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Voluntary Lid Closing Inability

Release of a Compulsive Reaction to the Exploration of the Environment

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Summary. Voluntary lid closing inability has been observed in two patients with right-sided frontal ischemic damage. The patients developed a transient inability to close their eyelids voluntarily at the same time as a transient forced grasping in the left hand and left hemiparesis not affecting the face. Automatic and reflex lid closures were retained as well as the ability to keep the eyes closed and to reopen them readily on command. In previous reports, inability to close eyelids voluntarily has been attributed to apraxia, paralysis, or motor impersistence. The localization of the lesion observed in our patients suggests other pathogenetic hypotheses similar to compulsive gaze [19]. It is conceivable that the voluntary lid closing inability produced by frontal lobe lesions is due to the release of a compulsion to maintain the lids elevated in the waking state, because of the overactive effect of the visual stimuli.

Key words: Lid closure inability – Apraxia of lid closure – Anterior cerebral artery – Frontal lobe.

Zusammenfassung. Die willkürliche Unfähigkeit zum Lidschluß wurde bei zwei Patienten mit rechtsseitig-frontalen Ischämieherden im Bereich der A. cerebri anterior beobachtet. Die Lidschlußapraxie war vorübergehend und entwickelte sich zusammen mit linksseitigem Zwangsgreifen und linker Hemiparese ohne Facialisbeteiligung. Der automatische und reflektorische Lidschluß war erhalten, ebenso wie die Fähigkeit, die Augen geschlossen zu halten und sie auf Kommando wieder zu öffnen. Bisher wurde die Fähigkeit zum Lidschluß meist als Apraxielähmung, motorische Impersistenz oder Zwangsblicken erklärt. Die Verbindung mit Zwangsgreifen und die Lokalisation der Läsion bei unseren Patienten spricht für eine pathogenetische Hypogenese mit Zwangscharakter. Es wird angenommen, daß die Unfähigkeit zum willkürlichen Lidschluß bei Frontalläsionen durch eine *Enthemmung der forcierten Lidöffnung im Wachzustand* bedingt ist mit überaktiver Wirkung visueller Reize, ähnlich dem „Zwangsblicken" Zutts [19].

Schlüsselwörter: Unfähigkeit zum Lidschluß – Lidschlußapraxie – A. cerebri anterior – Frontallappen.

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Introduction

In 1907 Lewandowsky [15] described a disorder characterized by the inability to close the eyes and to keep them closed voluntarily in a patient with left hemiplegia. He called this disorder *Apraxie des Lidschlusses* (apraxia of lid closure). Subsequent observations were made of this disorder in various neurological diseases, most of which were characterized by diffuse brain involvement. In different reports, voluntary lid closing inability was considered to be due to apraxia, paralysis, or motor impersistence.

In the present study, we describe a disorder strictly limited to voluntary lid closure in two patients with right frontal lobe lesion caused by obstruction of the anterior cerebral artery. After a review of the literature, the nature of this phenomenon is discussed in relation to the site of the lesion observed in our patients.

Case Reports

Case 1. B. M., a right-handed male, aged 64, was admitted on May 16, 1977, following a sudden loss of consciousness. He had a recent history of short episodes of weakness in the left leg. In 1972, moderate hypertension had been diagnosed. A few days prior to his admission he had complained of weakness in the left leg and arm, lasting for several hours. For the next two days, he complained of severe right frontal headache.

On admission, he was stuporous. General physical examination showed a slight cardiac enlargement and arteriosclerotic changes in the retinal vessels. Blood pressure was 180/100 mm Hg; respiration was eupneic at 20/min; pulse was rhythmic at 80/min. On neurological examination, he was not capable of cooperating sufficiently, and when unstimulated, he appeared to sleep. Aroused, he spoke a few words and when asked he moved his right arm pointing to his forehead as the site of pain. His eyes and head deviated to the right. The pupils were equal and reacted to light and to cervical pinch. The corneal reflexes were normal. The weakness of the left limbs was more marked in the leg, with increased tendon reflexes on this side and evidence of Babinski's plantar extension. Somatic sensory examination showed no abnormality. A forced grasping in the left hand was observed. The EEG showed diffuse, slow abnormalities to be more pronounced in the anterior region of the right hemisphere. General laboratory findings were essentially within normal limits.

