Brit. J. Psychiat. (1968), **114**, 639–642

Some Observations on the Psychogalvanic Reflex

By ABRAHAM VERGHESE

The psychogalvanic reflex (P.G.R.) is a decrease in the electrical resistance of the skin which occurs in response to a wide variety of stimuli.

In 1888, Féré, a French clinician, found that pain of any sort caused a fall in skin resistance, and in 1890 Tarchanoff, a Russian physiologist, discovered that the fall in skin resistance was associated with a change in skin potential. Veraguth, a Swiss neurologist, investigated this phenomenon and named it the psychogalvanic reflex. In 1922, however, Gildemeister, a German physiologist, maintained that this phenomenon should be called the galvanic skin reflex, as it was not purely a psychological phenomenon (Wang, 1957).

Ever since the discovery of the P.G.R., psychologists have been interested in its application in psychological research. Jung (1907) used it in his investigations to find out the role of association tests in the diagnosis of schizophrenia. Peterson (1907) reported that the P.G.R. was a good measure of emotion. Other workers also reported similar findings (Starch, 1910; Cattell, 1929). Wechsler, in 1925, published his monograph on *The measurement of emotional reactions* which was based on his work on the P.G.R. Wagner (1950) used the P.G.R. as an objective test for the diagnosis of hysterical blindness. Bordley *et al.* (1948) used it as a test for hearing. Reports on the role of the P.G.R. in differentiating psychiatric conditions are conflicting (Silverman and Powell, 1944; Malmo and Shagass, 1949; Wenger, 1948; Eysenck, 1956; Jurko *et al.*, 1952). During the course of a psychosomatic study of chest pains after myocardial infarction it was found that of all the autonomic parameters only the P.G.R. had a positive correlation with the degree of neuroticism, as measured by the Eysenck Personality Inventory and the Cornell Medical Index (Verghese, 1966).

A full review of the P.G.R. from a physiological standpoint was made by Wang (1957, 1958). Two theories have been proposed to explain the phenomenon. The vascular theory, proposed by Féré and elaborated by McDowall (1933) maintained that it was caused by a change in the tone of the blood vessels of the skin. The second theory, advanced by Tarchanoff and supported by Darrow (1927) suggested that it was due to changes in the secretory activity of the sweat glands of the skin. Subsequent experimental evidence has tended to support Tarchanoff's theory (Wang, 1957).

Experiments on animals have shown that the P.G.R. is mediated through the cholinergic fibres of the sympathetic nervous system. In the cat, sympathectomy abolishes the P.G.R. (Wang, 1957). This has not been demonstrated in man. It was thought that if sympathectomy abolished the P.G.R. in man, a sympathectomized limb might be used to study the chemical mediators of the reflex. The purpose of this paper is to report the effects on the P.G.R. of infusions of adrenaline, noradrenaline and acetylcholine into the brachial artery of a subject who had had bilateral cervical sympathectomy.

Method

The subject was a female, 25 years old, who had had bilateral cervical sympathectomy three weeks previously for long-standing Raynaud's disease.

An Offner Type 9892 A skin resistance coupler and chlorided silver electrodes, concave and 1 cm. in diameter were used.

To examine the effect of sympathectomy on the P.G.R. the electrodes were securely fastened, one to the centre of each palm, by adhesive tape, after cleaning the skin with absolute alcohol. Electrode paste was not used, since it alters the skin resistance. A third earthing electrode was fixed to a finger, for which electrode jelly was used (Beckman Offner paste). A continuous tracing of the P.G.R. was taken when the subject was relaxing and during the application of three stimuli—a loud sound, thermal pain induced with a pain-threshold apparatus

https://doi.org/10.1192/bjp.114.510.639 Published online by Cambridge University Press

similar to the Hardy-Wolff-Goodell Dolorimeter, and mental arithmetic. The electrodes were then changed from the hands to the feet, one electrode being fixed to the sole of each foot after cleaning the skin with absolute alcohol. The earthing electrode was not changed. The experiment was repeated as before.

