Syndrome of “Crocodile Tears”

Pharmacological study of a bilateral case

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Pliny the Elder, whose writings are consistently more notable for their dramatic interest than for their scientific accuracy, related the story of the crocodile’s hypocritical tears. After devouring the body of a victim, the reptile supposedly shed many tears over the head, before eating that also. Bogorad (1928) and Kaminsky (1929) applied the name “crocodile tears” to the syndrome of excessive lacrimation while eating. The more prosaic term for the syndrome, “paroxysmal lacrimation”, is deservedly less popular.

Although relatively uncommon, the crocodile tears syndrome is of considerable interest. Most cases are acquired and unilateral and usually follow a Bell’s palsy affecting the same side. Normally, the facial nerve contains fibres which are secretomotor for the sub-mandibular salivary glands, as well as secretory fibres to the lacrimal gland. Anatomical features are discussed in detail by Savin (1939). The generally accepted explanation of the pathogenesis of crocodile tears (Walsh, 1957) is that, during recovery from Bell’s palsy, regenerating salivary fibres in the facial nerve become misdirected and innervate the lacrimal gland. Stimuli such as the taste or smell of food, which normally would excite a flow of saliva, may then produce a simultaneous flow of tears on the same side as the original Bell’s palsy. Chorobski (1951), in his extensive review of the syndrome, suggested that paroxysmal lacrimation was due, not to anomalous re-innervation of the lacrimal gland, but to ephaptic cross-stimulation of demyelinated but otherwise intact lacrimal fibres by efferent impulses in the salivary secretomotor fibres of the facial nerve. However, this hypothesis is not supported by Chorobski’s own observation that, in cases of paroxysmal lacrimation, spontaneous emotional lacrimation is usually lost.

Crocodile tears have also been described as a congenital abnormality. Lutman (1947) discussed three patients with congenital paroxysmal lacrimation and paralysis of the lateral rectus muscle of the eye. In two of these cases both lesions were bilateral, and in the third both anomalies were unilateral, affecting the same eye. It was suggested that a lesion in the pons involved the abducent nucleus together with either the superior salivatory nucleus or the genu internum of the facial nerve as it loops around the abducent nucleus.

The present communication describes an unusual case of bilateral crocodile tears in which the lesion was acquired and followed a unilateral Bell’s palsy. The patient was followed over several months and the effects of various drugs on lacrimation were studied. These studies may be helpful in the management of this sometimes very distressing syndrome.

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Case report

A 26-year-old Nigerian science graduate had sustained a fairly severe right Bell's palsy 10 years previously. This improved considerably, but 2 years later he had a recurrence on the same side, which also improved steadily, and thereafter from time to time he experienced intermittent flickering in the muscles of the right side of the face. One year before presentation he noticed that his right eye became moist when he was chewing. This became increasingly troublesome so that eventually tears overflowed from his right eye during most meals. His left eye then began to water during meals and within a few months the condition became, in his opinion, symmetrical. Lacrimation occurred every day and was more copious with highly spiced foods. Tears ran freely down his cheeks and became a social embarrassment. There was no history of a left facial weakness. Physical examination showed no abnormality other than mild weakness of the lower part of the right side of the face when grimacing. His eyes, eyelids, and optic fundi were normal and the lacrimal ducts were patent.

Observations

Tear production was measured by a modification of the blotting paper test of Schirmer (1903). Strips of Whatman No. 52 filter paper, 0.5 × 3.5 cm., were weighed and inserted into the lower conjunctival fornix of each eye, being folded at right angles over the ciliary border of the lid and left in position for 5 minutes. During this period the patient chewed three Whatman No. 1 filter papers previously dipped in a 2.5 per cent. solution of citric acid and air-dried. In this way the patient received a reproducible stimulus from both taste and mastication. After 5 minutes the conjunctival filter papers were reweighed and the tear output expressed in mg.

