

CASE REPORT

Facial Nerve Palsy In Aviation Facial Baroparesis

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We report a case of a patient who experienced recurrent facial nerve palsies associated with flying. Although this condition is well recognised in divers, only 7 cases have been reported related with aviation. Non of these describe a long-lasting facial nerve palsy, as once experienced by our patient. The presentation, management and the possible pathophysiology of this condition is discussed.

Submitted : 17 November 2009

Revised : 07 April 2010

Accepted : 08 April 2010

Introduction

The rare condition of facial nerve palsy during ascent has been reported both in divers and aviators and in hypothesis associated with ipsilateral middle ear overpressure^[1,2]. This overpressure is caused by the inability to equalize middle ear and ambient pressures due to impaired Eustachian tube functioning. The excessive pressure may be exerted on the facial nerve through a dehiscence of the horizontal part of the fallopian canal, thereby compressing the vasa nervorum. This may cause hypoxia and consequently neuropraxia^[1]. Predominantly, this neuropraxia is transitory resolving between 3 minutes and 3 hours^[2], however long-lasting and even permanent facial nerve palsies have been reported in literature^[1, 3, 8]. This case report presents an aviation associated long-lasting increase in facial nerve palsy in a patient with pre-existing iatrogenic facial nerve damage. The presentation, management and the probable pathophysiological mechanism of this unique case and of facial baroparesis in general is discussed, and a review of the currently available literature is presented.

Case report

A 56-year old man known with pre-existing iatrogenic right facial nerve paralysis, reported a recurrent increase of his facial nerve palsy associated with flying. On two subsequent occasions an increase of the facial paralysis was noticed during ascent, swiftly disappearing on descent. At that time, no medical advice was sought because of the transient character.

Following a more recent flight, a long-lasting facial nerve palsy was developed. During 4 ½ hours of air travel, the patient experienced otalgia and had the sensation of “blocked ears”. The day after the flight, increased drooping of the right side of the mouth was noticed. No other symptoms were reported.

This patient is known with pollinosis and the Treacher-Collins Syndrome, in his case presenting with mandibular hypoplasia, absence of tear ducts, malformation of auricles, external ear canal defects and conductive deafness. During his childhood a canalplasty was performed and parts of the facial nerve were exposed. This procedure was complicated by postoperative facial paralysis. Unfortunately there was no spontaneous recovery and an attempt to reconstruct failed. An incomplete right facial nerve paralysis persisted (House-Brackmann classification V).

The patient consulted the neurologist 5 days after the flight, presenting with persistent increase of the pre-existing right facial palsy. Clinical examination indeed showed total facial paresis, with a maximum House-Brackmann classification. Serology for *Borrelia burgdorferi* and Herpes Simplex Virus (HSV) antigens were tested negative. A performed MRI-scan showed known characteristics of Treacher-Collins Syndrome, plus signs of a right maxillary sinusitis. The patient was referred to a Ear- Nose- Throat specialist for further examination, who subsequently referred the patient to an university medical centre. The maxillary sinusitis was treated with antibiotics in the intervening time.

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Four months after the event, the patient attended the department of otorhinolaryngology at our university medical centre. Gradually, the function of the facial nerve had recovered back to the previous level. Examination revealed characteristics of the Treacher-Collins Syndrome, with a pre-existing incomplete paralysis of the right seventh cranial nerve (House-Brackmann classification grade V). On otoscopy, atresia of both ear canals was detected.

The patient was diagnosed with recurrent facial baroparesis, once complicated by prolonged recovery. For prevention of recurrence, the patient was instructed to use nasal xylomethazoline spray in both nostrils prior to ascent on subsequent flights. In case of reappearance of complete paresis, the patient was instructed how to use the Toynbee manoeuvre. If persisting after the flight, the patient was urged to seek direct medical attention.

On two subsequent inter-European flights this advice was taken in account. Fifteen minutes prior to departure of a 2 ¼ hours during flight, the patient used xylomethazoline nasal spray in both nostrils. Special attention was paid to occurring symptoms. Half an hour after ascent the patient suffered from otalgia and a total loss of motor function on the right side of his face. These symptoms disappeared swiftly on descent. Previous to the returning flight, the patient used the same nasal spray one hour and also 15 minutes prior ascent. No symptoms were reported during or after the flight.

Discussion

As an aircraft ascends, the atmospheric pressure decreases and gas in the middle ear expands according to Boyle's law. In a modern aircraft, the cabin is pressurized creating an air pressure of approximately three-quarters that of the ground atmospheric level, corresponding with an altitude of 8000 feet ^[4]. If a flight starts at sea level, the theoretical maximum pressure difference developing between the middle ear and the cabin atmosphere is calculated to be 261 mBars. This pressure exceeds the capillary bloodpressure^[1] (Figure 1).

