A syndrome consisting of a unilateral lesion of the last four cranial nerves was described by Collet\(^1\) and Sircad\(^2\) and is now called the Collet-Sicard syndrome. For the differential diagnosis, a brainstem syndrome, the Villaret syndrome, and the cervical internal carotid artery dissection are relevant. A brainstem syndrome could definitely be excluded by physical examination. The Villaret syndrome consists of a unilateral lesion of the last four cranial nerves together with an ipsilateral incomplete Horner's syndrome with miosis and slight ptosis.\(^3\) This syndrome is generally caused by a mass in the retromandibular space, especially carcinomas and sarcomas behind the parotid gland extending into the parapharyngeal space. Our patient, however, did not have Horner's syndrome. Cervical internal carotid artery dissection might result in multiple cranial nerve dysfunction often accompanied by Horner's syndrome and mostly neck pain; our patient had no pain or autonomic dysfunction.\(^4\) A fairly good recovery, as was the case in our patient, has been mentioned before.\(^5\)

Lapresle \textit{et al} reported on a patient who had a reversible vascular episode resulting in multiple cranial nerve dysfunction, probably due to catheterisation of the APA during an attempt to reach the distal external carotid artery; nine months later, this patient showed almost complete spontaneous recovery. Devoise \textit{et al} described four patients with paralysis of the lower four cranial nerves due to accidental or therapeutic embolisation in the APA during angiography. All the patients recovered or showed substantial regression of the deficits within six to nine months.\(^6\)

The vascularisation of the distal cranial nerves has been studied by Lasjaunias and Doyon\(^7\) and Lapresle and Lasjaunias.\(^8\) The APA arises from the external carotid artery and supplies the last four cranial nerves. The eleventh nerve receives dual vascularisation from the jugular as well as the musculospinal subdivision of the posterior branch of the APA, which explains why the nerve is sometimes spared in pathological events involving the APA.\(^9\) The trapezius muscle is not often involved in the CSS as was the case in our patient. Sufficient vascularisation is provided by the fact that the APA musculospinal subdivision also forms an anastomosis with the ascending cervical artery which supplies the middle cervical nerves.\(^10\)

Late onset radiation-induced motor neuron syndrome

Radiation-induced lumbosacral lower motor neuron syndrome is a rare complication of radiotherapy to lumbar fields,\(^11\) and previous reports have described its onset from four months possibly up to 13 years following radiotherapy, though detailed clinical information was not provided.\(^12\) We report a case where symptoms began 23 years after irradiation for testicular neoplasia.

In December 1964 a 26 year old electrician had radiotherapy with a total dose of 4500 rads to his testicle which had been present for two months. Histology revealed testicular teratoma and he received cobalt irradiation to pelvis, para-aortic nodes and scrotum in thirty six fractions over seven weeks to a maximum dose of 4500 rads (estimated total dose to lower end of spinal cord and cauda equina—4920 rads). Chest x ray and abdominal examination obtained normal therefor, but he developed a dusky skin reaction at the site of radiotherapy which was treated with topicals. He was followed up for nine years with no signs of recurrence. In 1973 he developed hypertension which was controlled on alaclotide. In April 1988 he developed a slowly progressive predominantly distal leg weakness which was marked on the right leg with revealed coarse fasciculations in both calves and right quadriceps. In the left leg there was mild weakness of knee flexion and extension and moderate weakness of all movements at the ankle. Power in the right leg was normal. Knee jerks were bilaterally brisk, but ankle jerks were depressed on the right and absent on the left. Sensation was normal and general