Summary

The methods adopted to establish a blood bank in Cyprus are described.

The ABO frequencies of the two main racial stocks in Cyprus are so similar as to suggest a common ethnic derivation.

The ABO frequencies in Cyprus resemble those of the inhabitants of the adjacent mainland rather than those in Greece. The resemblance is so close as to suggest that Cypriots are ethnically related to their neighbours on the Asiatic mainland.

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REFERENCES

- Bliss, F. J. (1912). *The Religions of Modern Syria and Palestine*, Edinburgh, T. & T. Clark.
 Hill, Sir G. (1952). *A History of Cyprus*, Vol. IV. University Press, Cambridge.
 Luke, Sir Harry (1957). *Cyprus, a Portrait and an Appreciation*. Harrap, London.
 Mourant, A. E. (1954). *The Distribution of the Human Blood Groups*. Blackwell Scientific Publications, Oxford.
 — Kopeć, Ada C., and Domaniewska-Sobczak, Kazimiera (1958). *The ABO Blood Groups*. Blackwell Scientific Publications, Oxford. In press.
 Nassif, R. E. (1953). *J. méd. liban.*, 6, 346.
 Storrs, Sir Ronald (1949). *Orientalism*. Definitive Edition. Nicholson and Watson, London.

THE MECHANISM OF POST-PAROTIDECTOMY GUSTATORY SWEATING (THE AURICULO-TEMPORAL SYNDROME)

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With a Note on Surgical Considerations by
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Gustatory sweating—that is, sweating during the eating of food—may occur in the distribution of the auriculo-temporal nerve following parotidectomy, penetrating wounds of the parotid, or suppurative parotitis. It may be associated with flushing of the skin in the same region, though in the cases that we have seen sweating has been the predominant feature and flushing minimal or absent. The syndrome does not develop until some months after the operation or injury, and at times only after some years. Once developed, it appears to persist indefinitely. The degree of sweating varies from a slight dampness to sweating profuse enough to be a minor social embarrassment.

The earliest recorded case is attributed to Duphénix (1757), who is stated to have described the syndrome following drainage of a parotid abscess. Later, Baillarger (1853) recorded a case in which the condition occurred bilaterally after bilateral parotitis, and Weber

(1897–8) another following suppurative lymphadenitis in the parotid region. Frey (1923) recorded a case following an infected bullet wound of the parotid, and noted that the sweating corresponded to the cutaneous distribution of the auriculotemporal nerve. The syndrome has since been known as the “auriculotemporal syndrome” or “Frey’s syndrome.”

The reported frequency of the condition varies, probably depending on the thoroughness with which minor examples are searched for and on the length of follow-up. Freedberg *et al.* in 1948 were able to collect only 35 cases from the literature, but with the increase in parotid surgery in the last decade the condition is now recognized to be common. In a personal series (D.H.P.), 15 out of 42 patients had noted sweating after six months. Moyse (1955) noted its presence in 20 out of 25 patients five years after operation. Laage-Hellman (1957) noted some degree of gustatory sweating or flushing in all but 3 of 123 personal cases.

We have studied 16 selected post-parotidectomy patients, two in detail and 14 in less detail, with the object of eliciting the mechanism and pathways by which gustatory sweating is produced. 10 of the 16 patients showed gustatory sweating. The paper reports the results of our studies. The two cases recorded in detail are:—Case 1: a man of 71 who first noticed profuse gustatory sweating nine months after a left superficial parotidectomy for adenolymphoma; a minute or so after the taking of food, beads of sweat formed on the left temporal region and ran down towards his neck; there was no flushing. Case 2: a man of 58 who first noticed gustatory sweating six months after a left partial parotidectomy; sweating was less profuse than in Case 1; there was no flushing.

Methods of Investigation

Two standard gustatory stimuli were used, which were found to give repeatable responses: (a) chewing moistened cotton-wool; (b) sucking an acid drop. The latter was the stronger stimulus. Both methods elicited sweating in positive cases within half a minute, but to obtain the maximum response records were not made until after two minutes.

A permanent record of sweat-gland activity was obtained by the method of Thomson and Sutarman (1953). The area of skin to be studied was cleaned with surgical spirit, and a plastic impression paint containing colloidal graphite was applied evenly with a camel-hair brush. When dry (15–30 seconds) the paint was stripped off with “sellotape” and mounted on a glass microscope slide. Direct photographic enlargements were made from the slides. In these positive prints active sweat glands are shown as black dots, the size and number of the dots being a measure of sweat-gland activity.

