

branch of the carotid artery provides exclusive blood supply to cranial nerves X and XII, and thus carotid dissection after minor trauma may lead to sudden cranial nerve X and XII palsies. For this reason, in the present case, MR angiography and carotid triplex scan took place but did not reveal any abnormality. Additionally, Tapia's syndrome can be considered as a localized lesion at the crossing of the recurrent laryngeal and hypoglossal nerves. It has been suggested that pressure neuropathy occurs owing to hyperinflation or malposition of the cuff of the endotracheal tube within the larynx causing compression on both nerves at this crossing point [4]. In the present case, the cuff pressure was low and no nitrous oxide was administered.

Interestingly, Tapia's syndrome has been caused by central nervous system tumours. Kranianski and colleagues have reported an interesting case of hypoglossal and vagus nerve palsy in a patient with metastatic haemangiosarcoma [5]. To exclude a tumour, an MR scan of the brain was performed and did not show any lesion.

In our case, we could find no clear mechanism inducing the bilateral hypoglossal and recurrent laryngeal nerve palsy. Inadvertent hyperextension and lateral flexion of the neck at some point during sternotomy phase, in conjunction with endotracheal tube malposition, might have led to compression at the crossing point of the vagal and hypoglossal nerves, but this is speculation. Another possibility is that originally described by Boisseau and colleagues, where Tapia's syndrome developed after compression by the tracheal tube caused by displacement of the head during shoulder surgery in the sitting position [6].

We believe that this is a rare cause of failed extubation and that it has occurred in cardiovascular anaesthesia for the first time. Extreme care must be taken in the placement of the head during every procedure and in endotracheal tube position as this may result in bilateral hypoglossal and recurrent

laryngeal nerve paralysis, which should be considered possible, whenever extubation fails owing to upper airway obstruction.

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Complex regional pain syndrome in all four limbs

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EDITOR:

Few symptoms have been known by more names and have been the subject of more heated discus-

sions than complex regional pain syndrome (CRPS) type I.

A 45-yr-old male developed CRPS type I in all four limbs after having an electric shock in his right hand. To our knowledge, no case has been reported where a patient developed CRPS type I in all four limbs.

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The patient, while working with an electrical generator, received an electric shock of 6000 V when he touched a power source. He lost consciousness for a brief period and once he regained consciousness, he was transferred to hospital for wound management. He had an entry wound and vesicles in the right palm, and an exit wound in the right foot (3 cm diameter full thickness burn). He received wound care and was discharged from hospital after 12 days. During his stay in hospital, he also complained of palpitations. The electrocardiogram showed sinus tachycardia. He was started on a beta-blocker. Around 3–4 months after the incident he started to develop pain in the right upper and lower limbs and burning sensation in all the four limbs. The patient visited his General Practitioner and was initially managed by the General Practitioner. An initial diagnosis of intermittent claudication was made, and he was started on pentoxifylline, paracetamol and non-steroidal anti-inflammatory drugs (NSAIDs) and was referred to a vascular surgeon. Duplex angiography was done which was grossly normal with mild atherosclerosis in the external and internal iliac artery. Nerve conduction studies and electromyogram studies were also normal. Rheumatoid factor was also normal. The patient was then referred to an anaesthetist for pain management. A detailed history was taken. The patient mentioned that the pain first started in his right upper and lower limbs, and after a few weeks he started to get pain in the left upper and lower limbs. The pain was burning in nature. He mentioned an area of hyperalgesia in the whole of the right hand and foot, the medial side of the left arm and the left big toe. He also mentioned that he had developed oedema in his right hand and right foot a few days after the accident, which improved over the weeks. On examination, muscle strength was normal. According to the patient he was unable to sustain powerful work and felt tired early and, on occasions he also became stiff in both hands and feet. He had brittle and ridged nails in all four limbs. No change in colour was noted in all four limbs. Patchy areas of hair loss were noted in all four limbs. A decrease in muscle mass was noted in the left leg compared with the right leg. Areas of hyperalgesia and allodynia were noted in the dorsum of the right hand and forearm, on the medial side of the forearm and the middle two fingers of the left upper limb, on the entire right foot and on the medial aspect of the left foot including the ankle. He also mentioned temperature changes in all limbs compared with the rest of his body.

