Ptosis¹

LOUIS R. CAPLAN²

From the Stroke Service of the Massachusetts General Hospital, Boston, Mass., U.S.A.

SUMMARY Twenty-five examples of ptosis occurring with an acute stroke are analysed. Thirteen of these patients had hemispherical infarctions in which ptosis could not be explained by third nerve or sympathetic dysfunction. The ptosis in these 'cerebral' cases was bilateral, with other factors such as pyramidal tract damage determining the asymmetry of the ptosis. In some patients, the eyelid was ptosed on the side of a hemiparesis, narrowing the palpebral fissure. The anatomical basis for this is probably damage to pyramidal neurones or their fibres. The 10 cases of ptosis in relationship to brain-stem infarction included two patients with isolated complete ptosis in one eye in association with a contralateral third nerve palsy.

Eyelid ptosis, a common neurological sign, is classically explained by weakness of the levator palpebrae superioris muscle due to oculomotor nerve disturbance, or by weakness of Müller's muscle secondary to involvement of its sympathetic innervation, or by intrinsic disorders of the lids and their musculature. A less known form is 'cerebral' ptosis which occurs in association with hemispherical lesions (Márquez, 1936; Cogan, 1956; Walsh and Hoyt, 1969). Most of the reported cases of cerebral ptosis have been in patients with brain tumours, with the ptosis having been attributed to pressure on the brain-stem, third nerve, or sympathetic nerves.

The present report had its origin in the observation that in some patients with hemiparesis due to hemispherical infarction, ptosis was noted which was not readily explained by either a third nerve or sympathetic paralysis. Twenty-five consecutive cases of ptosis of recent origin from the Stroke Service of the Massachusetts General Hospital have been studied and analysed. These cases were seen within a nine month period during which 284 stroke patients were examined. Thirteen cases qualify for 'cerebral' ptosis, and 10 had a lesion in the brain-stem. Two patients had ptosis as part of a Horner's syndrome related to local disease of the carotid artery. All patients

were examined three or more times during the acute phase of their strokes. Drowsy patients were excluded. The inclusion only of alert patients with vascular disease minimized the role of pressure phenomena.

The findings proved to be somewhat complex in so far as ptosis was present in some cases on the hemiparetic side, in others on the non-paralysed side, and in still others bilaterally. The need to be precise in the selection of cases required that the term ptosis be defined for the purposes of the study. This was not simple, for no definition that included measurable criteria could be found in the literature. Therefore the lids of 20 control patients were examined in order to determine the range of normal lid measurements.

RESULTS

1. CONTROLS The control group, selected randomly from the medical wards, had an average age of 66 years, identical with that of the stroke group. Figure 1 depicts the measurements utilized with the patient fixing gaze straight ahead. The vertical height of the upper eyelid ranged from 2-12 mm. The palpebral fissure varied from 6-12 mm with no definite inverse relationship to the lid height. The diameter of the cornea was relatively uniform at 12 mm (varying from 11.5 to 12.5 mm). In 19 of the 20 controls, the upper lid covered less than 4 mm (one-third)

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² Address for reprints: Dr. Louis R. Caplan, Neurology Service, Beth Israel Hospital, 330 Brookline Avenue, Boston, Mass. 02215, U.S.A.

FIG. 1. *Measurements of lid and palpebral fissure.*
 1. Vertical height of upper lid. 2. Diameter of cornea.
 3. Palpebral fissure.

of the cornea. In the exception, the patient had a neurological lesion. From these findings, the following criteria were derived to define ptosis acquired during an acute stroke.

1. Drooping of the upper lid to cover one-third (4 mm) or more of the cornea.
2. A vertical measurement of the lid more than 8 mm.
3. A definite increase in eyelid droop compared with the previous state, according to relatives, friends, a physician, or a photograph.

Slight degrees of ptosis have been excluded by these criteria. Ptosis was called 'mild' if it met the above criteria, 'moderate' if the lid covered more than one-half of the cornea, and 'severe' if the palpebral fissure was nearly closed by the drooped lid.