Anatomical Considerations (Fig. 1)

The parotid gland is innervated by parasympathetic secretomotor fibres and by vasomotor fibres of the sympathetic nervous system. The nerve supply to the sweat glands of the head and neck is also sympathetic in origin. The parasympathetic nerves to the parotid gland arise in the upper part of the medulla and leave the skull in the glossopharyngeal nerve. They leave this nerve in its tympanic branch which passes back into the cranial cavity to form the lesser superficial petrosal nerve. This nerve passes through the foramen ovale to reach the otic ganglion, where its fibres relay. The post-ganglionic fibres join the auriculotemporal nerve, and hence pass into the parotid gland.

The sympathetic nerves to the head and neck arise in the upper two thoracic segments and pass up in the cervical chain to relay in the superior cervical ganglion. The post-ganglionic fibres form a plexus around the external carotid artery and its branches, and hence reach the auriculotem-

FIG. 1.—Nerve supply to parotid gland and sweat glands. V, trigeminal nerve; IX, glossopharyngeal nerve; T.P., tympanic plexus; S.C.G., superior cervical ganglion; L.S.P.N., lesser superficial petrosal nerve; Otic G., otic ganglion; A.T.N., auriculotemporal nerve.

poral nerve, from which they are distributed both to the parotid gland and to the sweat glands of the temporal region.

The auriculotemporal nerve thus contains both sympathetic and parasympathetic fibres which can be differentially blocked by injections of local anaesthetic before they join the nerve. The functions of the two types of fibre can in this way be differentiated.

The Afferent Pathway

Anaesthetization of the mouth with amethocaine reduced but did not abolish gustatory sweating in response to the two standard stimuli. Sweating could be elicited in the absence of jaw or tongue movements by placing in the mouth a wad of cotton-wool soaked in lemon juice. This effect was abolished by previous anaesthetization with amethocaine. It was concluded that the reflex arises in sensory endings both in the mouth and in the muscles of mastication.

Pharmacological Responses of the Involved Sweat Glands

The behaviour of the sweat glands in the affected area was studied following intradermal administration of acetylcholine and atropine. The responses in all cases were compared with those of normal sweat glands in the corresponding region on the other side of the face.

(a) *Acetylcholine*.—Acetylcholine was injected intradermally in dilutions varying from 10^{-5} to 10^{-7} (that is, 10 to

$0.1 \mu\text{g./ml.}$). In Case 1 an injection of $0.05 \text{ ml. } 10^{-5}$ acetylcholine ($0.5 \mu\text{g.}$) produced marked sweating over the injection bleb on the affected side; the same dose on the opposite side evoked no response (Fig. 2). The minimum dose of acetylcholine necessary to produce sweating was determined in all 16 patients studied (see Table). Of the 10 patients with gustatory sweating, 8 showed an increased sensitivity on the affected as compared with the normal side of the order of 10 to 100 times. Of the 6 patients without gustatory sweating, only one showed an increased sensitivity to acetylcholine.

(b) *Atropine*.—Intradermal injections of $0.2 \text{ ml. } 10^{-4}$ atropine ($20 \mu\text{g.}$) (Cases 1 and 2) abolished gustatory sweating in a localized area around the injection site. An injection of the same bulk of saline had no effect, thus excluding a mechanical explanation.

The Efferent Pathway

The auriculotemporal nerve was blocked in front of the tragus with lignocaine and adrenaline (Case 2). This abolished gustatory sweating. If the nerve fibres mediating gustatory sweating are distributed with sensory fibres in the auriculotemporal nerve, then blocking of this nerve should not only abolish sweating in the temporal region but should also produce skin anaesthesia in the same area. This was found to be the case. These findings were later confirmed by avulsion of the auriculotemporal nerve, which abolished gustatory sweating and left a similar area of anaesthesia. The findings confirm the well-accepted fact that the final path of the efferent arc of the gustatory sweating reflex runs in the auriculotemporal nerve.

The auriculotemporal nerve contains both sympathetic and parasympathetic components. Either component might mediate the gustatory sweating reflex. Further study was made by means of differential nerve blocks.