Two days after admission, the patient was alert and cooperative. Deviation of the eyes and head towards the right had disappeared. On neurological examination, a specific inability to close the eyes voluntarily on verbal command or in imitation was observed. When asked to close his eyes, he remained motionless and looked at the examiner as if he had not understood. When urged to do so, he said "I understand . . . I must close my eyes . . . well . . . no, they are open . . . that's funny!" Sometimes, he lowered his eyelids manually. The ability to keep his eyelids closed until told to open them, was normal. When requested to reopen them, he did so readily. Both spontaneous and reflex blinking were normal. Lid closure could be elicited by touching the cornea, tapping the brow, or making threatening gestures towards the patient's eyes. Lid movement associated with vertical motion of the eyes was present. Lid movements accompanying emotional reactions, such as crying or laughing, were normal. Apart from this specific inability to close the eyes on command, no other abnormalities were observed on examination of the cranial region. Ocular movements were fully and normally executed on command or on following a moving object. Pupillary responses, visual acuity, and visual field were all normal. No asymmetry of the face at rest, or during voluntary movements was observed. He could react normally, when asked to wrinkle his forehead, retract the buccal angles, blow, or put out his tongue, or imitate facial grimaces, except the lid closure. Impairment of active movement was severe in the lower left limb and slight in the upper.

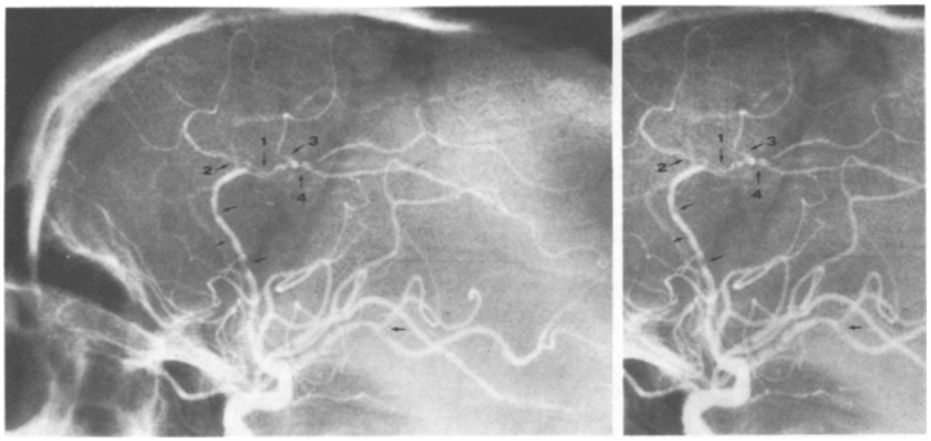

Fig. 1. Case 1: right internal carotid angiography. The lateral view demonstrates segmental narrowing of the anterior cerebral artery (*1*) involving the origin of the anterior (*2*) and middle (*3*) internal frontal branches and the pericallosal artery (*4*). Small atheromatous plaques are present in the wall of cerebral arteries (*arrows*)

Contact of any object with the palmar surface of the left hand caused a forced grasping. When asked to relax his grasp, he noticed that he was unable to do so. "The hand closes by itself... it's not me," he would say. No significant neuropsychological disorder was detected. Language, praxis, gnosis, and body perception were normal. On the Wechsler-Bellevue Intelligence Scale, his verbal score was 116, his performance score 108.

All these manifestations persisted for two weeks. Toward the end of this period, both the inability to close the eyelids voluntarily and the forced grasping disappeared. He regained normal use of the left arm, while a severe weakness in the left leg persisted. Repeated EEG records showed persisting signs of localized damage to the right frontal region.