Under normal circumstances these high pressure differences are equalized by the function of the Eustachian tube, which passively opens and vents of the positive pressure through the nasopharynx. This process of passive venting is seldom a problem on ascent and normally occurs every 122 meters (400ft) of increasing altitude^[5]. Failures of pressure equilibration may be the result of impaired function of the Eustachian. This may be caused by any condition that narrows the lumen by oedema, increases the amount of viscosity of the mucus coating of the tubal membrane or impairs the ability of the tube to open^[4]. Congenital and traumatic malformations of the nasal skeleton and gross malocclusion of the teeth and jaw are also recognized to impair Eustachian tube functioning^[6]. This failure to vent of air pressure in order to achieve an equilibrium results in an elevated middle ear pressure. This excessive pressure could be exerted on the facial nerve.

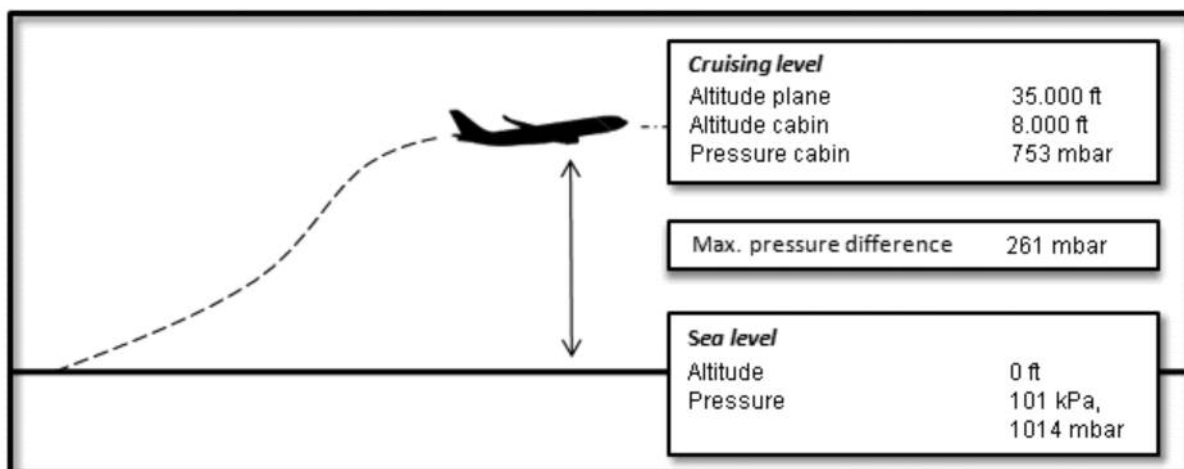


Figure 1. Pressure differences on ascent of a commercial aircraft.

Although reduced conductivity and neuropraxia as a result of extremely elevated hydrostatic pressure in the middle ear have been demonstrated in isolated nerve preparations, the most widely accepted hypothesis for facial baroparesis is ischemic neuropraxia^[1]. This theory reasons that excessive middle ear pressure transmitted through a dehiscence of the facial canal, decreases the blood flow in the vasa nervorum of the facial nerve. This causes ischemia and consequently neuropraxia^[1]. In cats, such neuropraxia has been induced by applying pressures to an unexposed part of the facial nerve^[7]. Experiments in a guinea pig animal model indeed showed that increased middle ear pressure transmitted through a dehiscence of the facial canal decreases the blood flow in the vasa nervorum of the facial nerve^[8]. In anatomical studies of autopsy cases, a dehiscence of the facial nerve canal is shown in 19.7% up to 57% of all human temporal bones^[9-11]. All these studies support the ischemic neuropraxia theory. An alternative, less acknowledged hypotheses, is that in a non-dehiscent horizontal canal elevated middle ear pressure may be transmitted through the fenestra of the chorda tympani.

If middle ear pressure is relieved by descent of the airplane, opening of the Eustachian tube, myringotomy, or if perfusion pressure is increased sufficiently, the nerve's circulation will swiftly be re-

established and normal nerve function will rapidly return. However if middle ear pressure is not reduced or perfusion pressure is not increased adequately, a chain of events leads to the demyelination and interruption of axons, causing the nerve fibres to degenerate. Still, even at this delayed stage, partial motor recovery may occur if adequate perfusion is re-established^[1]. However, if the pressure interferes with the nerve supply for a sufficient long time, the internal structure of the bundle will be destroyed and the nerve converted to a fibrous cord beyond functional recovery^[1]. In cats, irreversible nerve damage has been demonstrated after 3.5 hours of pressure induced ischemia^[7].

Fortunately, most cases of facial paralysis associated with aviation resolve spontaneously during descent, as shown in Table 1. Predominantly, these cases don't need immediate medical attention as they are transient of character^[2, 12-16].