To examine the effect of intra-arterially infused substances, the two electrodes were removed from the feet and fixed to the left palm. A Cournard needle was inserted into the left brachial artery in the anticubital fossa and remained *in situ* throughout the experiment. Through it, either 0·9 per cent. saline or a solution in saline of the drug to be tested was infused at 0.4 ml. per minute, using a mechanically propelled syringe. The drugs used were noradrenaline 0.4 μg./minute); adrenaline (0·4 μg./minute); and acetylcholine (20 μg./minute). Noradrenaline, adrenaline and acetylcholine were infused separately for 5-minute periods. Before and after each infusion, a 10-minute infusion of saline was given. During the experiment, the P.G.R. and pulse rate were recorded continuously.

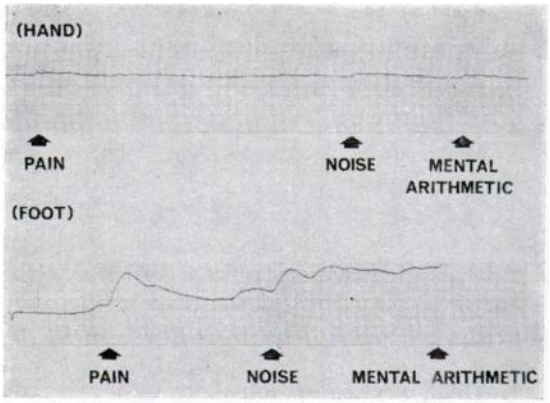


FIG. 1.—An upward deflection denotes a fall in skin resistance. (1 division=80 kilo-ohms.) 1 division (horizontally) represents 1 second.

There were no responses from the hand (upper tracing) while the foot showed normal P.G.R. responses (lower tracing).

RESULTS

Fig. 1 shows that before the infusion of the drugs the P.G.R. could be obtained from the feet, but not from the hands.

Fig. 2 shows that when noradrenaline infusion was started, there was a slight decrease in skin resistance, which was seen during the infusion of adrenaline also. When acetylcholine was started, there was a marked fall in skin resistance. The measurements are summarized in the Table.

During the infusion of both adrenaline and noradrenaline, the left hand became cold and white, the pallor disappeared when the drug was stopped and the saline infusion started.

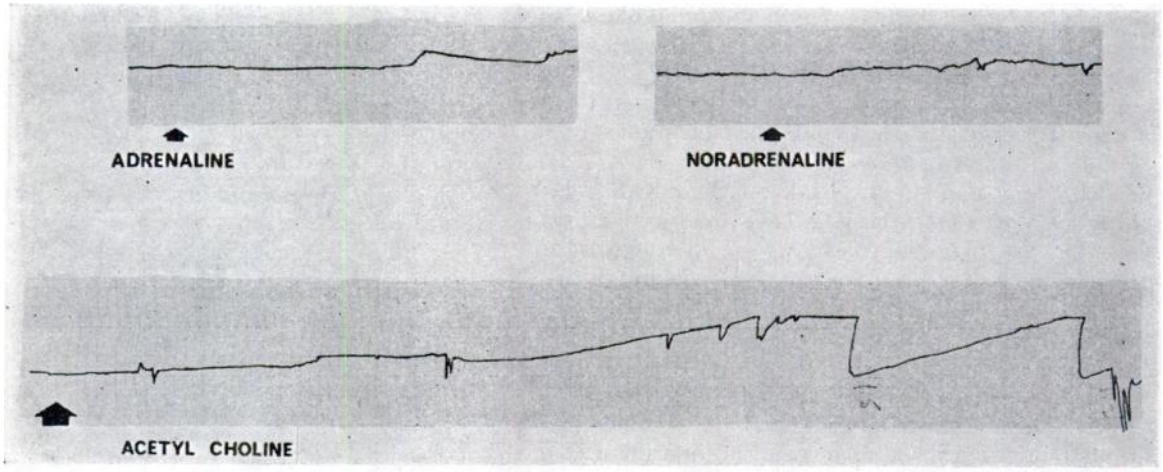


FIG. 2.—An upward deflection denotes a fall in skin resistance. (1 division=80 kilo-ohms.) 1 division (horizontally) represents 1 second.

