Sir: We wish to report an unusual and unexpected complication following the removal of an acoustic neuroma.

A 37-year-old woman was admitted for excision of a large right sided acoustic neuroma. Computed tomographic (CT) scanning of the brain showed in addition to the cerebellopontine mass, three small right sided meninogia, in the frontoplolar, focial and supratentorial regions, without any mass effect. The acoustic neuroma was completely removed through a suboccipital approach while the patient was in a left lateral recumbent position. After the operation, there was complete hearing loss on the right but preserved function of the facial nerve. The postoperative recovery was excellent. The patient received chloramphenicol and high doses of prednisolone. The second day after operation she was allowed to sit erect and suddenly developed complete deafness accompanied by vertigo and left sided peripheral facial palsy. Beside this, neurological examination disclosed horizontal nystagmus to the right and deviation to the left with the Romberg test. Audiometric testing showed a complete hearing loss on the right and an almost complete sensorineural hearing loss on the left. Brain stem auditory evoked responses were absent on both sides. The stapedius reflex was absent on the left. On electronystagmography a spontaneous nystagmus to the right was recorded and caloric testing revealed a marked diminished excitability of the left labyrinth. CT scanning of the brain, nine days after the operation, showed the operative changes and the three meningiomas, but no other abnormalities. Corticoid therapy was resumed for some days without any improvement.

During the following months there was no recovery of this left sided deficit. The patient described here developed a sudden sensorineural hearing loss, loss of vestibular function and a peripheral facial paralysis on the non operated side, the second day after uncomplicated removal of an acoustic neuroma. This complication has to our knowledge not been reported previously. However, recently three cases of sudden contralateral hearing loss after operation in acoustic neuroma patients have been described. In two of them, the hearing loss was of the perceptive type with the lesion situated respectively retrogolcoal and cochlear, appearing the seventh and fourth day after the intervention. There were no other neurological signs. The deficit slowly improved, but the aetiology remained obscure. In the third case, the hearing loss resulted from a serious middle ear effusion. The first two cases show some similarity with our patient, except that they had no facial nerve involvement. The exact mechanism responsible for the event in our case could not be established. However, the lesion must be localised on both the left cochoevestibular nerves and/or inner ear and the left facial nerve. A possible explanation, in our opinion the most acceptable, is that there has been an occlusion of the left internal auditory artery. A postoperative shift of the brain stem to the right, possibly delayed by the presence of oedema at the operative site, could have resulted in a stretching and subsequent thrombosis of this vessel. This theory is supported by the sudden onset of the deficit, and the fact that this artery supplies a part of both nerves and the inner ear. Occlusion of the internal auditory artery is indeed a well known cause of sudden deafness and loss of vestibular function, but it is generally not considered as a cause of peripheral facial palsy. Nevertheless, the part of the facial nerve situated in the internal auditory canal is supplied by this artery. However, there exists a great variability in the vascularisation of these structures. Usually there is more than one internal auditory artery, and the facial nerve is for the most part supplied by branches of the occipital and middle meningeal arteries and by the anterior inferior cerebellar artery. Therefore, if the internal auditory artery (or arteries) is (are) occluded, the facial nerve function will be spared in most cases. Consequently, this vascular hypothesis offers also a possible explanation for the cases only affected by a sudden contralateral hearing loss following acoustic tumour removal. These cases are probably not restricted to the two patients mentioned above. Our case was but a variation of a possible complication of cerebellopontine mass removal that, although rare, merits further attention. A better understanding of these events could lead to preventive measures to avoid such a dramatic complication.

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Sudden hearing loss and facial palsy at the contralateral side following acoustic tumour removal.

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