TYMPANIC NEURECTOMY*

By P. H. GOLDING-WOOD (Maidstone)

LEMPERT (1946) proposed resection of the tympanic plexus for the relief of tinnitus—a procedure he called "tympanosympathectomy". The rationale is dubious for these fibres appear not to reach the cochlea, and certainly his operation has not gained wide acceptance. My own limited experience of it for tinnitus has with one exception been disappointing. This consideration apart, the tympanic plexus has remained an anatomical curiosity devoid of surgical interest.

I hope to show today that it is of real importance in a number of clinical states that can be promptly relieved by suitable surgery.

The nerve-fibres concerned however, are gustatory, secreto-motor, or subserve pain, whereas sympathetic fibres properly so-called are vaso-constrictor or sudo-motor in function. Thus I reject the term "tympano-sympathectomy". Certainly a few purely sympathetic fibres join the plexus, but if it is their resection that is desired then the middle ear is not the site to choose. I propose to use the term tympanic neurectomy to indicate resection of the tympanic branch of IX, with or without resection of the chorda tympani, as an elective procedure.

Anatomical Considerations

The tympanic plexus is formed primarily by the tympanic branch of the glossopharyngeal nerve which enters the temporal bone through a canaliculus on the ridge between jugular vein and internal carotid artery. Thereafter it courses over the promontory—ultimately forming the lesser superficial petrosal nerve which passes to the otic ganglion just below foramen ovale. Here the secreto-motor fibres relay before being distributed to the parotid gland with sensory fibres of the auriculo-temporal nerve.

The main tympanic branch runs more or less vertically across the promontory a little anterior to the round window niche. Generally it lies on the bone, under cover of the mucosa, through which it is readily visible. Alternatively it grooves the bone, and in some 20 per cent. cases it lies invisible in a bony canal. Over the promontory it gives minor branches and receives some communicating fibres from the carotid sympathetic plexus.

Just beyond the tympanum, the lesser superficial petrosal nerve lies, for a short distance, closely adjacent to the greater, to which it gives a communicating twig (Fig. 1). This fine anatomical point may be clinically important.

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Pain

Excluding the common local or reflex causes, aural pain may be entirely neuralgic in character, and several clinical patterns exist.

1. Paroxysms of agonizing and unbearable pain are felt deep in the ear. Whilst merely touching the ear may induce pain, with its onset there is an irresistible urge to dig a finger into the ear as deeply and as forcibly as possible.

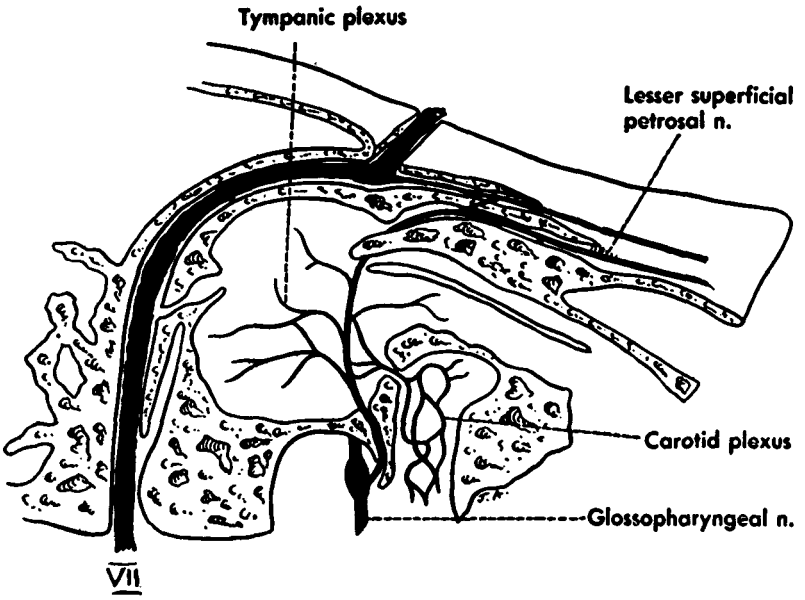


FIG. 1.
The Tympanic Plexus and its Connections.