If recurrent, these events could be prevented by the use of adequate self equalization techniques during flight. Prophylactic and therapeutic use of decongestants to prevent or treat middle ear barotrauma are recommended by numerous sources^[17]. Passengers with allergies may benefit from the use of antihistamines for the prevention of aural barotraumas^[4]. Hence facial baroparesis is secondary to aural

Table 1. Documented cases of barotraumatic facial nerve palsy in aviation or in ascent to high altitudes (4, 18).

Reference	Year of Report	Age	Sex	Side	No. of Incidences	Treatment	Outcomes
Bennett and Liske 1	1967	30	M	R	3	-	Normalization
Anonymous report 2	1978	40	M	L/R	2	-	Normalization
Silverstein 3	1978	29	M	L	3	-	Normalization
Woodhead 4 *	1986	?	F	L	5	Equalisation tube insertion	Normalisation
	1988	50	M	L	multiple	Equalisation tube insertion, submucous esection nasal septum and trimming of inferior turbinated	Normalization
Motamed 5	2000	38	M	?	6	-	Normalization
Berghaus 6	2001	20	F	L	1	-	Normalization
Grossman 7	2004	24	M	R	1	-	Normalization
Ardehali 18 *	2009	34	F	R	1	Oral prednisolon treatment	Minimal sensory hearing loss
This case	2010	56	M	R	4	Xylomethazoline nasal spray	Normalisation

* Case of facial barogaresis during ascent to high altitudes, using a road motor vehicle

barotrauma, these recommendations may also be applicable in for the prevention of this condition. In selected cases, ventilation tube insertion is also considered helpful [13, 14]. In case of persistent paralysis after descent, urgent myringotomy should be performed, with the insertion of a ventilation tube for equalization to prevent irreversible damage of the facial nerve. Oral steroid treatment should be considered in persistent facial paralysis if combined with other major complications [18].

Discussion of case

The event as described above represents an unique case of facial baroparesis. Due to pre-existing defects, this report could not be considered a text book example of condition, nevertheless this case demonstrates several important aspects illustrative for the ischemic neuropraxia.

In our patient a reconstruction of the right ear canal was performed for correction of defects caused by the Treacher-Collins syndrome. During this canalplasty, parts of the facial nerve were exposed, creating an iatrogenic dehiscence in the facial canal, which made the seventh cranial nerve more susceptible for pressure changes in the middle ear. Given the fact the patient is known with pollinosis and the Treacher-Collins syndrome, Eustachian tube malfunctioning is very assumable. This could have caused difficulties in middle ear and ambient pressure equalization. The tympanic membrane of our patient is more fibrotic and less elastic compared with normal a tympanic membrane, herewith deteriorating the compensation ratio. During ascent of the airplane, those malfunctions have led to the inability to equalize and compensate pressure differences, leading to an elevated middle ear pressure, which was exerted on the vasa nervorum of the facial nerve through the iatrogenic dehiscence. This resulted in an ischemic neuropraxia of the right facial nerve on 4 subsequent flights.

On one occasion the facial nerve paralysis was long-lasting, and gradually recovered over a period of 2 ½ months. The continuing paralysis is probably caused by long standing elevated pressures in the middle ear. Recovery was eventually achieved, suggesting damaging of the nerve, but not full destruction. More rapid recovery may have been established if direct myringotomy could have been performed.

In our patient, the facial baroparesis was recurrent, occurring in 4 successive flights. This is remarkable,

hence on numerous previous flights no symptoms of such were mentioned. Alterations in tube functioning, such as an upper respiratory tract infection, may have caused the sudden appearance of these events. Although signs of a maxillary sinusitis were detected on MRI-scanning on one occasion, our patient reported no related complaints. No satisfactory explanation for this sudden appearance of events could be given.

A suggested treatment modality for recurrent facial baroparesis is ventilation tube insertion [13, 17]. Unfortunately, due to atresia of the ear canals this procedure could not be performed in our patient. A conservative approach using xylomethazonline prior to flights was used. Two separate doses of nasal decongestants prior to ascent seemed to be effective. Furthermore, the use of an antihistamine could have been beneficial in prevention of facial baroparesis in this case. Direct medical attention should always be sought in case of prolonged recovery.

Conclusions

Facial baroparesis in a rare, mostly temporary paresis of the seventh cranial nerve described in both aviation and diving. The most convincing pathogenetic mechanism is that of ischemic neuropraxia. With this report, an unique case of a long-lasting facial nerve palsy in aviation is presented.

If recurrent, facial baroparesis could be prevented by adequate self equalization techniques during the flight. Prophylactic use of topical nasal decongestants is also considered helpful. If these conservative approaches fail, ventilation tube insertion may be considered.

If long standing facial baroparesis develops in aviation, immediate myringotomy with ventilation tube insertion should be performed to prevent permanent facial nerve damage.

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