Right carotid arteriograms demonstrated numerous small atheromatous plaques in the major branches of the cerebral arteries. There was a segmental stenosis of the pericallosal artery also involving the anterior and middle frontal branches at their origin (Fig. 1).

Case 2. C. L., a right-handed woman, aged 36, was admitted on September 3, 1977, 2 h after she had collapsed unconscious to the floor. Her past history included recurrent episodes of rheumatic fever during adolescence. At the age of 22, she began to complain of recurrent attacks of paroxysmal nocturnal dyspnea and palpitation. The clinical diagnosis was mitral stenosis.

On admission, she appeared stuporous and general physical examination revealed normal blood pressure, a pulse rate of 96, and eupneic respiration. The heart was slightly enlarged. On neurological examination, she reacted to pain but not to verbal stimuli. Deviation of the head and eyes towards the right side was noted. The pupils were equal and reacted to cervical pinch. The corneal reflexes were present. Left hemiparesis without affecting the face was found. While a response to a pinprick was elicited from the arm, no response was noted in the leg. EEG showed diffuse abnormalities, predominantly in the right hemisphere, with continual slow focal activity in the frontal region. Angiography of both the right and left internal carotid arteries was carried out. The angiography of the right internal carotid showed, in early phases, a complete occlusion of the proximal (*A1*) portion of the right anterior cerebral artery (Fig. 2). Angiography of the left internal carotid, performed during compression of the right carotid, showed a well-filled left anterior cerebral artery. There was no filling of either the anterior communicating nor the right anterior cerebral arteries (Fig. 3). Cerebral embolism was diagnosed and a treatment with anticoagulants and steroid was begun.

Three days after admission, she was alert and well-oriented. She was aware of her disability. Memory of past and recent events was intact, as well as awareness of contemporary events.

