

## An unusual case of gustatory sweating

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### Summary

**A non-insulin-dependent diabetic who developed gustatory sweating is reported. Generalized auto-immune disease and anosmia were also present.**

### Introduction

Facial sweating after food (gustatory sweating) is a recognized complication of long-standing insulin-dependent diabetes mellitus (Watkins, 1973). It is usually provoked by cheese, salty or spicy foods and accompanies other features of autonomic and peripheral neuropathy. The patient herewith reported is unusual in that he developed gustatory sweating as a major, but relatively isolated complication, whilst on treatment with oral hypoglycaemic agents, albeit during a period of deteriorating diabetic control.

### Case report

A 62-year-old male was found to be diabetic in 1971 and was readily stabilized on diet and glibenclamide. On regular follow-up, the diabetes remained stable until late in 1980. Metformin was started in January 1981 and was increased in amount without improvement in the diabetic control. He was therefore admitted to hospital in August 1981 for stabilization with insulin.

In 1952 he had a partial thyroidectomy for hyperthyroidism and in 1970 was found to be hypothyroid when he was hospitalized with a myocardial infarct. In 1978 pernicious anaemia was diagnosed. In 1979 he began to complain of impotence and also noted the sudden development of anosmia. He has one brother with pernicious anaemia and anosmia which also developed suddenly. In 1950 his appendix was removed and he has had five herniorrhaphy operations since 1934.

In May 1981 he began to complain of profuse sweating and slight flushing of the face and sides of the neck, and a buzzing sensation in his head associated with eating, particularly breakfast. Initially, this was thought to be a hypoglycaemic manifestation aborted by eating, but on going into

the sequence of events in detail it was clear that the features developed as a result of eating and never preceded the meal. This response to eating was observed regularly when he was hospitalized and hypoglycaemia was never demonstrated. Sweating was the outstanding feature. In hospital his diet was continued, insulin was given and he was stabilized on 12 units of Actrapid and 20 units of Monotard insulin daily. Physical examination showed a mild peripheral neuropathy manifested by decreased appreciation of light touch and pinprick over the feet and ankles. The left testis was absent and the right atrophic. The anosmia was complete. The retinae were normal. The Valsalva manoeuvre, carried out three times with electrocardiographic control of the heart rate, consistently showed no change during the breath-holding and an increase in rate of a few beats per minute after the breath-holding. The blood pressure was not recorded. Routine haematology, biochemistry and urinalysis were normal. A tetracosactrin (Synacthen) test gave a normal response. Auto-antibodies to thyroglobulin, pancreatic islet cells, parathyroid and testicular tissue were not found, antithyroid cytoplasmic and antiparietal cell antibodies were present. Anti-nuclear factor was not demonstrated. The HLA Pattern was A2, A3, B7, and B17.

Hyoscine taken by mouth 30 to 60 min before meals, in a dose of 20 mg, lessened the sweating, flushing and tinnitus induced by meals, particularly the sweating, but did not completely abolish the response.

### Discussion

Watkins (1973) described six patients with gustatory sweating. All had been diabetic for at least 18 years, were insulin dependent and had severe diabetic complications, including retinopathy. Very few other patients with this problem have been reported and with one exception all were insulin dependent with severe diabetic complications (Freedman, 1973; Stuart, 1978; Janka, Standle and Mehnert, 1979).

Freedman's second patient, a 78-year-old female, had diabetic symptoms for only 2 weeks before developing gustatory sweating, had no other diabetic complications and was subsequently controlled on oral hypoglycaemic therapy. Our patient had been diabetic for only 10 years and although now on insulin, had been well controlled on diet and oral therapy for the first 9 years. Admittedly his symptoms developed during the few months while his diabetic control was deteriorating. His only complications were a mild peripheral neuropathy and an autonomic neuropathy, manifested by the abnormal response to the Valsalva manoeuvre and possibly by the impotence. This latter problem may be endocrine in origin however, as he had testicular atrophy.

Where the anosmia seen in this patient fits in the picture of diabetes and its complications is uncertain, particularly as the patient has a brother with anosmia. Whether or not there is a link between the anosmia and the abnormal gustatory response is equally unclear. The gustatory sweating which sometimes occurs after cervico-thoracic sympathectomy is rarely triggered by smelling food (Bloor, 1969).

The mechanism by which gustatory sweating develops is discussed by Watkins (1973), Bronshvag (1978) and Stuart (1978). It is generally felt to be related to axonal degeneration with abnormal sprouting from contiguous axons. Watkins proposed that interconnections might develop between the parasympathetic fibres of the vagus nerve and sudomotor tracts of the superior cervical sympathetic ganglion during the development of diabetic neuropathy. Stimuli are diverted to sympathetic cholinergic axons destined for the face and possibly the arm (Bronshvag, 1978). Bronshvag also believes that adrenergic vasomotor impulses from the cervical sympathetic chain cause the other less constant and less com-

monly seen parts of the syndrome such as gooseflesh, vasoconstriction, paraesthesia and flushing. He does not explain the opposing responses of vasoconstriction and flushing, but we presume that these do not both occur in the same patient, as our patient showed only flushing. On the other hand, Stuart (1978) considered that the symmetry of the response on the two sides of the face and neck is not explained satisfactorily on the basis of axonal degeneration and abnormal connections between parasympathetic and sympathetic fibres. He believes that diabetic gustatory sweating is merely physiological sweating greatly exaggerated by the diabetic neuropathy.

Whatever the mechanism, most patients are helped by anti-cholinergic drugs taken before meals and such was certainly the case with our patient. Hyoscine (Buscopan 20 mg) taken  $\frac{1}{2}$ -1 hr before meals almost stopped the sweating, and interestingly also lessened the associated flushing and tinnitus, a response we cannot satisfactorily explain. Janka *et al.* (1978) reported an equally effective response to clonidine given for associated hypertension.

## References

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