2. CEREBRAL (HEMISPHERAL) a. *Unilateral ptosis on the side of hemiparesis* (six cases) Three of these patients had the sudden onset of contralateral hemiparesis, hemisensory loss, and hemianopsia with aphasia or apractagnosia. Two of these three patients had a recent myocardial infarction; one had mitral stenosis, and all were thought to have had middle cerebral emboli. Two of the other patients fulfilled

TABLE
 CLASSIFICATION OF CASES

1. Cerebral

- a. Hemisphere or pyramidal tract infarction with unilateral ptosis on the hemiparetic side (6)
 - (3) Embolic infarction in middle cerebral artery territory
 - (2) Pure motor hemiparesis, lacunar infarction
 - (1) Internal carotid artery stenosis
- b. Hemisphere lesion with bilateral ptosis, more severe on the hemiparetic side (3)
 - (1) Anterior cerebral artery occlusion
 - (1) ? slit haemorrhage, posterior hemisphere
- c. Hemisphere lesion with bilateral ptosis, less on the hemiparetic side (2)
 - (2) Bilateral cerebral infarction, old and recent
- d. Hemisphere lesion with unilateral ptosis on the non-hemiparetic side (2)
 - (2) Unilateral embolic cerebral infarction
- e. Internal carotid artery occlusion with ptosis ipsilateral to the occlusion as a part of a Horner's syndrome (2)

2. Brain-stem

- a. Part of a Horner's syndrome (6)
 - (2) Lateral medullary infarction
 - (1) ? embolus to the top of the basilar artery
 - (3) Basilar occlusion or branch occlusion with pontine infarction
- b. Unilateral ptosis on the hemiparetic side without pupillary change (1)
 - (1) Medial pontine syndrome
- c. Bilateral complete ptosis (3)
 - (3) Rostral brain-stem infarction

Fisher's criteria for pure motor hemiparesis indicating a lacunar infarction (Fisher and Curry, 1965). One patient had a slight right hemiparesis and aphasia due to severe stenosis of the internal carotid artery at the syphon. Four patients in this group had mild ptosis, and in two others the ptosis was moderate in degree (Fig. 2).

In these cases the palpebral fissure was much narrower on the side of the facial paresis, a finding that runs counter to the usual teaching that it is wider on that side. Since this type of ptosis occurred both in large hemispherical lesions and in lacunar cases in which the infarct lies in the internal capsule, the responsible insult probably involves descending suprasegmental upper motor neurone pathways.

b. *Bilateral ptosis, greater on hemiparetic side* (three cases) One patient had an angiographically demonstrated anterior cerebral artery occlusion with left lower extremity and shoulder paresis but no facial weakness. The second had headache, left hemianopsia, left hemisensory loss, bloody spinal fluid, focal seizures, and a

FIG. 2. A patient with moderate left ptosis and left hemiparesis.

moderately severe left facial paresis. All of the physical signs remitted within a week except for the facial paresis and ptosis. The lesion was thought to be a subcortical haemorrhage in the posterior right hemisphere. The third case had an angiographically demonstrated left internal carotid artery occlusion with severe aphasia and right hemiplegia. These were considered examples of bilateral cerebral ptosis in which the ptosis was accentuated on the side corresponding to the pyramidal tract deficit. The three patients all had ptosis of moderate severity.

c. Bilateral ptosis, less on hemiparetic side (two cases) Both patients had a history of an old hemiparesis that had cleared and suddenly developed a hemispherical lesion in the previously uninvolved hemisphere. Neither had ptosis as a residue of their first stroke. Each patient had a severe facial paresis, and mild ptosis on the hemiparetic side and severe ptosis on the non-hemiparetic side. These were believed to be cases of bilateral cerebral ptosis in which a wider palpebral fissure occurred on the side of the hemiparesis.

d. Unilateral ptosis, on non-hemiparetic side (two cases) One patient with rheumatic heart

disease and documented bacterial endocarditis and aortic insufficiency suddenly developed a left hemiplegia and left hemisensory loss. The second patient had a cardiac pacemaker and suddenly developed a right hemiplegia with aphasia. Both were diagnosed as embolism to a cerebral hemisphere. Each had moderate ptosis on the normal side, and a severe facial paresis contralaterally. These were also believed to be cases of 'bilateral' cerebral ptosis (as described in (c) above) in which the widened palpebral fissure on the hemiparetic side had completely obscured the ptosis on that side.

e. Horner's syndrome on non-hemiparetic side in relationship to ipsilateral internal carotid artery occlusion (two cases) Two cases had mild ptosis and miosis on the side of an occluded carotid artery. One internal carotid artery was occluded at the common carotid artery bifurcation in the neck; the other had an occlusion of the internal carotid artery at the syphon with retrograde extension of the clot into the neck. Each patient had a severe hemispherical deficit.

3. BRAIN-STEM *a. Complete bilateral ptosis (three cases)* Two of these patients had a complete third nerve paralysis on one side, while on

the other side complete ptosis occurred in isolation and was not accompanied by any other signs of third nerve deficit. These three cases probably had midbrain infarction and represent examples of the 'syndrome of the mesencephalic artery' recently reviewed by Segarra (1970).

b. *Ptosis on side of hemiparesis* (as in 2a) (one case) This patient, a diabetic lady, awakened with a right hemiparesis, left intranuclear ophthalmoplegia, mild right ptosis, and slight position sense loss in the right hand. The lesion was clinically localized in the medial pons. The pupils were of normal size and equal. This case was believed to be similar to group 2a in which the responsible insult probably involved the descending pyramidal fibres in the brain-stem.