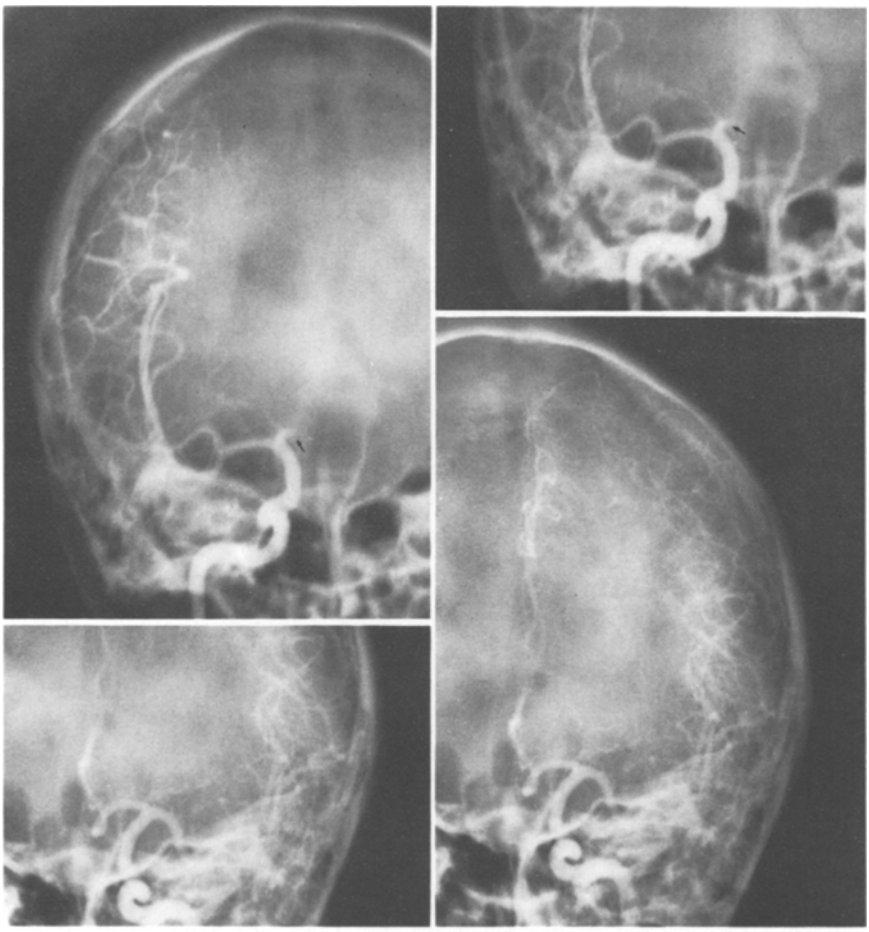

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Fig. 2. Case 2: right internal carotid angiography. The frontal view demonstrates filling of the anterior cerebral artery limited to the first centimeter of its course (*arrow*)

Fig. 3. Case 2: left internal carotid angiography during compression of the opposite common carotid artery. The frontal view demonstrates normal filling of the left anterior cerebral artery. There is no filling of the right anterior cerebral and anterior communicating arteries

Deviation of the head and eyes towards the right side had disappeared. The optic fundi were normal. Pupils, visual acuity, and visual field were all normal. Bilateral confrontation excluded hemianopic inattention. Ocular movements were fully and normally executed on command or on following a moving object. On examination of the facial movements, an inability to close the eyelids on verbal command or imitation was detected. When urged to do so, she said 'I understand . . . that's that,' and lowered her lids manually. Requested to maintain the eyes closed until told to reopen them, she did so normally. On command, she reopened her eyelids readily. Spontaneous blinking as well as blinking in response to threat were normal. Corneal reflexes were present. Lid closure could be elicited by tapping the patient's brow. Lid movement connected with vertical motion of the eyes, was present. No facial weakness was noted. She showed no abnormality when she wrinkled her forehead, retracted the buccal angles, put out her

tongue on verbal command, or imitated any facial grimaces performed in front of her, except the lid closure. Left hemiparesis, more marked on the lower limb, was noted. When the palmar surface of the affected hand came into contact with any object, a forced grasping reaction was caused. Initial signs of spasticity could be recognized in the left leg, and the plantar response was extensor. Sensory examination showed no abnormalities. On neuropsychological examination, no anomalous finding was shown by testing language, praxis, gnosis, and body perception. On the Wechsler-Bellevue Intelligence Scale, her verbal score was 126, her performance score 104.

During the first week after admission, she continued to improve. On the morning of the ninth day, as soon as she woke up, both the inability to close the eyelids and the forced grasping had disappeared. Over a period of 15 days, she gradually regained the complete use of her upper left limb. She was dismissed from hospital after three weeks with only a residual weakness of the lower left limb.

Comment

In the cases just described, an ischemic lesion of the right frontal lobe was the likely cause of the reversible inability to close the eyelids voluntarily, as well as causing the left hemiparesis without affecting the face, and the transient forced grasping of the left hand. In both patients the automatic and reflex lid closing, and the ability to keep the eyes closed and open them readily on command were retained. The patients were alert and cooperative and did not show any sign of mental deterioration.

A motor disorder limited to voluntary lid closure, without affecting any other facial movement, has never previously been described in subjects where the damage is limited to the territory of the right anterior cerebral artery. The available literature indicates that defects of voluntary lid closure have been observed in cases of encephalitis periaxialis diffusa [18], chorea minor [18], diffuse cortical atrophy due to arteriosclerosis [3, 19], subarachnoid hemorrhage due to rupture of an aneurysm of the right internal carotid artery [19], left- and right-sided damage in the territory of the middle cerebral artery [4, 11], Balò's concentric sclerosis [1], mental retardation [17], and multiple sclerosis [6]. In the majority of these cases, the significance of the phenomenon is difficult to evaluate, because of the existence of diffuse brain damage. The few cases with focal brain damage showed an inability to keep their eyelids closed until told to open them, in addition to the lack of voluntary lid closure. This inability to keep the eyes closed, was also present in the first patient described in 1907 by Lewandowsky [15], who called the inability to close the eyelids readily on command 'apraxia of lid closure.' The coexistence of a lack of voluntary lid closure and inability to keep the eyelids lowered, suggests a 'motor impersistence' as the causal mechanism [12].

