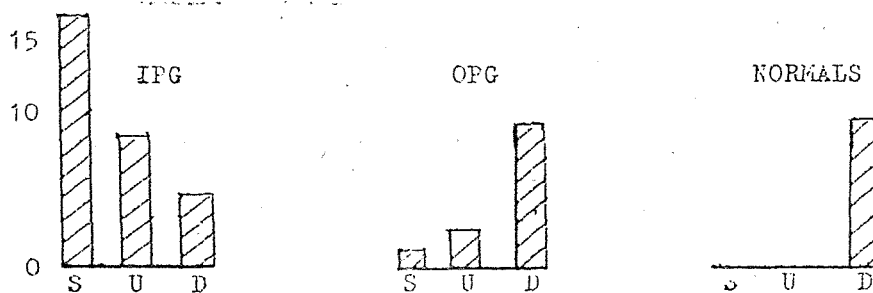


Figure 2 : Distribution of the PAR patterns in the three groups.

S = No change in the PSI after stress

U = Paradoxical rise in the PSI after stress

D = Normal palmar anhidrotic response to stress

there was a paradoxical rise in the PSI after stress in 8 subjects, a fall in 4 subjects and no change in 16 subjects; in the out-patient group there was a paradoxical rise in the PSI in 2 subjects, a fall in 9 subjects and no change in 2 subjects; there was a fall in the PSI after stress in all the controls. The criterion adopted for "no change" was a rise or fall in the PSI of less than 4% of the immediately preceding (pre-stress) level.

An analysis of variance of the PSI over times showed that the changes seen in the group of in-patients were not significant and changes in the group of out-patients were significant only if the analysis of variance of the PSI was carried out over the first three readings; this was

probably due to the fact that the two patients showed a delayed paradoxical response, which may also account for the rapid return of the PSI to pre-stress levels in this group.

Inter-group differences in the basal level of the PSI were also noted; the in-patient group showed a lower basal PSI than the out-patient group and the difference in the mean values was significant ($t = 1.983$, $p < 0.10$); significant differences in the mean basal PSI were also seen between the group of in-patients and the controls ($t = 5.978$, $p < 0.001$) and between the group of out-patients and the group of controls ($t = 2.242$, $p < 0.05$).

Clinical Rating: The mean scores in the two patient groups on the withdrawal

and prognostic rating scales are shown in Table 2. Higher scores on both of these scales are indicative of a greater degree of morbidity.

TABLE 2

	Withdrawal	Prognostic Rating
In-patients	15.32	19.64
S.D.	4.97	4.50
Out-patients	11.08	14.42
S.D.	2.15	4.68

The difference in the mean scores between the two groups were significant for both the withdrawal rating scale ($t = 2.894$, $p < 0.01$) and for the prognostic rating scale ($t = 3.27$, $p < 0.01$).

Drugs: In order to assess the effects of differences in the level of drug between the two patient groups on the PSI, the mean drug levels were t-tested and the differences were found to be non-significant.

Discussion

There has been only one reported study of the PAR to stress in schizophrenic patients (Mackinnon, 1969). The findings of this experiment confirm the general notion suggested by Mackinnon that the response of the palmar sweat glands to stress in schizophrenics differs from that seen in normals. As in Mackinnon's study, the basal PSI was lower in schizophrenics than in normals and there was a tendency towards a paradoxical rise in the PSI after a self-induced stress in some schizophrenic patients.

The cause of the low PSI level in schizophrenia is not clear and notions on its significance can only be speculative; it has been suggested that there is a high correlation between the number of active sweat glands and the basal skin conductance level (Thomas and Korr, 1957) but Martin and Venables (1966) have reported a much lower correlation (0.40, $p < 0.05$) and have emphasised that sweat glands

counts are spot counts and have the added disadvantage of not including partially filled ducts, which are known to contribute significantly to the level of skin conductance — on the other hand, however, the electrical properties of the skin are known to be derived in part from non-sudorific elements in the skin. The finding of a lower PSI in schizophrenics would suggest, however, that lower levels of skin conductance are to be found in schizophrenics than in normals; skin resistance studies by Howe (1958), Malmo and Shagass (1949a) and Zahn (1964) have been inconclusive as there has been evidence of both higher and lower levels of skin resistance in schizophrenics than in normal controls.