4. **HORNER'S SYNDROME** (six cases) Six patients with a Horner's syndrome had brain-stem lesions. Two of these patients had typical lateral medullary infarctions and three had pontine infarctions. In one case of pontine infarction with bilateral miotic pupils, there was bilateral ptosis greater on the hemiparetic side (as in 2a and 3b). The remaining patient with a Horner's syndrome had an unusual clinical picture with sudden onset of rostral brain-stem and parieto-occipital infarction. The Horner's syndrome in this case was probably related to diencephalic infarction.

DISCUSSION

In this series, 11 patients had ptosis attributable to either a Horner's syndrome or third nerve palsy. In the 15 remaining cases of hemiparesis, ptosis was unilateral on the hemiparetic side in seven, bilateral but greater on the hemiparetic side in four, and greater on the non-hemiparetic side in four other cases. These findings suggest that some control of elevation of eyelids is exerted cortically, probably bilaterally from one hemisphere, so that a hemisphere lesion causes bilateral ptosis. The symmetry of the ptosis may be modified by widening of the palpebral fissure due to facial weakness; or by an accentuation of ptosis on the hemiparetic side due to a disturbance of pyramidal fibres.

Evidence for cerebral control of the eyelids comes from several neurophysiological studies.

Experiments by Ferrier (1875), Sherrington and Grünbaum (1902), and Leyton and Sherrington (1917) showed that, in the monkey, stimulating an area anterior to the motor strip in the second and third frontal convolutions consistently produced bilateral eye opening, usually with turning of the eyes, head, and neck. Occipital stimulation in the monkey produced eye opening often accompanied by conjugate turning of the eyes (Leyton and Sherrington, 1917; Walker and Weaver, 1940). Penfield and Rasmussen (1968) evoked eye opening by stimulating in awake humans the prefrontal, occipital, and, rarely, the precentral cortex.

Jackson hypothesized that bilateral lid movements, like thoracic and abdominal motions, were probably represented in each hemisphere. Gay *et al.* (1967) and Walsh and Hoyt (1969) cite extensive evidence that the eyelids work synergistically. In the present series, five of the cerebral cases had bilateral ptosis, and two others possibly had the bilaterality of the ptosis obscured by a unilaterally widened palpebral fissure. Bilaterality of the ptosis might well be anticipated according to the experiments cited in which unilateral stimulation produced bilateral eye opening. It is a common experience that one cannot open one eye while keeping the other completely still. Although faulty eye opening is a possible explanation of the bilateral ptosis, in this series of cases asking the patient to open his eyes fully produced nearly complete elevation of the upper lids. Another possible explanation for the ptosis is weakness or a decrease in tone of the frontalis muscle, known to receive bilateral innervation.

Blepharospasm sometimes occurs in patients with hemiparesis (Fisher, 1963) and is a possible mechanism for lid ptosis. In fact, four of the cases in this series with bilateral ptosis exhibited forced lid closure when the lids were touched. Hysterical ptosis, another form of cerebral ptosis, which was described in many 19th and early 20th century textbooks of neurology and ophthalmology is probably related to quasi-volitional lid spasm. Gowers (1893) emphasized that hysterical ptosis is generally accompanied by a spasm in the orbicularis oculi, the latter usually readily proven by asking the patient to look upward making the spasm of the orbicularis greater in order to prevent the lid from moving

upward with the eyeball. The phenomenon of hysterical ptosis would be an example of blepharospasm producing lids ptosed at rest.

The finding of a widened palpebral fissure associated with a central facial palsy is in agreement with the work of Penfield and Rasmussen (1968) who found that lid closure was the commonest eyelid movement evoked by electrical stimulation of precentral cortex in man. Smith (1949) identified an area in the precentral cortex of monkeys just posterior to the arcuate sulcus, as a region from which eye closure could be repeatedly evoked by electrical stimulation. Since electrical stimulation of pyramidal tract regions leads to eye closure, a widened palpebral fissure could be readily interpreted as weakness of eye closure. Though textbooks usually speak of lower facial weakness in upper motor neurone facial paresis, Déjerine (1914) emphasized that the superior part of the face is always affected. In this series, four of the cerebral cases with ptosis had a wide palpebral fissure on the hemiparetic side.

