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Prevention of tourniquet paralysis during the use of Pneumatic tourniquets

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Summary This article focuses on the prevention of tourniquet paralysis that may arise as a result of use of modern pneumatic tourniquets. Tourniquet paralysis is an injury caused by pneumatic tourniquet resulting from mechanical pressure on the nerves and anoxia. The injury can range from paraesthesia to complete paralysis. The motor functions are usually affected with sparing of sensation. High risk groups for tourniquet paralysis include older patients, hypertensive, obese patients and those with atherosclerosis. Adequate knowledge of the complications that can occur while using tourniquets is important to enable nurses to prevent and detect them early.
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Introduction

The tourniquet is a device used to create a bloodless surgical field, enabling visualization of the anatomical structures during surgical procedures. This is especially useful in the field of hand and microsurgery, plastic surgery and orthopaedic surgery. The use of tourniquets also reduces intra-operative blood loss which is a potential major complication of orthopaedic surgery. As the patient's blood pressure can fluctuate during surgery, some surgeons adopt a 'one glove fits all' approach by applying 300—350 mm Hg of tourniquet pressure for the lower limb and 200—250 mm Hg for the upper limb. The use of a tourniquet is not without its complications; the most common ones reported in the literature are nerve injury, post tourniquet syndrome, compartment syndrome, skin damage, chemical burns and thrombosis.

This article focuses on the prevention of tourniquet paralysis that can occur as a result of the use of the modern pneumatic tourniquet. This is an injury caused by the use of pneumatic tourniquets, resulting from mechanical pressure on the nerves and surrounding structures. The injury can range from paraesthesia to complete paralysis (Middleton and Varian, 1974; Flatt, 1972; Rorabeck and Kennedy, 1980). The occurrence is more common in the upper limb, especially involving the radial nerve. Involvement of the lower limb is less common. High risk groups for tourniquet paralysis include older patients, patients with hypertension, those who are obese and those with atherosclerosis (Kam et al., 2001).

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Pathophysiology

Tourniquet paralysis results from excessive pressure (Klenerman, 1983). The pathophysiology of localized nerve conduction block as a result of direct pressure on the nerve has been discussed by various authors over many decades. When they explored three cases of tourniquet paralysis, Spiegel and Lewin (1945) noted a reduction by one half to one quarter in the size of the diameter of the nerve at the site of compression. Aho et al. (1983) described a case of tourniquet paralysis that occurred due to a faulty tourniquet gauge. Brunner (1951) described palsy that occurred after using a tourniquet that had an error where the cuff pressure was twice that required. Mayor and Denny-Brown (1964) studied the velocity of nerve conduction at the site of the tourniquet and found it to be reduced when compared to the velocity proximal and distal to the device. Ochoa et al. (1972) found displacement of the Nodes of Ranvier which was at its most severe under the edges of the cuff with less displacement under the centre, suggesting that a pressure gradient may be responsible for the displacement. Rudge et al. (1974) suggested that the conduction block in the peripheral nerve occurs as a result of direct mechanical effect from the pressure applied on the nerve fibres. Fowler et al. (1972) performed experiments with baboons and showed that there was constant blocking of nerve conduction when the tourniquet pressure was 1000 mm Hg. There was minimal conduction block when the pressure was 500 mm Hg, and no persistent conduction block when the pressure was 250 mm Hg. This all suggests that tourniquet paralysis results from excessive pressure.

The larger nerve fibres are usually affected. Motor functions are usually affected without affecting sensation. Since smaller diameter fibres are spared, pain, temperature and autonomic function are usually preserved. Permanent deficits rarely occur and most lesions heal in less than 6 months (Bolton and McFarlane, 1978). Motor deficits usually take longer to recover when compared to sensory deficits. In the upper limb the radial nerve is the most susceptible, followed by the median and ulnar nerves.

Clinical diagnosis

The clinical diagnosis of tourniquet paralysis is made in the postoperative period when the patient is not able to move any part of the upper limb below the elbow. There may be numbness and reduced sensation in the fingers. The clinical picture can vary depending on which nerve is affected. Wasting of the forearm muscle may be detected at a later stage. To differentiate between a true division of the nerves and tourniquet palsy (which is the paralysis of the muscles of the upper or lower limb resulting from the use of the tourniquet), regular assessment of sensory and motor symptoms must be undertaken. In tourniquet palsy the sensory changes recover rapidly while motor recovery takes longer. Such dissociation between sensory and motor symptoms is suggestive of tourniquet palsy as opposed to complete division of the nerve fibres (Eckhoff, 1931), but diagnosis is often less clear cut in the clinical situation. It is important to differentiate between tourniquet palsy and division of nerves because in the latter urgent surgical intervention is required.

Pre-operative assessment should always be conducted so that pre-existing nerve lesions can be ruled out postoperatively. Lucas and Davies (2005) have described some simple methods to clinically assess the sensory and motor functions of the radial, ulnar and median nerves. Orthopaedic theatre nurses/practitioners and anaesthetic nurses/practitioners can be taught how to undertake a quick assessment of the 3 main nerves of the upper limb — namely the radial, ulnar and median nerves — as part of the pre-operative checklist. Any existing neurological deficit can then be brought to the attention of the surgeon. This can be charted on the anaesthetic record sheet and used for later reference if required. The surgeon should also be responsible for performing a thorough pre-operative assessment to detect any pre-existing neurological deficits.

Investigations

Seddon in 1943 (Andrew Kaye, 1991) classified nerve injuries into three grades, neuropraxia, axonotmesis, and neurotmesis (Table 1) based on the severity of the lesion. A nerve conduction study is useful to confirm the diagnosis. In the case of true division of the nerve, the nerve conduction findings will be suggestive of neurotmesis where the nerve conduction distal to the site of injury is absent and the motor unit action potential is absent, besides this there is no anatomical continuity in the nerve. The findings in tourniquet paralysis usually correspond to neuropraxia in which the nerve conduction distal to the site of injury is present and the motor unit action potential is absent. However unlike neurotmesis there is anatomical continuity of the nerve. Gilliatt (1980) described a double conduction block in which there is a nerve

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