Available online at www.sciencedirect.com
ScienceDirect
The Foot 18 (2008) 192–197
The Foot
www.elsevier.com/locate/foot

Local anaesthetic toxicity

Anthony J. Maher*, Stuart A. Metcalfe, Steve Parr

Department of Podiatric Surgery, Solihull Care Trust, chelmsley Wood Primary Care Centre, Crabtree Drive, Chelmsley Wood, Birmingham B37 5BU, United Kingdom

Received 5 November 2007; accepted 18 May 2008

Abstract

Background: Local anaesthesia has been used by British podiatrists since the early 1960s. The use of local anaesthesia has allowed for the advancement of scope of practice and the development of podiatric surgery. Local anaesthesia is however associated with potential risks and adverse reactions including toxicity. Objectives: To review the current literature on the subject of local anaesthetic toxicity and to consider recent developments in the management of acute toxicity. Conclusions: Local anaesthesia although safe has the potential to cause serious harm in the event of toxicity. Appropriate steps should be taken to minimise the risk of toxicity and should it occur measures should be applied to minimise the consequences of toxicity. Such measures may include the use of Intralipid for resuscitation.

Keywords: Local anaesthetics; Bupivacaine; Toxicity; Intralipid; Podiatry

1. Introduction

The profession of podiatry began using local anaesthetics in the early 1960s [1], however early progress was hampered by the restrictions laid in law by the Medicines Act of 1968 and a report by the Chiropodists board in the same year which frowned on the use of local analgesics by podiatrists [1,2]. These early concerns related to a lack of formalised training in the administration of local anaesthetics [2]. In response to the restrictions a London-based Group of Podiatrists named the Croydon Post-graduate Group developed courses in local anaesthesia with the cooperation of consultant anaesthetists and the support of the Society of Chiropodists [1–3]. In January 1972, the use of local anaesthetics was finally approved by the Chiropodists Board allowing mainstream use of local anaesthesia by the profession, with appropriate training [1,3].

Initially the Department of Health expressed concerns regarding the non-medical administration of local anaesthetics and as such placed restrictions on the maximum dosage which could be administered in a 24-h period [1,3]. The Society of Chiropodists successfully fought against this restriction and in 1980 the restriction was lifted by the Department of Health [1] allowing podiatrists to use their clinical judgement [3]. Local anaesthetic techniques are now taught as an integrated element of the undergraduate Podiatry program allowing podiatrists to administer local anaesthetics on completion of their degree training [3]. The restrictions of the Medicines Act of 1968 allowed podiatrists access to a specific range of local anaesthetic agents; bupivacaine and lidocaine with and without adrenaline, prilocaine and mepivacaine [4]. The act was recently amended by Statutory Instrument number 2807 extending the list of local anaesthetics to include ropivacaine and levobupivacaine [4,5].

Access to local anaesthetics allowed for an extension of scope of practice, podiatrists were now able to legally administer local anaesthetic digital blocks and local infiltrations [2]. This in turn allowed for the development of new skills in nail surgery and minor skin procedures [2]. With time some quarters of the profession began to develop their scope of practice to include surgical techniques for the management of deformity [2]. With the advancement of Podiatric Surgery, clinicians began to administer mayo blocks, and ankle blocks allowing for the development of forefoot surgery skills. More recently popliteal regional nerve blocks have fallen into favour with UK podiatric surgeons. These advanced blocks allow the undertaking of reconstructive hind foot surgery. The popliteal regional nerve block also provides markedly

* Corresponding author. Tel.: +44 7894582901.
E-mail address: anthony_maher79@yahoo.co.uk (A.J. Maher).

0958-2592/$ – see front matter © 2008 Elsevier Ltd. All rights reserved.
doi:10.1016/j.foot.2008.05.002

improved postoperative pain relief for patients undergoing any foot surgery. Both short acting and long acting local anaesthetics are used for a variety of indications from simple nail procedures through to major surgical reconstruction of the foot.

Of particular significance is that the local anaesthetic blocks utilised in podiatry are routinely administered by the podiatrist undertaking the treatment or in the case of podiatric surgery, an appropriately trained podiatrist team member often administers the block. This is in stark contrast to medicine where an anaesthetist would usually be expected to administer the local anaesthetic agent and significantly the anaesthetist would be expected to take responsibility for and manage any medical complications arising from the administration of the local anaesthetic agent.