In 1933, Alajouanine and Thurel [2] described a series of cases with the inability to close the eyes voluntarily. This series included personal observations and the cases previously described by Magnus, Tiling, Roth, and Foix. Nevertheless, in these cases the disorder was paralytic. The patients were affected by a bilateral paralysis of the face, tongue, pharynx, and larynx due to bilateral vascular damage of the rolandic opercula. Alajouanine and Thurel called this syndrome 'cortical form of pseudobulbar paralysis.'

In our opinion, a disorder of the voluntary movement strictly limited to the lid-closing mechanism, as observed in our patients, could hardly be attributed to apraxia. The areas of each hemisphere that control the movements of the trunk, eyes, and upper part of the face, govern both ipsi- and contralateral muscular functions [8]. Thus, apraxic disorders of these somatic areas are rarely observed [10]. Accordingly, a unilateral lesion, as in the cases reported here, should not be able to produce an apraxic disorder of lid closure. As a matter of fact, no apraxic disorders were discovered in our patients when the movements of the face, mouth, tongue, eyes, trunk, and limbs were tested.

The localization of the lesion in the right frontal lobe, suggests other pathogenetic hypotheses apart from apraxia. The lid-closing and lid-opening mechanisms are served by various interrelated cortical and subcortical neuronal circuits. Subcortical centers for opening and closing of the lids exist in animals, and possibly in man. In man, preservation of the blink reflex has been observed on occasion with complete cortical blindness [14] and in anencephaly [16]. Closing of the eyelids has been reported following the stimulation of the frontal and occipital lobes, in proximity to the centers for conjugate lateral gaze [7]. By analogy with ocular motor representation in the cerebrum, it may be assumed that the cortical centers for lid movements respond to voluntary impulses.

When considering the frontal areas in monkeys, it has been reported that lesions, specifically those in Area 8, produce a deficiency in the lid-closing mechanism [13]. In our patients, the instinctive grasping reaction was associated with the inability to close the eyelids voluntarily. Forced grasping in the presence of frontal lobe lesion is considered to be caused by a compulsive reaction to the exploration of the environment directed by the cerebral cortex [9]. A patient with a frontal lobe lesion may not be able to relax his grasp on bedclothes or other objects because of the persisting overreaction to contact stimuli. Similarly, the inability to close the eyelids voluntarily as a result of a frontal lobe lesion may be considered to be due to a release of a compulsion to maintain the lids elevated in the waking state, because of the overactive effect of the visual stimuli similar to the „Zwangsblicken" of Zutt [19]. When the eyes were closed manually, thus removing the visual stimulus, our patients were able to keep their eyes closed without difficulty. This association of lid closure inability with forced grasping was also present in the cases described by Zutt [19]. Nevertheless, in these cases the inability to keep the eyes closed was also present. In other words, it is conceivable that voluntary lid closing inability produced by a frontal lobe lesion can be interpreted as the release of elemental drives of the exploratory behavior in the environment in the waking state. In 1972, Cambier and Dehen [5] observed a disorder strictly limited to voluntary lid closing in two patients with a frontal lobe lesion due to obstruction of the left anterior cerebral artery.

In our opinion, this instinctive reaction to keep the eyelids elevated during the waking state cannot be defined as apraxia, unless we accept the approach of Denny-Brown [9] to kinetic apraxia. This author, in fact, considers kinetic apraxia to be the result of a loss of the normal equilibrium between 'instinctive grasping', released by frontal lobe lesions, and 'instinctive avoiding', released by parietal lesions.

