All diagnosis depends on a combination of three processes; history with examination, investigation, and observation. At each stage anxieties may be raised or reduced. Results from investigations should be used to complement the clinical findings rather than replace assessment and re-evaluation of the patient.

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REFERENCES

1 **Olesch CA**, Knight GJ. Invasive meningococcal infection in Western Australia. *J Paediatr Child Health* 1999;**35**:42–8.
2 **Bonsu BK**, Harper MB. Utility of the peripheral blood white cell count for identifying sick young infants who need lumbar puncture. *Ann Emerg Med* 2003;**41**:206–14.
3 **Borschsenius F**, Bruun JN, Michaelsen TE, *et al*. Serum C-reactive protein in systemic infections due to Neisseria meningitidis. *Norwegian Institute of Public Health Annals* 1986;**9**:15–21.

Lignocaine toxicity; a complication of local anaesthesia administered in the community

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Local anaesthetic agents are among the most commonly used drugs in the emergency department. A case is reported of a complication arising from systemic toxicity after the injection of lignocaine (lidocaine). Emergency practitioners should maintain a high level of vigilance for the symptoms of toxicity after local anaesthetic administration. Particular care should be taken when calculating the maximum safe dose for young children.

A 7 week old male infant was brought from home by a paramedic crew to the emergency department having undergone a circumcision by the family general practitioner. A dorsal penile nerve block (DPNB) was the analgesic technique used and lignocaine (lidocaine) was the anaesthetic agent. The parents of the infant phoned for an ambulance after a series of generalised seizures. The onset of these was about 20–30 minutes after the block had been administered.

On examination, the infant was able to maintain his airway; he was breathing spontaneously and had a capillary refill time of less than two seconds. He was unresponsive, hypertonic, and generalised seizure activity was noted. There was some fresh blood around the base of the penis, but no evidence of any rash or fever.

The first line treatment was oxygen and intravenous benzodiazepines at the appropriate dose.

On further questioning, we gathered that 3 ml of 1% lidocaine (30 mg) had been used for the DPNB and using the Oakley chart we estimated the infant's weight to be 4.5 kg. The maximum safe dose of lidocaine is 3 mg/kg (table 1) and therefore the maximum safe dose for this patient was calculated to be 13.5 mg (1.35 ml 1% lidocaine). A diagnosis of lidocaine toxicity was made.

Supportive treatment was continued for 45 minutes and during this time, the infant remained generally hypertonic but with no further seizure activity. After one hour, his tone had returned to normal and his condition stabilised. Two hours after he presented to the emergency department, he was fit for transfer to the ward.

The remainder of his stay was uneventful and he was permitted home the following day. At follow up two months later, there were no obvious ill effects and there were no developmental abnormalities.

DISCUSSION

Lidocaine is one of the most widely used local anaesthetic agents in the emergency department. It has been used safely and effectively for almost every possible type of local anaesthetic procedure. It has a rapid onset and it is effective for about 30–60 minutes in its plain form (or up to 90 minutes when used with a vasoconstrictor).

Lidocaine is a tertiary amine that is an amide derivative of diethylaminoacetic acid. Allergic reactions to the amide group of local anaesthetics are extremely rare and the vast majority of adverse reactions result from systemic toxicity. The symptoms are an extension of the drug's pharmacological action.

There are few reports of local anaesthetic toxicity in infants and children. However, seizures, arrhythmias, cardiac arrest, and transient neuropathic symptoms have been reported.[1] Lidocaine toxicity has been reported after subcutaneous administration, oral administration, and intravascular injection.[2–4]

A retrospective study of 1358 circumcised male infants revealed that 1022 had a DPNB as the anaesthetic technique.[5] In these, there were no reports of local anaesthetic toxicity. A meta-analysis of injection related adverse effects during DPNB by injected lidocaine revealed no reports of systemic toxicity from injected local anaesthesia.[6] Similarly, a study of 133 patients to evaluate the efficacy and safety of intravenous regional anaesthesia revealed no reports of lidocaine toxicity.[7] In this study, intravenous regional anaesthesia was used in the treatment of unilateral closed fractures and dislocations of forearm or wrist in children and 0.5% lidocaine solution (3 mg/kg intravenously) was the agent used.

Despite the apparent safety of lidocaine, it is a widely held view that extra care should be taken when administering local anaesthesia to young children as it can be easy to overestimate the dose to weight ratio.