(a) *Sympathetic Block*.—Case 2:—The left cervical sympathetic chain was blocked with 2% lignocaine and adrenaline at the level C4-5 and at the stellate ganglion. A complete Horner's syndrome was produced, but gustatory sweating was unaffected. Case 1:—Simple stellate ganglion block confirmed these findings. From this it was concluded that gustatory sweating was not mediated by the sympathetic pathway responsible for normal thermal sweating. The possible role of the parasympathetic nervous system was therefore investigated.

(b) *Parasympathetic Block*.—The otic ganglion lies superficial to the mandibular nerve just below the foramen ovale. At this point it is theoretically possible to block parasympathetic fibres alone (see "Anatomical Considerations" and Fig. 1). Case 2:—A needle was introduced above the ramus of the left mandible between the condyle and the coronoid process, and directed medially and upwards until it reached the base of the skull. The patient at this point complained of pain in the jaw, indicating that the needle was in contact with the mandibular nerve. 10 ml. of lignocaine with adrenaline was injected. Within a few minutes there was anaesthesia of the skin over the mandible, and some dullness to pin-prick in part of the auriculotemporal distribution. Gustatory sweating was abolished (Fig. 3, a and b). To demonstrate that this was not due to spread of the block, the patient was heated to induce thermal sweating. Sweating thereupon occurred over the whole body, including the gustatory sweating area (Fig. 3, c). Further tests for gustatory sweating showed that the block persisted for several hours.

From this it was concluded that the fibres concerned in the gustatory sweating reflex pass through the otic ganglion, and are thus similar to those mediating normal gustatory salivation—that is, parasympathetic.

Response to Heating

A heat cradle was placed over the patient (Cases 1 and 2) with a blanket over the cradle to prevent heat reaching the face. Within 30-50 minutes the body temperature was

Table Relating Sensitivity of Sweat Glands to Intradermal Injections of Acetylcholine to the Presence of Gustatory Sweating

Case No.	Gustatory Sweating	Concentration of Intradermal Acetylcholine Producing Sweating		Increase in Sensitivity on Side of Opn
		Control Side	Side of Opn	
1	+	10^{-5}	10^{-7}	100 times
2	+	10^{-5}	10^{-6}	10 "
3	+	10^{-5}	10^{-7}	100 "
4	+	10^{-6}	10^{-7}	10 "
5	+	10^{-6}	10^{-7}	10 "
6	+	10^{-6}	10^{-7}	10 "
7	+	10^{-6}	10^{-7}	10 "
8	+	10^{-6}	10^{-7}	10 "
9	+	10^{-7}	10^{-7}	1 "
10	+	—	10^{-6}	—
11	—	10^{-5}	10^{-6}	10 "
12	—	10^{-6}	10^{-6}	1 "
13	—	10^{-6}	10^{-6}	1 "
14	—	—	10^{-6}	—
15	—	—	10^{-6}	—
16	—	—	10^{-6}	—

raised by 1.5° F. (0.8° C.), by which time generalized sweating had occurred. Sweating occurred in both temporal regions, but was less on the affected side.

Discussion

Gustatory sweating in the area of distribution of the auriculotemporal nerve following lesions in the region of the parotid gland must be distinguished both from the sweating which may occur around the lips, nose, and on the forehead in some normal people on eating spicy foods; and from the gustatory sweating which occasionally follows sympathectomy (List and Peet, 1938; Haxton, 1948; Herxheimer, 1958). Gustatory sweating analogous to the auriculotemporal syndrome may also occur in the submandibular region following operation on or near the submandibular gland (Uprus *et al.*, 1934; Young, 1956).

Several theories have been proposed to explain the auriculotemporal syndrome, as follows.

Irritation of the Auriculotemporal Nerve.—Frey (1923) and Needles (1936) suggested that sudomotor fibres are irritated, as they pass through scar tissue in the parotid region, by jaw movements or by swelling of the parotid gland.

Diffusion of a Choline-like Substance.—List and Peet (1938) suggested that acetylcholine or some choline-like substance diffuses from the cut surface of the parotid gland and stimulates sweat glands in the overlying skin.

Hypersensitivity.—Freedberg *et al.* (1948) suggested that the phenomenon might represent the response of denervated and thus hypersensitive sweat glands to "previously unapparent cranial sudomotor impulses."