References

1. Ajuriaguerra, J. De, Hécaen, H., Angelergues, R.: Les apraxies. Variétés cliniques et latéralisation lésionelle. Rev. Neurol. (Paris) **102**, 566—594 (1960)
2. Alajouanine, Th., Thurel, R.: La diplégie faciale cérébrale, forme corticale de la paralysie pseudobulbaire. Rev. Neurol. (Paris) T. **II**, 441—458 (1933)
3. Arrigo, A.: Un caso di incapacità di chiusura delle palpebre. Studio clinico ed elettromiografico. Riv. Patol. Nerv. Ment. **83**, 40—58 (1962)
4. Berlin, L.: Compulsive eye opening and associated phenomena. Arch. Neurol. Psychiatry **73**, 597—601 (1955)
5. Cambier, J., Dehen, H.: Le syndrome de l'artère cérébrale antérieure. Sémeiologie analytique. Rapport aux journées internationales de circulation cérébrale, Toulouse, 22 avril 1972
6. Cartei, M. A.: Un caso di aprassia delle palpebre in soggetto affetto da sclerosi a placche: considerazioni clinico-patogenetiche. Riv. Neurol. **46**, 57—72 (1976)
7. Cogan, D. G.: Sopranuclear connections of the ocular motor system—Lid movements. In: Neurology of the Ocular Muscles, Second Edition, pp. 139—148. Springfield (Ill.): Charles C. Thomas (1972)
8. Crosby, E. C., Humphrey, T., Lauer, E. W.: Correlative anatomy of the nervous system. New York: Macmillan 1962
9. Denny-Brown, D.: The nature of apraxia. J. Nerv. Ment. Dis. **126**, 9—32 (1958)
10. Faglioni, P.: Aprassia. In: Neuropsicologia Clinica (Bisiach, E., Denes, F., De Renzi, E., Faglioni, P., Gainotti, G., Pizzamiglio, L., Spinnler, H. R., Vignolo, L. A., eds., pp. 67—135. Milano: Franco Angeli 1977
11. Ferro, F. M., Arnepeta, L., Maurillo, M.: Considerazioni su due casi di aprassia della chiusura e della apertura degli occhi. Riv. Patol. Nerv. Ment. **90**, 300—308 (1969)
12. Fisher, M.: Left hemiplegia and motor impersistence. J. Nerv. Ment. Dis. **123**, 201—218 (1956)
13. Kennard, M. A.: Alterations in response to visual stimuli following lesions of the frontal lobe in monkeys. Arch. Neurol. Psychiatry **41**, 1153—1165 (1939)
14. Levinsohn, G.: Der optische Blinzreflex. Z. ges. Neurol. Psychiat. **20**, 377—381 (1913)
15. Lewandowsky, M.: ÜberApraxie des Lidschlusses. Klin. Wochenschr. (Berl.) **44**, 921—923 (1907)
16. Rothmann, M., cited by Best, F.: Die Augenveränderungen bei den organischen nichtentzündlichen Erkrankungen des Zentralnervensystems. In: Kurzes Handbuch der Ophthalmologie (Schieck, F., Brückner, A., eds.), Vol. 6, p. 531. Berlin: Springer 1931
17. Schachter, M.: A proposito de una dispraxia palpebral rara: la incapacidad de mantener cerrados los ojos al ordenarlo. Rev. Esp. Otoneurooftalmol. Neurocir. **97**, 165—168 (1958)
18. Schilder, P.: Zur Pathologie des Lidschlusses. Apraxie des Lidschlusses by Chorea minor, Unfähigkeit zu dauerndem Lidschluß bei Chorea und Encephalitis. Z. Nervenheilk. **90**, 172—176 (1926)
19. Zutt, J.: Über die Unfähigkeit, die Augen geschlossen zu halten. Apraxie des Lidschlusses oder Zwangsblicken? Nervenarzt **21**, 339—345 (1950)

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