Peak blood levels of lidocaine usually occur 10–25 minutes after injection. This is the point at which the toxic effects are most likely to be observed. The onset of symptoms is faster if accidental intravascular injection has occurred. The first symptoms and signs of local anaesthetic toxicity are usually

Table 1 Chart showing the maximum safe dose of anaesthetic

Local anaesthetic	Maximimum dose	Duration of action (h)
Bupivicaine	2 mg/kg	2–4
Lidocaine	3 mg/kg	1
Lidocaine/vasoconstrictor (avoid near end organ)	5 mg/kg	1.5
Prilocaine	6 mg/kg	1.5

neurological with numbness of the mouth and tongue. Shortly afterwards, there is the onset of tinnitus, confusion, seizures, and potentially coma.

Cardiovascular toxicity usually manifests itself as tachycardia and hypertension but with increasing toxicity bradycardia and hypotension occur. Ventricular arrythmias and cardiac arrest are also known side effects.[4]

The treatment of local anaesthetic toxicity is essentially supportive. The airway should be maintained and oxygen should be administered. Monitoring of blood pressure and ECG is mandatory. If convulsions occur they should be controlled with benzodiazepines along established guidelines. Bradycardia is usually self limiting, but if persistent and associated with hypotension, atropine and cardiac pacing may be necessary. The symptoms of toxicity persist as long as the plasma concentrations remain above the therapeutic index. Seventy per cent of the dose is metabolised in the liver and less than 10% is excreted unchanged in the urine with an excretion half life of 90–110 minutes. The toxicity from lidocaine is of shorter duration than from other agents with a longer half life, for example, bupivicaine.

In conclusion, this case highlights the need for vigilance for symptoms of systemic toxicity when administering any local anaesthetic. Particular care must be taken when they are used in young children. It is essential that the weight of the child is measured before starting any procedure that entails local anaesthesia. If the weight cannot be measured, then an estimate should be made using well established guidelines.

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REFERENCES

1 **Gunter JB**. Benefit and risk of local anesthetics in infants and children. *Paediatr Drugs* 2002;**4**:649–72.
2 **Alfano SN**, Leight MJ, Skiendzielewski JJ. Lidocaine toxicity following subcutaneous administration. *Ann Emerg Med* 1984;**13**:465–7.
3 **Smith M**, Wolfram W, Rose R. Toxicity seizures in an infant caused by (or related to) oral viscous lidocaine use. *J Emerg Med* 1992;**10**:587–90.
4 **Brown DL**, Skiendzielewski JJ. Lidocaine toxicity. *Ann Emerg Med* 1980;**9**:627–9.
5 **Fotaine P**, Dittberner D, Scheltema KE. The safety of dorsal penile nerve block for neonatal circumcision. *J Fam Pract* 1994;**39**:243–8.
6 **Taddio A**. Pain management for neonatal circumcision. *Paediatr Drugs* 2001;**3**:101–11.
7 **Colizza WA**, Said E. Intravenous regional anesthesia in the treatment of forearm and wrist fractures and dislocations in children. *Can J Surg* 1993;**36**:225–8.

Delayed postanoxic encephalopathy after carbon monoxide poisoning

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Delayed postanoxic encephalopathy causes deterioration and relapse of cognitive ability and behavioural movement a few weeks after complete recovery from initial hypoxic injury. A case is reported of delayed postanoxic encephalopathy after carbon monoxide poisoning, which was diagnosed with diffusion weighted magnetic resonance imaging. The literature is also reviewed.

A 65 year old man was admitted to the emergency department (ED) with memory impairment and movement disturbance. One month previously he had presented to our ED with a drowsy mental status because of carbon monoxide (CO) poisoning after he had attempted suicide by a briquette fire. However, he had recovered with normobaric oxygen therapy that resulted in a decrease in the blood carboxyhaemoglobin level from 30.5% on arrival to 2.3% just before discharge. On his second presentation, he had no other remarkable medical history. His blood pressure was 120/80 mm Hg, heart rate 82/min, and respiratory rate 24/min. He was alert, comparatively well coordinated, but slightly disoriented with regard to time and person. He showed no neurological abnormality apart from parkinsonian movement.

Diffusion weighted magnetic resonance imaging (DWMRI) showed diffuse high signal intensity in both periventricular and deep white matter (fig 1). He was admitted to the neurology ward for supportive care, and discharged on the

Abbreviations: ED, emergency department; CO, carbon monoxide; DPE, delayed postanoxic encephalopathy; DWMRI, diffusion weighted magnetic resonance imaging