Control observations were made in six healthy young men using the same standard taste stimulus.

Serial observations of tear output were made over a period of 8 months and the effects of various local and systemic drug treatments were assessed. These observations are summarized in the Table and in the Figure (overleaf).

Comment

The reproducibility of readings which are closely related in time and where effects of treatment are not in question (e.g. those for the left eye on 6.2.69 and 14.2.69) suggests that this is a reasonably accurate method of measuring lacrimal secretion. When the control observations alone are considered, it is seen that the initial tear output was approximately twice the mean value for normal subjects. There was an initial inequality between the outputs of the two eyes: the patient was unaware of this. Furthermore, the serial observations show that, over several months, the greater tear output was sometimes from one eye and sometimes from the other: this did not correlate with alterations in symptoms.

The total tear output is seen to fall and later to rise, only to decrease once more, during the period of observation. The patient noticed a fluctuation in his symptoms; sometimes several meals would pass with no overflow of tears. He insisted that this fluctuation in symptoms was a new event and that for several months before investigation tears had overflowed from both eyes at every meal. On the last two occasions he was seen, the total tear output approached the mean normal value and the patient volunteered that his symptoms were progressively improving. This variability in lacrimal secretion makes any assessment of the effects of attempted therapy difficult and it is possible that the patient's condition began a spontaneous remission shortly after observations commenced.
Table  Output of tears (mg.) with a standard taste stimulus applied for 5 minutes

<table>
<thead>
<tr>
<th>Subject</th>
<th>Date</th>
<th>Week No.</th>
<th>Eye</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patient</td>
<td>6.</td>
<td>2.69</td>
<td>Right: 17.6</td>
<td>Nil</td>
</tr>
<tr>
<td></td>
<td>14.</td>
<td>2.69</td>
<td>Left: 29.7</td>
<td>Guanethidine right eye (7 days)</td>
</tr>
<tr>
<td></td>
<td>21.</td>
<td>2.69</td>
<td>Total: 47.6</td>
<td>Homatropine left eye (3 days)</td>
</tr>
<tr>
<td></td>
<td>28.</td>
<td>2.69</td>
<td>Right: 21.1</td>
<td>Nil</td>
</tr>
<tr>
<td></td>
<td>14.</td>
<td>3.69</td>
<td>Left: 12.8</td>
<td>Nil</td>
</tr>
<tr>
<td></td>
<td>11.</td>
<td>4.69</td>
<td>Total: 33.9</td>
<td>Nil</td>
</tr>
<tr>
<td></td>
<td>18.</td>
<td>7.69</td>
<td>Right: 18.2</td>
<td>Nil</td>
</tr>
<tr>
<td></td>
<td>25.</td>
<td>7.69</td>
<td>Left: 19.6</td>
<td>Homatropine right eye (2 hrs)</td>
</tr>
<tr>
<td></td>
<td>29.</td>
<td>8.69</td>
<td>Total: 41.2</td>
<td>Nil</td>
</tr>
<tr>
<td></td>
<td>5.</td>
<td>9.69</td>
<td>Right: 15.5</td>
<td>Propantheline orally (6 days)</td>
</tr>
<tr>
<td></td>
<td>19.</td>
<td>9.69</td>
<td>Left: 10.9</td>
<td>Propantheline orally (1 day)</td>
</tr>
<tr>
<td></td>
<td>3.10</td>
<td>6.9</td>
<td>Total: 25.8</td>
<td>Nil</td>
</tr>
<tr>
<td>Six normal young men (mean)</td>
<td>13.7</td>
<td>11.8</td>
<td>25.5</td>
<td>Nil</td>
</tr>
</tbody>
</table>

*For details of treatment, doses, and duration see text.