Aberrant Regeneration.—Ford (1933) postulated that, following damage to the auriculotemporal nerve, aberrant regeneration takes place so that parasympathetic fibres previously innervating the parotid gland come to innervate sweat glands.

Transaxonal Excitation.—Chorobski (1951), discussing the comparable phenomenon of "crocodile tears," suggested that both it and the auriculo-temporal syndrome might be due to "cross talk" between parasympathetic and sympathetic nerve fibres whose insulating sheaths had been damaged.

Frey's theory can be excluded, since gustatory sweating can be elicited by a taste stimulus (lemon juice) in the absence of jaw or tongue movements. The theory of a diffusion of a choline-like substance from the

parotid gland can also be excluded, since the syndrome may occur after total parotidectomy.

The theory of hypersensitivity warrants more detailed consideration. That there is an increased sensitivity to acetylcholine of the sweat glands concerned in the auriculo-temporal syndrome has been demonstrated by many workers (List and Peet, 1938; Freedberg *et al.*, 1948; Hogeman, 1951), and confirmed in the present study. The hypersensitivity has been assumed to be due to sympathetic denervation, but, since it does not

FIG. 2.—Case 1. Sweating evoked (black dots) by local intradermal injection of 0.05 ml. 10^{-5} acetylcholine into (a) the auriculotemporal area on the side of the operation, and (b) an equivalent area on the normal side.

FIG. 3.—Case 2. Records of sweating taken from the same region of the affected auriculotemporal area. (a) Gustatory sweating; (b) absence of gustatory sweating in same area following otic ganglion block; and (c) thermal sweating in same area during otic ganglion block.

occur after formal sympathectomy, either pre- or post-ganglionic (Gardner and McCubbin, 1956), we think it is perhaps better explained by partial sympathetic denervation, the existence of which is shown in these studies by the diminished response of the sweat glands concerned to thermal stimulation. The theory of hypersensitivity by itself is, however, insufficient as an explanation of gustatory sweating, since it fails to explain the dissociation between the responses of the sweat glands to gustatory and thermal stimuli.

We are thus left with the theories of aberrant regeneration and of transaxonal excitation, which are alike in that both postulate that nerve impulses of parasympathetic origin reach sweat glands (Fig. 4). Both would fit the anatomical

of the glossopharyngeal nerve. A functional connexion between parasympathetic nerve fibres and sweat glands is feasible, since post-ganglionic fibres to sweat glands are cholinergic.

If parasympathetic fibres can activate sympathetic structures, then theoretically the converse might occur—that is, sympathetic fibres might activate parasympathetic structures, and the parotid gland obtain a functional connexion with sympathetic fibres formerly innervating sweat glands. In this case a rise in body temperature should produce "thermal salivation." Case 2, a case of partial parotidectomy in which the parotid duct and approximately half of the parotid gland were still present, was suitable for the investigation of this possibility.

FIG. 4.—Diagram showing theoretical mechanisms of production of gustatory sweating in the auriculotemporal syndrome. Sympathetic fibres (S), continuous line. Parasympathetic fibres (P), broken line.

facts and the experimental observations noted in this study. The auriculotemporal nerve is peculiarly constituted in that, in addition to its sensory fibres, it contains both parasympathetic and sympathetic fibres which leave it to supply adjacent structures. These fibres might be damaged by operation or inflammation in the neighbourhood, and parasympathetic secretomotor fibres at least must be divided when parotid tissue is excised. The rare cases in which gustatory sweating occurs within the area of the great auricular nerve (Hogeman) could be explained by the presence of communicating branches between it and the auriculotemporal nerve. The effects of sympathetic and parasympathetic blocking noted in these studies show that the parasympathetic constitutes the efferent path of the arc, and that parasympathetic fibres must have obtained a functional connexion with sweat glands, which are sympathetic end-organs. This conclusion is confirmed by the observations of Gardner and McCubbin, who cured the auriculotemporal syndrome in two patients by intracranial section

Experiment

Saliva was collected simultaneously from both parotid ducts and the effects of gustatory and thermal stimulation were investigated. The results are shown in Fig. 5. Sucking an acid drop produced a marked flow of saliva, the flow from the left gland being about half that from the right; this was to be expected, since part of the left gland had

FIG. 5.—Salivary flow from the right normal parotid (white columns) and left operated parotid (black columns) in response to gustatory stimulation (periods G and G') and to a rise in body temperature (period Heat).

been removed. When this response had died away, the patient was heated in a hot-air cradle. The flow of saliva from the normal gland remained at a low level, but that from the operated gland increased until it was twice that from the normal gland. A second gustatory stimulus produced a repetition of the original response despite the continuation of heat stimulation. These results demonstrate "thermal salivation" from the remaining portion of the operated gland and no response to heat from the normal gland, a phenomenon which does not appear to have been previously demonstrated.