While acetylcholine produced a marked reduction in skin resistance, adrenaline and noradrenaline did not. The change in skin resistance with acetylcholine was so marked at points marked 1·5 and 1·2, that the pen had to be returned to the baseline.

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TABLE I

Drug	Amount infused	Maximum fall in skin resistance (kilo-ohms)	Clinical observation
Noradrenaline ..	2·0 μg.	100	Hand became cold and white
Adrenaline	2·0 μg.	80	,,
Acetylcholine	100 μg.	960	Sweating of hand

During the infusion of acetylcholine the subject reported that her left hand was sweating; this was confirmed on inspection. Sweating ceased when the drug was stopped and the saline infusion started.

DISCUSSION

The finding that the P.G.R. was absent after sympathectomy shows that in man, as in animals, the P.G.R. involves the sympathetic pathway. The marked fall in skin resistance of the sympathectomized limb when acetylcholine was infused suggests that acetylcholine is the main substance which mediates the P.G.R. peripherally. The sweat gland activity has been shown to depend on acetylcholine release. The accompaniment of the fall in skin resistance following acetylcholine infusion by sweating further supports Tarchanoff's view that the P.G.R. depends on the activity of the sweat glands.

Lader and Montagu (1962) studied the effects of atropine and bertyllium, introduced locally by iontophoresis, on the skin resistance and the pulse volume, recorded simultaneously from the same finger. Atropine was found to abolish the P.G.R. without affecting the pulse volume. Bertyllium abolished the pulse volume without affecting the P.G.R. They concluded that the P.G.R. is dependent on the secretory activity of the sweat glands.

Sonnenschein *et al.* (1949), Wada (1950) and Chalmers and Keele (1952) reported local sweating after intradermal injection of adrenalin and noradrenaline. It is therefore possible that they may have some effect on sweat gland activity either directly or as a result of the intermediary production of acetylcholine in the metabolism of adrenaline and noradrenaline (Burn, 1963). The present study shows that if they have any effect it is very small indeed.

It would have been desirable if the P.G.R. studies were conducted before sympathectomy also, but this could not be done. One could argue that such a procedure might have provided evidence about the vascular theory of the P.G.R., since the patient suffered from Raynaud's disease. It is difficult to answer this criticism, but it is quite unlikely that this would in any way have affected the main finding of the study, that acetylcholine evoked marked P.G.R. changes in a sympathectomized limb.

SUMMARY

Some P.G.R. studies in a female subject who had bilateral cervical sympathectomy were described. It was found that sympathectomy abolished P.G.R. and that intra-arterial infusion of acetylcholine evoked marked P.G.R. changes in the sympathectomized limb. These findings support the theory that the P.G.R. is mediated through the cholinergic fibres of the sympathetic nervous system.

ACKNOWLEDGMENT

These experiments were conducted in the University of Melbourne Department of Medicine, Royal Melbourne Hospital. Professor A. E. Doyle allowed me to do these experiments on one of his patients. Dr. J. R. E. Fraser and Dr. W. J. Louis helped me with some technical aspects. The help and guidance of Professor R. R. H. Lovell, Professor B. Davies and Dr. R. Mowbray are gratefully acknowledged.

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Abraham Verghese, M.D., D.P.M., *Department of Psychiatry, Christian Medical College & Hospital, Vellore, India*

(*Received 22 May, 1967*)

https://doi.org/10.1192/bjp.114.510.639 Published online by Cambridge University Press