SUDOMOTOR REACTION IN NEUROSYPHILIS*

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Clinical evidence of impaired autonomic function in patients with neurosyphilis has slowly accumulated over the years, but there has been little systematic investigation into this problem. Symptoms suggestive of autonomic defects have been reported in tabes dorsalis, and include postural hypotension (Strisower, 1933; Ellis and Haynes, 1936; Spingarn and Hitzig, 1942), visceral analgesia (Connor, 1910; Hanser, 1919; Grimble and Csonka, 1952), and sexual and sphincter dysfunction. Visceral crises are usually ascribed to autonomic disturbance, but this has never been proved (Simons, 1939; Stokes, Beerman, and Ingraham, 1944; Lees, 1953; and many others).

This paper describes the sudomotor response to body heating in 24 patients with neurosyphilis and five control subjects. In man, sweat glands are distributed over the entire surface of the body and are innervated by post-ganglionic sympathetic fibres which are, however, stimulated by cholinergic drugs.

Sweating Response to Body Heating

Technique.—The patient is given 10 gr. aspirin and placed under a heat cradle with twelve 25-watt carbon filament lamps. The heat is trapped around the patient by blankets, leaving only the head free. The skin of the patient is prepared by dusting with Quinizarin powder which is gently rubbed in with cotton wool (Guttmann, 1947). Sweating is easily shown by the change in colour from the faint bluish-grey of the dry powder to dark mauve when wet. The temperature aimed at within the air envelope around the patient is 120°F.

In the normal person, generalized sweating was seen in 20–35 minutes; in some of our patients with sweating-deficiencies, the onset of sweating was delayed up to 60 min. from the start of body heating. In all cases the experiment was continued until the maximal sweat-response had been reached.

Results.—24 patients with neurosyphilis and five normal controls from St. Mary's Hospital, Paddington, and Addenbrooke's Hospital, Cambridge, were tested (Table). Some of the sweating-responses are recorded in the series of eighteen Figures (see pp. 169–171). Twenty out of 24 patients with neurosyphilis showed marked sweating-deficiencies, which suggests that the involvement of the sympathetic nervous system is not uncommon. The control subjects gave normal sweating-responses.

TABLE

Diagnosis	Age (yrs)	Sex	Oral Temperature at Start and End of Body Heating (°F.)	Sweating Response	Illustration Number
Control Subjects	28	M	97.6–99.8	Normal	1
	29	M	98.2–100.0		
	30	M	97.6–99.4		
	35	M	97.4–98.8		
	42	M	97.8–99.4		
Tabes Dorsalis	60	F	98.0–100.2	Deficient	2
	66	F	98.0–100.0		3
	51	M	97.2–99.4		4
	42	M	97.8–99.6		5
	49	F	97.6–99.8		6
	40	M	97.0–99.8		7
	66	M	97.8–100.2		8
	46	F	97.2–99.4		9
	48	M	98.0–99.4		10
	61	F	98.0–99.6		11
	50	M	97.4–99.4		12
	57	M	97.8–99.6		—
	48	M	97.8–99.8		—
	58	M	97.2–99.6		—
	56	M	97.6–100.0	Normal	—
	50	M	98.0–100.0		—
	60	F	97.8–99.4	—	—
Taboparesis ..	54	M	97.2–99.4	Deficient	13
	73	M	97.0–99.0		14
Meningovascular Syphilis ..	53	M	97.6–99.4	Deficient	15
	49	M	98.2–100.2		16
	58	M	97.4–99.6		17
Minimal Neurological Abnormalities	54	M	97.2–100.0	Deficient	18
General Paresis (congenital) ..	18	M	97.8–99.4	Normal	—

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Normal sweating
 Patchy sweating
 No sweating

Certain features were noteworthy:

- (1) The sweating-defects were strictly symmetrical in fifteen patients and asymmetrical in five. This latter group contained the only two patients with Charcot joints, and it was found that an area including the affected joints showed a more marked sweating-deficiency than the corresponding part on the other side.
- (2) There were fourteen patients with marked lightning pains; four of these showed normal sweating in the limbs affected by pain and ten gave diminished or totally absent sweating-responses which included the painful areas. Two patients were of special interest as their lightning pains were localized in one lower limb only, which was also more deficient in sweating-response than its fellow.

Sweating-Response to Local Heat

In order to test the functioning of the sweat glands themselves in areas in which they failed to respond to general body heating, local intense heat was applied, as it has been shown that intact sweat glands will respond to

this stimulus even if they are denervated (Janowitz and Grossman (1950); Bárány and Cooper, 1956). Hot air from an electric hair-drier was directed on to the selected skin area from a distance of 6 in. for 2 to 3 min. and the skin tested for sweating. In five out of six patients this direct heating produced sweating where body heating failed to do so.

Conclusions

The sweating experiments show that there is impaired sympathetic function in many patients with neurosyphilis. The site of the lesion has yet to be determined, but further investigations with neuro-effector drugs are in progress and will be reported elsewhere. There was no clear-cut correlation between sensory loss and sympathetic dysfunction, and further experience is needed to establish what relationship exists, if any, between autonomic defect and such neurosyphilitic manifestations as Charcot joints, lightning pains, and visceral crises.

Summary

(1) 24 patients with neurosyphilis were investigated for evidence of autonomic nerve involvement by means of the sweat response to general body heating.

(2) Twenty patients showed marked sudomotor deficiency which was symmetrical in fifteen and asymmetrical in five cases. This suggests that the sympathetic pathway is commonly involved in neurosyphilis.

(3) Abnormal sweat responses were obtained in tabes dorsalis, taboparesis, and chronic meningovascular syphilis, and were absent in the single patient with congenital general paresis. The control subjects gave normal responses.

(4) Clinical data are briefly discussed in relation to sweating reactions.

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