Eleven cases had ptosis solely or accentuated on the hemiparetic side. This observation gains physiological support from the observation that Leyton and Sherrington (1917) were able occasionally to evoke eye opening by stimulating portions of the precentral cortex in monkeys, and Penfield and Rasmussen (1968) were able to evoke eye opening by stimulating human precentral cortex. Ptosis could be explained by faulty unilateral eye opening on the side corresponding to the pyramidal tract deficit. However, in each of these studies eye closure was more commonly evoked from stimulation of precentral cortex than eye opening. Cole (1968) has emphasized decreased lid tone as the sign of upper motor neurone facial paresis in the stuporous patient. A decrease in tone of the orbicularis oculi muscle may play a role in the ptosis associated with pyramidal tract lesions.

As already mentioned, in most cases of cerebral ptosis in the literature, pressure effects on the third nerve or brain-stem were incriminated, although in the present series of vascular cases this mechanism would appear to be excluded. Best (1931) described bilateral symmetrical ptosis from a frontal lesion. Munk (1890) and von Monakow (1914) reported ptosis from lesions in the angular gyrus. Goldflam (1926) in dis-

cussing temporal lobe abscess noted that ptosis was present in some cases. He felt that ptosis, like pain in the first division of the trigeminal nerve, was produced by pressure on the sympathetic or cranial nerves in the middle cranial fossa. Wilbrand and Sängner (1900) in reporting 25 cases of cerebral ptosis pointed out the difficulty in attributing the lid sign to the cerebral lesion, since most patients had large tumours with possible pressure effects. Krishna (1965) in a review of 60 cases of ptosis included 12 'cerebral' cases (seven gliomas, four meningiomas, and two cases of subdural haematoma) ascribing the ptosis in tumour cases to third nerve pressure. Walsh and Hoyt (1969) added a case of cerebral ptosis contralateral to a temporal lobe epileptic focus. Fisher (1967) described a case of subdural haematoma with ipsilateral ptosis without pupillary change. He tentatively attributed the ptosis to mechanical effects on elements of the third nerve. Márquez (1936) in a review of abnormalities of the palpebral fissure declared that ptosis could occur as part of a supranuclear paralysis of third nerve function produced by lesions in the cerebral cortex or internal capsule.

Thus it is likely that there are hemispherical sites both frontally and more posteriorly, destruction of which produces bilateral ptosis. The present cases do not identify the regions responsible, since infarction in the territories of the middle and anterior cerebral arteries as well as temporoparietal haemorrhage all produced bilateral ptosis. Facial paresis was not a necessary concomitant as it was absent in one case. This suggests that precentral and pyramidal tract pathways subserving supranuclear control of the facial muscles need not be damaged in cases of cerebral ptosis. At the same time, however, pyramidal tract damage may modify the symmetry of ptosis produced by lesions elsewhere, accentuating ptosis on the hemiparetic side in some cases, and in others by widening the palpebral fissure on the side contralateral to the lesion giving the patient the appearance of ipsilateral ptosis.

Our observations on the duration of ptosis in cerebral cases are fragmentary. In one case it lasted for two weeks and cleared while the hemiplegia persisted. In four other cases the ptosis cleared as the remainder of the deficit dis-

appeared. In two cases the ptosis still persisted when the patients were last seen a month after the onset of the stroke.

The occurrence of bilateral complete ptosis as the result of a presumably unilateral central third nerve lesion is of great interest. The explanation may be found in the work of Warwick (1953, 1964) who, in an anatomical study of the oculomotor nucleus in the monkey identified a caudal midline nucleus which contained a pool of motor neurones for the levators of the eyelids. The levators were the only oculomotor muscles in which he could identify intimate bilateral central connections. This might explain the synkinesis of levator function already referred to above. Both cases in this series of isolated levator paralysis in one eye had a complete third nerve paralysis of the contralateral eye. The corollary might be proposed that bilateral ptosis distinguishes a third nerve lesion near or at its nucleus from a peripheral third nerve palsy. The complete inability to open a closed eye might indicate the third nerve or its central neurones as the site of the lesion rather than a cerebral or pontine location.

Ptosis associated with a Horner's syndrome is well known. Groch *et al.* (1960) reported five instances of Horner's syndrome in patients with carotid artery occlusion, the sympathetic paralysis being related to involvement of the nerves along the carotid artery. O'Doherty and Green (1958) described a partial Horner's syndrome in 12 of 18 cases of carotid artery occlusion emphasizing that the sign may be mild and transient but offers an important clue as to the location of the thrombosis. In this series there were two cases of Horner's syndrome related to carotid artery occlusion and six cases with the syndrome related to brain-stem interruption of sympathetic pathways.

This preliminary study has shown that ptosis occurs in hemispherical vascular lesions in the absence of third nerve or sympathetic paralysis. Further observations are needed to identify the pathological anatomy and physiology of the cerebral ptosis. Recognition of cerebral ptosis and ptosis associated with pyramidal tract lesions should help to clarify heretofore puzzling cases in both vascular and non-vascular disorders.

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