The studies on gustatory sweating and the demonstration of thermal salivation prove that some form of cross-connexion between parasympathetic and sympathetic fibres occurs in the auriculotemporal syndrome. On the experimental evidence available up to the present, it is not possible to differentiate with certainty between the "aberrant regeneration" theory and the "transaxonal excitation" theory, both of which could explain the observed phenomena. If the syndrome were found to persist after post-ganglionic cervical sympathectomy and degeneration of the sympathetic fibres, the cross-talk theory would be untenable; but we know of no such case. When, however, the auriculotemporal syndrome is compared with causalgia, which is accepted as due to transaxonal excitation (Granit *et al.*, 1944; Doupe *et al.*, 1944), there are significant differences. Causalgia usually arises early after damage to a nerve and tends towards spontaneous recovery. The auriculotemporal syndrome, on the other hand, does not usually develop for at least some months after the exciting cause, in our cases from three months to three years, but thereafter persists indefinitely. We therefore favour the

aberrant regeneration theory as the explanation of the auriculotemporal syndrome. The degree of hypersensitivity of the sweat glands of the affected areas is probably of importance in determining the severity of the condition once established.

Surgical Considerations

Since the sweating occurs in the distribution of the auriculotemporal nerve, the obvious treatment of this particular variety of gustatory sweating is avulsion of the auriculotemporal nerve. Unfortunately this procedure tends to be followed within a few months by recurrence of the syndrome, presumably from regeneration of nerve fibres. We have recently been trying the effect of prophylactic avulsion at the time of parotidectomy, but, though we are not yet in a position to give full results, one case of gustatory sweating has already occurred. The question arises of why auriculotemporal avulsion is so often only temporarily successful. An observation on a recent case of prophylactic avulsion may provide the answer. A total conservative parotidectomy was being performed, and after completion of the superficial phase the auriculotemporal nerve was identified and avulsed. As is our practice, the length of the main nerve trunk avulsed was measured and found to be approximately two inches (5 cm.), which was considered to be satisfactory. After the subfacial phase of the parotidectomy had been completed, however, most of the auriculotemporal nerve was found in its normal position in the parotid bed. The apparently satisfactory length of nerve avulsed was an expression of stretching rather than of efficient avulsion.

There are obvious anatomical reasons for the technical inefficiency of auriculotemporal avulsion. The deep surface of the upper pole of the parotid is bound down by dense fibrous adhesions in the region of the temporomandibular joint, and the auriculotemporal nerve runs through these adhesions. Avulsion of the nerve with the parotid or the subfacial parotid *in situ* results in breaking of the nerve at the level of these adhesions. If the procedure is carried out after the whole parotid has been removed, an efficient avulsion back to the otic ganglion should be possible. The demonstration of the technical inefficiency of auriculotemporal avulsion with the parotid *in situ* explains also why this procedure has proved unsatisfactory as a means of suppression of parotid secretion.

In view of the difficulty of dealing with gustatory sweating at the level of the auriculotemporal nerve, the question naturally arises of the possibility of an attack at other levels. Intracranial division of the glossopharyngeal nerve interrupts the parasympathetic fibres concerned, and, as already noted, this procedure has been successfully carried out by Gardner and McCubbin. The magnitude of the operative procedure as compared with the minor character of the disability would, however, seem to rule out this approach apart from very exceptional circumstances. Interruption of the parasympathetic fibres concerned would also occur if the glossopharyngeal nerve were interrupted at the base of the skull before the origin of its tympanic branch. A direct approach to the nerve at this level would be difficult, but an avulsion technique might be feasible and be worth trying. The success of the operation would depend on avulsion of the petrous ganglion.