This type was designated tympanic plexus neuralgia by Reichert (1933) and by Erickson (1936). Fortunately it is rare, for the patient's distress is terrible to witness.

2. Alternatively there is constant dull aching pain in the ear, sometimes with wider radiation over the face, and the patient feels the ear may burst.

This type has often been described as geniculate neuralgia. Whilst the exact site of origin of pain remains unproved, similar cases are recorded with complete relief given by intracranial section of the nervous intermedius alone (Clark and Taylor, 1909; Furlow, 1942).

Between these extremes pain may follow other patterns, intermittent or constant, with or without trigger areas in the external auditory canal.

All those with experience however, know the difficulty of distinguishing between involvement of nervous intermedius, glossopharyngeal and vagus

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as the cause of pain in the ear. The difficulty is not surprising when one considers the complexity and extensive overlap of the innervation.

The role of upper vagal rootlets is clear from cases reported by Dandy (1945) and by Karnosh, Gardner and Stowell (1947) where pain not relieved by glossopharyngeal rhizotomy was abolished by section of upper vagal rootlets.

This problem of severe aural pain has generally been left to neuro-surgeons and posterior fossa craniotomy. Neuro-surgeons believe that unless the pain can be shown by stimulation to be transmitted exclusively by the nervous intermedius, additional section of glossopharyngeal and upper vagal rootlets is imperative.

The possibility of successful otological approach to this problem was indicated by Rosen (1953). He explored the tympanum under local anaesthesia in two cases and stimulated the tympanic nerve and the chorda tympani with a bi-polar electrode. In both his cases only stimulation of the chorda tympani evoked the patient's characteristic pain and its resection alone gave complete relief for at least the 9 months preceding his report.

I have utilized this method in four cases—with the following results:

In two cases stimulation of tympanic nerve alone gave characteristic pain and its resection complete relief.

The third case was unsuccessful—tympanic stimulation gave equivocal results and local resection did not give relief. This was later obtained by vagal root section at posterior fossa craniotomy.

The fourth case was explored under general anaesthetic—both tympanic plexus and chorda tympani nerve being resected—a procedure that abolished aural pain hitherto constant and unremitting for several months.

Whilst the possibility of later recurrence cannot be ruled out its inevitability is quite unproved. In these cases relief of pain has now endured for intervals of 1, 2 and 4 years. Some cases will still demand formal craniotomy, but we may safely conclude that tympanic neurectomy will frequently give relief at much less risk, and will almost certainly yield information of the utmost value before neurosurgical exploration is undertaken.

Collateral Nerve Sprouting

Before turning to other considerations, I would draw your attention to the phenomenon of collateral nerve sprouting that is an established characteristic of all nervous tissue.

The process of nerve degeneration stimulates adjacent healthy fibres to produce small collateral sprouts that grow into the tubes of degenerating fibres. Elsewhere, this process is limited but, as Murray and Thompson (1957) have shown, it is enormously effective in the autonomic system—

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where sprouts excited even from adjacent nerves may make functional connections. In the experimental cat they demonstrated vagal re-innervation of the denervated superior cervical ganglion. Their findings have recently been confirmed.

We can conclude that whenever an autonomic nerve degenerates, any closely adjacent normal autonomic fibre will give off sprouts which can connect up with appropriate cholinergic or adrenergic endings. Either useful function or bizarre sequelae may result.

I have applied these observations to problems set by the Frey syndrome and by crocodile tearing after facial palsy. The mechanism of both has hitherto remained speculative and obscure and treatment has been unsatisfactory.