The salient feature in the investigation of the PAR in this study was that the anhidrosis observed in normals after a period of self-induced stress was either absent or substituted by a paradoxical increase in the number of active sweat glands in the immediate post-stress period in the group of in-patients, while the PAR in the group of out-patients was similar to that of the control group.

The group of in-patients was composed mainly of institutionalised patients in whom all attempts at rehabilitation had failed; such patients could be categorised as poor pre-morbid schizophrenics on the basis of outcome and, indeed, the mean score on the prognostic rating scale was significantly higher in this group than in the group of out-patients, who can be considered as good pre-morbid schizophrenics.

The invariant or paradoxical PAR in the patients can be taken as an index of autonomic disorganisation, and, as the findings from the rating scales suggest, it is likely that those patients who are relatively refractory to treatment and who are characterised by high chronicity, high withdrawal and a poor pre-morbid personality are also subject to some degree of automatic disorganisation.

Mackinnon interpreted her findings as lending support to Venables's (1967) theory of failure in the inhibitory section of the reticulo-activating system in schizo-

phrenia; since the central control of sweating involves excitatory and inhibitory influences from the hypothalamus, basal ganglia, frontal and sensorimotor cortex and a host of other structures, and in view of the effect of circulating progesterone on the PSI, it would perhaps be of value to include such structures in the formulation of a theory of imbalance among those structures concerned with the mediation of autonomic responses.

Acknowledgements

This study was carried out at Littlemore Hospital, Oxford, and financed by the Rhodes Trust and by the Nuffield Foundation. Thanks are due to Dr. F. J. Letemendia for permission to test patients under his care and for his useful advice on the experimental design; thanks are also due to Professor M. G. Gelder for his help, encouragement and advice.

- DMITRIEV L., BELYAKOVA L.I., BONDARENKO T. & NIKOLAEV G. (1968) *Zh. Nevropat. Psikhiat. Korsakov* 68, 713.
- GUNDERSON E. (1953) unpublished doctoral dissertation — Univ. of California, Los Angeles.
- HARRISON J. (1964) *J. Psychosom. Res.* 8, 187.
- HARRISON J., MACKINNON P.C.B., & MONK-JONES M.E. (1962) *Clin. Sci.* 23, 371.
- HARRISON J. & MACKINNON P.C.B. (1966) *J. Appl. Physiol.* 21, 88.
- HELD J.H., CROMWELL R.L., FRANK E.T. JNR. & FANN W.E. (1970) *J. Psychiat. Res.* 7, 209.
- HOWE E.S. (1958) *J. Abn. & Soc. Psychol.* 56, 183.
- MACKINNON P.C.B. (1969) *J. Psychiat. Res.* 7, 1.
- MACKINNON P.C.B., MACKINNON I.L., & WILLIAMS E.S. (1959) *Br. J. Med.* 1, 199.
- MALMO R. & SHAGASS C. (1949a) *Psychosom. Med.* 11, 9.
- MARTIN I. & VENABLES P.H. (1966) *Psychol. Bull.* 65, 347.
- PHILIPPS L. (1953) *J. Nerv. Ment. Dis.* 117, 515.
- SUTARMAN & THOMSON (1952) *J. Physiol.* 117, 51P.
- THOMAS P. & KORR I. (1957) *J. Appl. Physiol.* 10, 505.
- VENABLES P.H. (1957) *J. Ment. Sci.* 103, 197.
- VENABLES P.H. (1967) *Excerpta Medica Int. Congress Series No.* 151.
- VENABLES P.H. & O'CONNOR N. (1959) *J. Ment. Sci.* 105, 815.
- WHATMORE G.B. & ELLIS R.M. (1958) *Amer. J. Psychiat.* 114, 882.
- ZAHN T.P. (1964) *Psychiat. Res. Reports* 19, 156.
- ZAHN T.P., ROSENTHAL D., & LAWLOR W.G. (1968) *J. Psychiat. Res.* 6, 117.