The investigation has shown that sympathetic interruption has no effect on this type of gustatory sweating, and the final possibility is that of action at the level of the skin. X-irradiation can abolish sweating. The dosage required is, however, high, and the danger of skin damage would seem to contraindicate this approach for the treatment of sweating alone. If heavy irradiation were given for other reasons, the abolition of sweating might be expected as a subsidiary advantage. Finally, Laage-Hellman (1958) has recently published full reports of an extensive trial of scopolamine in an aqueous cream base applied locally. With 3% scopolamine he was able to control gustatory sweating, but there was a tendency to dryness of the mouth, presumably due to general absorption, and to dilatation of

the pupils from accidental contamination of the eyes. We have tried in two cases the local application before a meal of 1% atropine in an aqueous cream base, but this had no effect on the sweating.

While, therefore, the problem of gustatory sweating is theoretically capable of solution at a number of different levels, further work is required to determine the best solution in practice. In the meanwhile it should be emphasized that the complication, though an annoying one, is of minor importance compared with most of the conditions for which parotidectomy is performed.

Summary

Gustatory sweating in the auriculotemporal area is a common sequel of parotid operations.

Detailed studies have been carried out on two patients with the syndrome and in less detail on a further 14 post-parotidectomy patients, eight of whom showed gustatory sweating.

Gustatory sweating was abolished by blocking the otic ganglion, but was unaffected by blocking the cervical sympathetic. From this it was concluded that gustatory sweating was not mediated by the sympathetic pathway responsible for normal thermal sweating, but by the parasympathetic pathway responsible for normal gustatory salivation.

The theory which best explains the observed facts is that of aberrant regenerating parasympathetic fibres reaching sympathetic end-organs (that is, sweat glands).

The demonstration, we believe for the first time, of the converse phenomenon, thermal salivation, provides further evidence in favour of the theory of aberrant regeneration.

Hypersensitivity to acetylcholine of the partially denervated sweat glands of the auriculotemporal region is present, and its degree influences the severity of the syndrome.

The condition theoretically lends itself to treatment in a number of ways, but more work is required to find a satisfactory practical solution.

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REFERENCES

- Baillarger, (1853). *Gaz. méd. Paris*, 23, 194.
- Chorobski, J. (1951). *Arch. Neurol. Psychiat. (Chicago)*, 65, 299.
- Doupe, J., Cullen, C. H., and Chance, G. O. (1944). *J. Neurol. Psychiat.*, 7, 33.
- Duphénix (1757). Quoted by Baillarger (1853). *Gaz. méd. Paris*, 23, 194.
- Ford, F. R. (1933). *Arch. Neurol. Psychiat. (Chicago)*, 29, 1279.
- Freedberg, A. S., Shaw, R. S., and McManus, M. J. (1948). *J. clin. Invest.*, 27, 669.
- Frey, L. (1923). *Rev. neurol.*, 30 (2), 97.
- Gardner, W. J., and McCubbin, J. W. (1956). *J. Amer. med. Ass.*, 160, 272.
- Granit, R., Leksell, L., and Skoglund, C. R. (1944). *Brain*, 67, 125.
- Haxton, H. A. (1948). *Ibid.*, 71, 16.
- Herxheimer, A. (1958). *Brit. med. J.*, 1, 688.
- Hogeman, K. E. (1951). *Acta chir. scand., Suppl.*, 159, 94.
- Laage-Hellman, J. E. (1957). *Acta oto-laryng. (Stockh.)*, 48, 234.
- (1958). *Ibid.*, 49, 132.
- List, C. F., and Peet, M. M. (1938). *Arch. Neurol. Psychiat. (Chicago)*, 40, 269, 443.
- Moyse, P. (1955). *Mém. Acad. Chir.*, 81, 999.
- Needles, W. (1936). *Arch. Neurol. Psychiat. (Chicago)*, 35, 357.
- Thomson, M. L., and Sutarman (1953). *Trans. roy. trop. Med. Hyg.*, 47, 412.
- Uprus, V., Gaylor, J. B., and Carmichael, E. A. (1934). *Brain*, 57, 443.
- Weber, F. Parkes (1897-8). *Trans. clin. Soc. Lond.*, 31, 277.
- Young, A. G. (1956). *Brit. med. J.*, 2, 976.