The Frey Syndrome

This syndrome of gustatory flushing and sweating of the face, generally about the area of the auriculo-temporal nerve is not unusual after parotidectomy. About a year after such operation some 25-30 per cent. of patients have noticed at least minor degrees of this phenomenon. Occasionally it is severe and sweat dripping off the face with every meal occasions great embarrassment (Fig. 2).

Elsewhere I am recording in detail (Golding-Wood, 1962a) an investigation of 14 such patients—derived from 42, on whom I had previously carried out parotidectomy. I shall deal only with my conclusions here (Fig. 3).

(1) Parotidectomy necessarily sections sympathetic fibres supplying local sweat glands.

(2) Gustatory sweating develops gradually in this area after a latent period of several months.

(3) The phenomenon is clearly a reflex akin to the salivation evoked by chewing food.

(4) The effector pathway is primarily the glossopharyngeal nerve and its connections to the otic ganglion.

(5) Only re-innervation of denervated sweat glands by collateral sprouts from parasympathetic fibres normally evoking parotid salivation provides a rational explanation.

(6) If this is correct then section of these parasympathetic pathways should abolish this embarrassing reflex. This can most easily be attained by tympanic neurectomy.

On this basis I have operated on three patients with severe gustatory sweating after parotidectomy with these results:

Case I

Resection of tympanic nerve alone; immediate and complete relief.

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CASE II

Similar resection gave marked but incomplete relief, later section of the chorda tympani abolished symptoms.

Here one may remark that there is evidence that the chorda tympani

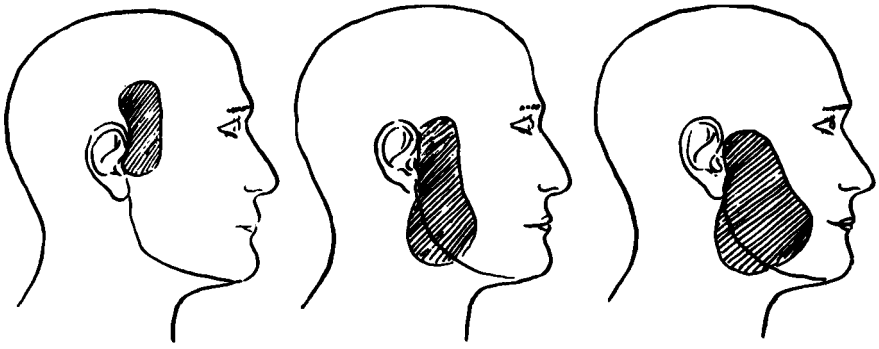


FIG. 2.
Sweating Areas in the Frey Syndrome.

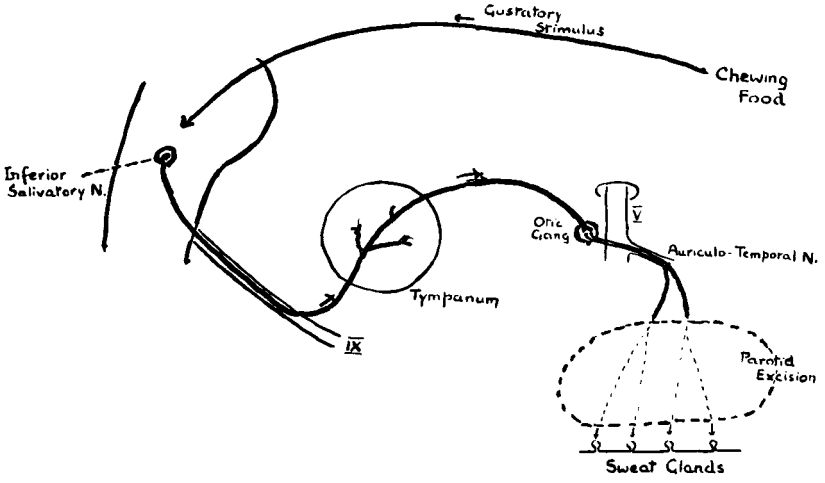


FIG. 3.
The Reflex Arc of the Frey Syndrome.

may play some part in parotid secreto-motor activity and may thus also provide collateral sprouts.