Effects of adrenergic blockade

To achieve adrenergic blockade, one drop of a 5 per cent. solution of guanethidine (Ismelin eye drops, CIBA) was instilled into the right eye twice daily for a week. This produced a miosis but no conjunctival injection. During treatment, the patient noticed less lacrimation from the treated eye. However, measurements after a week of treatment (14.2.69, see Table) show no effect on lacrimal secretion.
Anticholinergic agents by mouth
Initial treatment was with propantheline bromide 30 mg, four times daily by mouth, for 6 days before the test on 25.7.69. This medication produced a dry mouth but no constipation or visual blurring. The total tear output was unaffected. On a second occasion, 45 mg. propantheline was taken 6-hourly for 24 hours before testing. (Doses of this order are not commonly tolerated on a long-term basis.) No decrease in lacrimal secretion was demonstrable (see Table, 29.8.69).

Local cholinergic blockade
The close proximity of the lacrimal gland to the superior conjunctival fornix leads to a reasonable expectation that drugs instilled into the conjunctival sac will penetrate to the lacrimal gland and its secretomotor nerve supply. Eyedrops of 1 per cent. homatropine hydrobromide were used. On the first occasion, one drop was instilled into one eye twice daily for 3 days, and on two subsequent occasions measurements were made 2 hours after the instillation of a single dose of 5 drops into one eye. On all three occasions mydriasis and visual blurring were produced. From the Table it appears likely that on 21.2.69 and 5.9.69 a reduction in lacrimal secretion was produced, while on 3.10.69 no such effect is apparent. Since dosimetry is bound to be highly inaccurate when eyedrops are instilled and then diluted and drained away at a variable and unpredictable rate, the failure of effect on the last occasion needs no special explanation. Presumed spontaneous variations in lacrimal output, for example decreased secretion from the left eye on 28.2.69 and 5.9.69, make assessment of drug effects particularly difficult. It was concluded that, although cholinergic blockade probably did reduce lacrimal secretion, this held no therapeutic promise, since the degree of visual difficulty produced by this treatment was unacceptable.

Discussion
The clinical features of this patient are very unusual, in that bilateral paroxysmal lacrimation followed unilateral Bell’s palsy. It seems that this phenomenon can be explained only in terms of some central mechanism, as in congenital cases with bilateral symptoms. Possibly a pontine lesion has involved both superior salivatory nuclei together with the nucleus of the right seventh cranial nerve.

Although the syndrome of crocodile tears may cause discomfort and embarrassment, the remedy suggested by Savin (1939), excision of the palpebral portion of the lacrimal gland, is considered too drastic by many surgeons and is reported to have been refused by several patients. Other surgical procedures, such as cutting the chorda tympani, diathermy of the lacrimal ductules, or denervating the lacrimal gland by local dissection, may also be considered unjustified when symptoms are not greatly incapacitating. Blockade of the sphenopalatine ganglion (Savin, 1939; Gottesfeld and Leavitt, 1942) is a lesser procedure and may give substantial relief. Cocaine is applied to the lateral wall of the nose just posterior to the middle concha. If this is followed by several hours' relief of paroxysmal lacrimation, the sphenopalatine ganglion is injected with alcohol. The symptoms may recur after several months, and re-injection may be necessary. On the other hand, Walsh (1957) does not offer active treatment to his patients with paroxysmal lacrimation.

The present observations suggest that attempted pharmacological blockade of lacrimal secretion is not therapeutically useful, since even large doses of anticholinergic agents by mouth produce no measurable effect, while local instillation of homatropine produces
impairment of accommodation which is more troublesome than the excessive lacrimation. The bilateral nature of this patient's symptoms makes surgical treatment even less desirable than usual, while drug treatment producing bilateral impairment of accommodation is of course unacceptable.

**Summary**

A case of bilateral paroxysmal lacrimation following a unilateral Bell's palsy is described. Serial measurements of lacrimal secretion showed spontaneous variations in the severity of the condition. Drug-induced reduction in lacrimal secretion was effective only at the price of unacceptable impairment of accommodation.

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**References**