Changing incidence of non-accidental injury to children in South Glamorgan

The increasing number of reports in the media of severe or sometimes fatal non-accidental injury to children suggests that the incidence of this problem may have increased. A previous paper examined the incidence of child abuse in South Glamorgan from 1970 to 1981,¹ and this report is an update of that study.

Methods and results

We examined details of all referrals during 1982-5 in which non-accidental injury had been diagnosed. The procedure for child abuse in South Glamorgan requires that all children who are suspected of having suffered non-accidental injury are medically examined. All children with suspected or proved injury are admitted to hospital. Within 72 hours after referral there is a case conference, which is attended by a senior paediatrician. After considering medical reports and contributions from other agencies those at the case conference decide whether a diagnosis of non-accidental injury can be upheld. This process of reaching a diagnosis of non-accidental injury has not changed appreciably during the past 15 years.

The data for this report were taken from the minutes of the case conferences; copies of all these minutes are received and monitored centrally by the child abuse coordinator (JJ) for South Glamorgan Social Services. The age range of the children was 0-15 years. Injuries were divided into two groups: severe injuries, which included all fractures, internal injuries, and intracranial bleeding, and moderate injuries, which included all soft tissue injuries.

The figure shows a rapid increase in the number of children referred with moderate injuries after 1973. In 1974 there was a ninefold increase in the number of children aged 0-4 years who were referred with moderate injuries; subsequently an average of 22 children in this age group were referred each year. The number of children aged 5-9 who were referred with moderate injuries increased from four in 1974 to 18 in 1975; after 1974 there was an average of 15 referrals each year. The referral of children aged 10-15 with moderate injuries was a later phenomenon: during 1970-7 the average number of cases referred each year was 1.9 but after 1977 the average number of referrals increased to 13, showing that child abuse is no longer a syndrome affecting only infants.

Incidence of non-accidental injury to children, 1970-85. Vertical broken line indicates when child abuse procedures were introduced.

Comment

The report of the inquiry into the killing of Maria Colwell by her stepfather was published in 1974.² It was followed by a circular from the Department of Health and Social Security that urged local authorities to set up area review committees to be responsible for devising local multidisciplinary procedures and setting up child abuse registers.³ In South Glamorgan the existing procedures were revised and were circulated to many more people than before. Many training programmes were instigated for all those working in the care of children, with an emphasis on increasing the awareness of the problem and recognising and acting on warning signs. This resulted in earlier intervention, and the sharp rise in the number of referrals for moderate injuries occurred at this time. This rise therefore seems to have been due to the combination of an emphasis on publicity, improved management, and increased training in child abuse. Teachers, in particular, participated for the first time in training and procedures, which explains most of the increase in the number of referrals of school aged children. The drop in the number of children referred with severe injuries in the mid-1970s was sustained. One explanation for this might be the increased reporting of

children with suspected or proved moderate non-accidental injuries; this ensures that treatment begins before a more serious injury occurs and gives an opportunity for effective preventive work.

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Fatal bronchospasm after topical lignocaine before bronchoscopy

Fibreoptic bronchoscopy has a low morbidity and mortality. We report the first case of death due to hypersensitivity to topical local anaesthetic (lignocaine; 10% Xylocaine spray) given during routine premedication for this procedure.

Case report

A 67 year old man with chronic bronchitis and emphysema presented with increasing dyspnoea and weight loss. He had severe airflow obstruction (forced expiratory volume in one second 0.5 l, forced vital capacity 1.7 l). A chest radiograph was compatible with the diagnosis of emphysema but showed no suggestion of a carcinoma. An electrocardiogram showed sinus rhythm with occasional ventricular extrasystoles. Arterial oxygen tension was 7.6 kPa and arterial carbon dioxide tension 6.4 kPa when he was breathing air. Because of the weight loss bronchoscopy was arranged to exclude carcinoma.

He was admitted fasting and given nebulised salbutamol (5 mg) one hour before bronchoscopy and atropine (0.6 mg) intravenously as premedication without adverse effect. Two puffs of lignocaine (20 mg) were administered to one nostril and six puffs (60 mg) to the posterior pharyngeal wall. Two minutes later he developed wheezy dyspnoea and cyanosis, which were not relieved by inhaled salbutamol, and one minute later he suffered a cardiorespiratory arrest. External cardiac massage was started with manual ventilation through a face mask and oropharyngeal airway and subsequently a 7.5 mm endotracheal tube inserted by an anaesthetist. There was no evidence of laryngospasm, although manual ventilation was difficult, possibly because of severe bronchospasm. There was no evidence of aspiration of stomach contents. Sodium bicarbonate 8.4% solution (100 ml) was infused followed by 10 ml of 1/1000 adrenaline and 10 ml calcium chloride. This produced ventricular fibrillation; cardioversion (340 joules) and an intravenous bolus of 100 mg of lignocaine resulted in asystole. Despite treatment with intracardiac adrenaline and calcium chloride, isoprenaline 200 µg, and two further doses of adrenaline he remained asystolic. Twenty minutes after the onset of cardiorespiratory arrest attempts at resuscitation were stopped.

Necropsy confirmed the presence of a left sided pneumothorax with extensive centrilobular emphysema. Microscopy showed hypertrophy of smooth muscle and thickening of the basement membrane in the small airways.

Comment

Although we suspected that this man died of a hypersensitivity reaction to topical lignocaine, other factors had to be considered: atropine could have precipitated a ventricular arrhythmia, although his condition did not deteriorate until 12 minutes after its administration, tachycardia was not observed beforehand, and he was asystolic. The pneumothorax could have been due to the cardiac needle, cardiac massage, or manual ventilation in the presence of emphysema but is unlikely to have been an initiating factor for this event. In view of the short interval between the use of lignocaine spray and the onset of wheezing we concluded that the patient died of a hypersensitivity reaction to the local anaesthetic.

A survey of the complications of 24 000 fibreoptic bronchoscopies gave the incidence of major complications as 0.08% with a mortality of 0.01%.¹ No deaths were due to lignocaine, although one patient died of seizures after excessive use of local anaesthetic spray.

The manufacturers of lignocaine recommend a maximum dose of 200 mg for a 70 kg man. Our patient weighed 45 kg and was given a total of 80 mg.