CASE III

Resection of tympanic and chorda tympani nerves; gave complete relief. These results have now endured 18 months—2 years since operation.

Recently Brain has recorded a further success by resection of the tympanic plexus.

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Crocodile Tearing after Facial Palsy

We know that severe lesions of the facial nerve at, or proximal to, the geniculate ganglion may be followed by the later development of crocodile tears.

The analogy between gustatory lacrimation and post-parotidectomy gustatory sweating is as obvious as the patient's embarrassment.

My material shows that collateral sprouts from lesser to greater superficial petrosal nerve complete the reflex arc (Fig. 4).

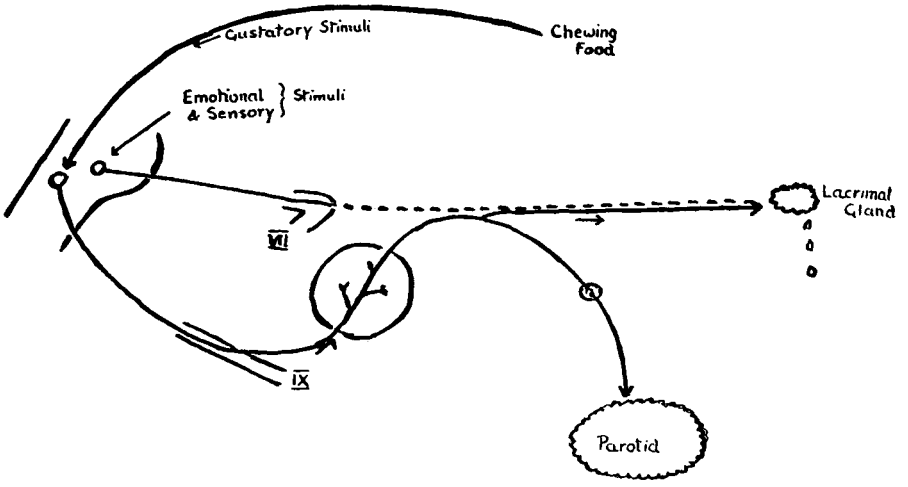


FIG. 4.
The Reflex Arc of Crocodile Tears.

CASE I

This was the girl on whom I carried out my first greater superficial petrosal neurectomy for vasomotor rhinitis 6 years ago—reported as Case I in my paper in *J.L.O.* (Golding-Wood, 1961).

Unilateral section of this nerve relieved the vasomotor rhinitis and gave a dry eye, results that have endured. Unfortunately operation was complicated by facial palsy, which but slowly recovered. Whilst the eye remained dry to emotional or sensory stimuli, after an interval of 4 years tears would stream from it with every meal.

It seemed clear that there had been no regeneration of greater superficial petrosal fibres as such, but collateral sprouts from lesser superficial petrosal had reached the lacrimal gland to establish a gustatory reflex. The communicating twig between the petrosal nerves possibly facilitating this.

Resection of tympanic nerve abolished this gustatory lacrimation forthwith.

CASE II

A woman who had had a severe Bell's palsy 8 years previously, the

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eye being dry whilst the palsy endured. Spontaneous recovery took a year but has been amazingly complete, without trace of synkinesia. Normal lacrimation was restored but with every meal, tears would roll down her right cheek.

The same gustatory reflex had been procured by collateral sprouts from lesser superficial petrosal nerve. Resection of the tympanic plexus immediately abolished the symptom, and the result is explicable only by breaking the reflex arc.

CASE III

A middle-aged man had developed crocodile tears 8 months after a severe facial palsy which showed but partial recovery. The middle ear was explored under local anæsthesia. Stimulation of Jacobson's nerve induced brisk lacrimation and its resection gave immediate relief. Stimulation of the chorda tympani elicited no such response.