A diagnosis of CRPS was carried out. The patient was started on gabapentin and tramadol and a plan for intravenous regional analgesia (IVRA) was

made. The patient received an IVRA first in his right arm and then left arm followed by both lower limbs. He received 10 mg guanethidine and prilocaine 0.5% 20 mL for the upper limbs and 20 mg guanethidine and prilocaine 0.5% 30 mL for the lower limbs. He had good relief from the pain and burning sensation for a few days, but the symptoms reappeared.

During the following year, the patient received multiple IVRAs in all limbs, the dose of gabapentin was gradually increased, and amitriptyline, NSAIDs and oral opioids were added. Pain and burning sensations improved in both upper limbs. However, in both lower limbs, pain and burning sensations recurred. A computed tomography scan of the lumbosacral spine was normal, and a bilateral lumbar sympathetic block was planned. The patient received first a right-sided and then a left-sided lumbar sympathetic block with bupivacaine 0.5% 20 mL and 80 mg depomedrol. Pain on the left side improved significantly but returned in the right lower limb after 4 months. The patient received a second right-sided lumbar sympathetic block. This time, there was significant improvement. Oral medication was tapered over the months and then discontinued. During the course of his treatment, he also visited a cardiologist for sweating and palpitations. After investigations, he was started on isosorbide mononitrate and clopidogrel. The patient also visited a psychologist as he was suffering from life-threatening nightmares like falling from a bridge and drowning. He was diagnosed with posttraumatic anxiety disorder and was started on dosulepin. Today, the patient has fully improved from his symptoms; however, he still feels occasional pain in his legs after walking.

The acute effects of electrical injuries are well known. However, the occurrence and mechanism of delayed sequelae are still unclear. The effects on the peripheral nerves and the sympathetic system are poorly documented [1]. CRPS type I, formerly known as reflex sympathetic dystrophy (RSD), is a symptom complex characterized by pain, hyperaesthesia and vasomotor instability. CRPS type I has long been considered a manifestation of sympathetic nervous system dysfunction because of characteristic oedema and vasomotor instability in addition to frequent alleviation of symptoms by sympathetic blocks. CRPS generally affects one limb although it is recognized that the contralateral limb is also abnormal and is at a risk of developing clinical CRPS symptoms without the advent of a new injury [2]. Our case and previous cases reported of patients developing CRPS in multiple limbs underscore the importance of the central nervous system in the pathophysiology and suggests this central influence is not

confined to one or adjacent segments of the spinal cord [2]. Many aspects of bilateral presentation or recurrences of the syndrome are unknown, and, for this reason, 1183 consecutive patients with syndrome were analysed [3]. In 10 patients, the syndrome started in symmetrical limbs, in 34, it recurred in the same limb after a period of no or few complaints, and in 76, it recurred in one or more limbs other than the first limb. Involvement of a second limb concerned the symmetrical limb in 47%. The diagnosis of RSD was in one report based on four of five symptoms: pain, oedema, altered skin temperature, altered skin colour or reduced range of motion extremity [4]. These criteria are controversial [5]. Our patient had pain in all four limbs and this is the main symptom for diagnosis of CRPS [5]. Swelling or oedema of the affected limb is also reported to be characteristic; however, in a large prospective trial, only a history of swelling was needed even though the examination did not reveal the swelling [4]. No skin colour change was reported by our patient. In one study, 70% of the patients reported skin colour changes; however, examiners found absence of colour change in 69% of the patients [6]. Our patient met all the diagnostic criteria for CRPS type I. He also developed psychological problems, which are often found in these patients; this may lead to the conclusion that psychological problems may play a role in the pathogenesis of the disorder [7]. Our patient improved over the years with pharmacological treatment, sympathetic blocks and psychological therapy. In conclusion, despite many reports on RSD, there

remains much controversy about the disorder. There is no convincing evidence that a primary organic dysfunction of the nervous system exists.

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Endocrine response to cataract surgery under total intravenous anaesthesia, local anaesthesia under sedation or local anaesthesia alone: a comparative study

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EDITOR:

There is evidence that cataract surgery, although a minor operation, elicits a neuroendocrine response. So far, the attempts to correlate stress response with

cataract surgery have been focussed mainly on cortisol, glucose and catecholamine release [1,2]. On the other hand, it has been reported in the literature that surgical operations and diagnostic procedures under either general anaesthesia or sedation are associated with a transient increase in serum prolactin levels [3] or an increase in thyroid-stimulating hormone (TSH) levels [4]. The present study was conducted to compare the influence of local anaesthesia alone or under sedation with propofol vs.

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