The responsibilities and risks faced by an anaesthetist are obviously no different to those faced by podiatrists administering local anaesthetics. Therefore, podiatrists must be trained and able to appropriately assess and manage the potential complications of local anaesthesia.

2. Complications of local anaesthesia

Two categories of local anaesthetics exist: esters and amides. Ester-based anaesthetics are more prone to causing allergic reactions and are not routinely used by podiatrists [6,7]. The amide anaesthetics are safer agents [6] but complications can and do occur. Complications can be divided into those which are specific to certain agents and those which apply to all amide local anaesthetics. Hypersensitivity reactions or allergic reactions can occur with all local anaesthetics, these tend to manifest as allergic dermatitis [6]. Rarely acute anaphylaxis may develop and again the clinician should be prepared to manage the consequences of anaphylaxis [6,8]. Perhaps the most sinister complication of local anaesthesia is acute toxicity impacting on the cardiovascular and central nervous system [9]. Toxicity may be a consequence of excessive dosage, pathological states such as liver failure, or more commonly as a direct result of accidental intravascular injection [9,10].

2.1. The incidence of toxicity

In UK alone three deaths were reported between 2000 and 2004 as a direct consequence of intravenous bupivacaine administration [10]. In epidural anaesthesia the incidence of toxicity was found to be 12/100,000 while brachial blocks which are perhaps similar to the popliteal regional nerve block suffered a toxicity incidence of 200/100,000 [11]. A large Japanese study found a toxicity incidence of 1.17/100,000 and a fatality rate of 0.023/100,000 [9,12]. The authors are unaware of any reports of local anaesthetic toxicity in the podiatry literature, however both ankle blocks and regional popliteal nerve blocks require the administration of local anaesthetics in relatively close proximity to venous and arterial vessels increasing the risk of toxic reactions.

2.2. Signs and symptoms of toxicity

Toxicity following an intravascular injection may develop rapidly whereas a perineural injection of local anaesthetic may result in a much slower onset of symptoms. Typically the patient would be expected to develop CNS signs of toxicity before cardiovascular symptoms occur, however this may not be the case with an intravascular injection [6,13,14]. Local anaesthetics are membrane stabilising drugs which inhibit voltage gated ion channels [6,10]. These channels are not only found in the peripheral nerves but also in cardiac tissue and the central nervous system [6]. Therefore, if the local anaesthetic were to come into contact with these tissues some impairment of function would follow. However, the specific nature of the toxicity varies amongst the different agents [7,9,10]. Lidocaine for example produces a progressive contractile failure, while bupivacaine may cause lethal dysrythmias [9]. The more recent agent levobupivacaine has caused fatalities through both mechanisms [9].

2.3. Central nervous system toxicity

The earliest sign of toxicity is a consequence of CNS stimulation and manifests as the patient becoming talkative, excited and euphoric [16]. The excitatory effects continue with tingling, tinnitus, numb tongue, light headedness, twitches, shivering and slurred speech. These are gradually replaced by loss of consciousness and convulsions and eventually coma [13,15,17]. Coma may be followed by inhibition of respiration and respiratory failure [7,18].

2.4. Cardiovascular toxicity

Severe CNS toxicity with respiratory collapse will inevitably lead to cardiovascular collapse, however local anaesthetics may also directly cause cardiovascular toxicity [16]. Cardiovascular toxicity typically develops at a much higher blood concentration than required for CNS toxicity [19]. High doses of local anaesthetics suppress the spontaneous pacemaker activity [19] increasing the refractory period of atrial, and ventricular tissue and the Purkinje fibres [14]. This cardiac suppression can eventually result in sinus bradycardia and sinus arrest [19]. Cardiovascular toxicity is demonstrated on ECG by prolonged PR interval, widened QRS and prolonged ST interval [20,21]. Local anaesthetics also exert a negative inotropic effect on cardiac tissue decreasing the force of contraction [19]. Clinically arrhythmias will become evident as the heart gradually loses its automaticity leading to ventricular arrest [15]. The local anaesthetic agent bupivacaine is capable of producing profound cardiovascular depression particularly when administered intravascularly [19].