These cases are all recent, and post-operative results have been assessed only 3, 8 and 9 months. From analogous cases I expect them to endure.

I have but one other case of crocodile tears. These too were first noted 8 months after onset of Bell's palsy that otherwise made excellent recovery. Operation was not required for, by blinking, she could divert most of her gustatory tears to the natural channels. The gustatory lacrimation was however, temporarily abolished by local infiltration at the jugular foramen. This, of course, blocked the glossopharyngeal nerve immediately proximal to its tympanic branch.

Recurring Non-Suppurative Parotitis

Recurrent painful swelling of the parotid gland often provides a difficult therapeutic problem. Simple dilatation of partial outlet obstruction of Stenson's duct relieves some—but others persist. Evidence of either infection or sialectasis is often lacking. Prolonged antibiotic therapy fails.

Irradiation to abolish parotid secretion is but transiently effective in reasonable dosage and often we have had to resort to formal parotidectomy for relief.

To avoid such operation Diamant (1959) proposed ligating Stenson's duct to enforce atrophy of acinar cells—but obviously the initial phase of glandular distension may be extremely painful and prolonged.

It seemed reasonable to attempt control by resection of the secretomotor supply, and in three cases this seems to have been completely effective (Fig. 5).

We know the main parotid supply is through the tympanic plexus and its connections to the otic ganglion. Reichert and Poth (1933) found the chorda tympani might also contribute to parotid secretion, although

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the pathways are unknown. I have other personal observations to confirm this, but time will not allow their elaboration here.

Accordingly, to abolish parotid parasympathetic supply I now resect both the tympanic fibres and chorda tympani.

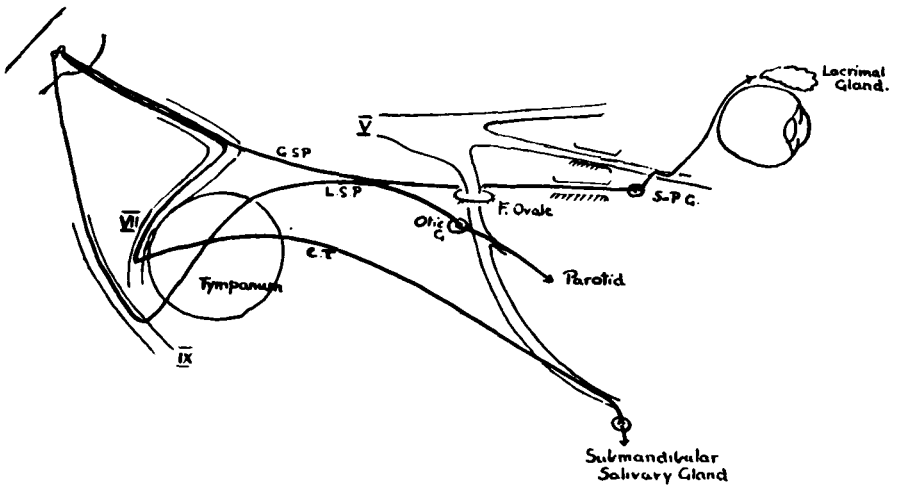


FIG. 5.
Lacrimal and Salivary Secreto-Motor Pathways.

Chronic Secretory Otitis Media

We are all aware of the common secretory otitis media that seems secondary to some eustachian obstruction, and its simple alleviation by tympanic paracentesis and inflation. Equally we are aware of occasional cases unassociated with eustachian obstruction that go on producing fluid despite repeated drainage procedures.

Attempts to establish these cases as examples of allergy have largely failed—at least in the pure terms of antigen-antibody reaction.

We are now aware how closely cholinergic stimulation can duplicate the glandular effects of histamine release, and this is the basis for parasympathetic section in chronic vasomotor rhinitis (Golding-Wood, 1961a, b). Similar conditions might explain this chronic secretory otitis media.

Anatomically, it is established that several small scattered autonomic ganglia exist on the branches of the tympanic plexus. Their function is as yet unknown but may reasonably be deduced from structural analogy. In my opinion, it is probable that these are parasympathetic ganglia giving secreto-motor supply to the mucosa of middle-ear cleft and adjacent eustachian tube.

On these assumptions I have carried out tympanic neurectomy in two cases which had defied previous therapy for 15 and 24 months respectively.

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Tympanotomy disclosed a thickened oedematous mucosa in one but a normal appearance in the other. Suction clearance of the fluid and resection of the tympanic plexus brought rapid relief and restoration of apparent normality to both.

Clearly this evidence is too slender to do more than permit the suggestion that these resistant cases of secretory otitis media may be of cholinergic origin. Further trial of this operation may clarify the position.

Technical Considerations

(Fig. 6)

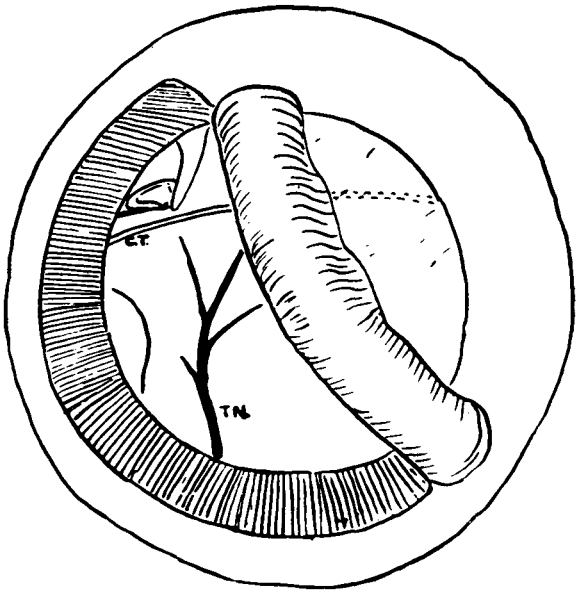


FIG. 6.
Operative Approach in Tympanic Neurectomy.

These need not detain us here. A permeatal incision rather like Rosen's is prolonged around the lower border of the drum which is turned forwards and upwards. The tympanic nerve is sought on the promontory. If hidden in a bony canal its site is frequently disclosed by a small vascular marking. The canal is opened with a fine pick, the nerve lifted out and resected as far as possible. I doubt if restoration of neural continuity would follow this simple procedure and so far have not seen evidence of this. Nevertheless it is generally possible to obliterate the lower end of such canal by careful use of a micro-drill. 16× or 24× magnification on the Zeiss microscope is employed and the blue filter is helpful.

So far I have not had any post-operative difficulty and hearing has not been affected.

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Conclusion

My results can be tabulated thus:

TABLE I.
TYMPANIC NEURECTOMY

Indication	Cases	Relieved
Tinnitus	4	1
Aural Neuralgia	4	3
Frey Syndrome	3	3
Crocodile Tears	3	3
Recurring Non-Suppurative Parotitis	3	3
Chronic Secretory Otitis Media	2	2

Excluding tinnitus, for which I doubt its purpose, these results surely commend the operation for further trial. It seems reasonable to claim that the mechanism of Frey's syndrome and of crocodile tearing has been clearly defined. These results indicate the importance of collateral nerve sprouting in the cranial autonomic system, and provide the first definite evidence of it in man.

In recurring parotitis and in chronic secretory otitis media much remains to be done to evaluate this approach. Clearly, however, otologists have a unique opportunity of contributing to the solution of various problems of secreto-motor function that have baffled physiologists for many years.

Whilst I gratefully acknowledge the lead given by Julius Lempert and Sam Rosen I trust these observations will introduce many otologists to fresh fields of thought and provide a guide to effective surgical relief.

Acknowledgements

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