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SHORT REPORTS

Effect of seat belt legislation on the incidence of sternal fractures seen in the accident department

In the United Kingdom legislation on the compulsory wearing of seat belts was introduced on 1 February 1983. Subsequently the number of front seat occupants of vehicles wearing seat belts increased dramatically. There is no doubt that seat belts correctly worn greatly reduce the incidence of serious and fatal injuries in automobile accidents,[1][2] and in Cambridge a reduction of 20-25% has been seen since legislation.

Seat belts may, however, be associated with certain injuries[3]—for example, fractured sternums—and since legislation we might expect to see an increased incidence of these.[4][5] At Addenbrooke's Hospital, Cambridge, I noticed that a comparatively large proportion of front seat car occupants hurt in automobile accidents were presenting with anterior chest pain and were found to have sternal fractures. The aim of this study was therefore to determine whether the incidence of patients presenting to the casualty department with a sternal fracture had increased as a result of seat belt legislation.

Method and results

The data were collected from the casualty records of Addenbrooke's Hospital (which sees about 40 000 casualties a year). All patients seen in the casualty department for injuries received in automobile accidents are documented as such. During 1 February 1981 to 31 January 1983 and 1 February 1983 to 31 January 1985—that is, the two years before and after legislation—all patients sustaining injuries as front seat occupants in automobile accidents were identified. Those who received chest injuries and also those who received multiple injuries where a chest injury was not specifically recorded had their records drawn. From these it was determined which patients had sustained a sternal fracture and whether or not they had been wearing a seat belt at the time of their accident.

Also included were patients brought into hospital dead as a result of an automobile accident and those admitted but who subsequently died of their injuries. From necropsy reports it was determined if any had sustained a sternal fracture (table).

Comparison of the two study periods showed a highly significant increase in the number of patients presenting with sternal fractures since legislation ($\chi^2 = 13\cdot14$; $p < 0\cdot001$). About 90% of these fractures occurred in patients claiming to have been wearing a seat belt at the time of their accident. By contrast, there was no significant change in the overall incidence of all chest injuries in the two periods ($\chi^2 = 0\cdot21$; $p > 0\cdot5$). This last finding agrees with other studies.[5]

Numerical data on front seat occupants of vehicles admitted to casualty department after road traffic accidents during two years before and two years after introduction of compulsory wearing of seat belts (1 February 1983)

	February 1981 to January 1983	February 1983 to January 1985
No of patients seen in casualty department after automobile accidents	1970	1620
No with chest injuries	226	178
Fractured sternums:		
Total	7	24
Belted	3	21
Unbelted	3	2
Unknown	1	1
Driver	6	16
Passenger	1	8
Mean age (range)	39 (18-61)	58 (34-84)
Male: female ratio	5:2	2:1

Comment

Before wearing seat belts became compulsory most sternal fractures were caused by the steering wheel or dashboard in unrestrained front seat occupants. Some people sustained fatal, multiple injuries and never reached the casualty department, having been certified dead at the accident site. These deaths accounted for about 5-10% of all deaths among front seat occupants (the latter totalling about 35 a year before legislation) and the proportion was similar in the period after legislation. Although the number of deaths before legislation was greater by about 20%, probably the number of sternal fractures in these people was not greatly different between the two periods. Lack of information on this relatively small group would not alter the significance of the increase in sternal fractures seen since seat belt legislation.

My study shows a threefold increase in the incidence of sternal fractures since legislation, coupled with an increase in the number of people surviving high speed automobile accidents and also a pronounced increase in the number of people wearing seat belts. Most patients sustaining a sternal fracture while wearing a seat belt received no other serious injury and the fracture was uncomplicated.

Evidence to date confirms the great benefit of correctly worn seat belts in automobile accidents. It therefore seems reasonable to conclude that an uncomplicated fracture of the sternum, albeit painful, is an acceptable price for the compulsory wearing of seat belts in exchange for more serious multiple injuries or even death.

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Cyanide toxicity after immersion and the hazards of dicobalt edetate

We report on a patient with cyanide toxicity who developed severe oedema after treatment with dicobalt edetate.

Case report

A 43 year old industrial chemist was admitted to casualty 15 minutes after total immersion for three minutes in a vat containing 1000 gallons (4546 litres) of hot cupric cyanide. On arrival he was deeply unconscious, cyanosed, and breathing irregularly. Oxygen 100% was administered by mask and intravenous infusion begun. His contaminated clothing was removed and the "cyanide box" in casualty opened. Arterial blood was obtained for measurement of gas tensions; cyanide, urea, electrolyte, and glucose concentrations; and full blood count. In accordance with the instructions he was given 300

mg dicobalt edetate over three minutes and, as his condition did not improve, a further 300 mg again over three minutes.

He then developed periorbital oedema, which was thought to have been caused by immersion in the hot vat rather than to be a reaction to the drug. He was given 200 mg hydrocortisone intravenously, and laryngoscopy after 20 mg etomidate showed early laryngeal oedema. Intubation was performed using an orotracheal cuffed Portex 9·0 tube (under suxamethonium relaxation and with cricoid pressure). Laryngoscopy 60 seconds later showed gross laryngeal oedema obscuring the cords and epiglottis. Immediately after successful atraumatic intubation gentle tracheal suction produced copious pink frothy fluid.

He received sodium bicarbonate 100 mmol(mEq)/l intravenously to correct his acidosis (pH=6·9, base excess=−20 mmol(mEq)/l), further arterial samples were taken (from a left radial arterial catheter), and a catheter was inserted in the right internal jugular vein to assess central venous pressure. There was still no improvement, and three ampoules of amyl nitrite were broken into a reservoir bag and administered while sodium thiosulphate and sodium nitrite were given intravenously.

Clinical evidence of pulmonary oedema was now overwhelming, and intermittent positive pressure ventilation was started using a Manley Servovent and pancuronium bromide and papaveretum to aid ventilation. Effective ventilation required positive end expiratory pressure of 5 cm H₂O. A catheter was inserted, 450 ml of contaminated urine drained, and the catheter left in situ to measure urine output. After gastric lavage 100 ml thiosulphate solution was left in his stomach. Blood gas tensions, cyanide and glucose concentrations, and packed cell volume were measured, and he was transferred to the intensive care unit.

He had severe facial oedema, and it was impossible to assess his pupillary reflexes or open his mouth. His central venous and blood pressure were satisfactory, but he showed peripheral vasoconstriction and gross plasma extravasation (packed cell volume 0·75, haemoglobin concentration 200 g/l, white cell count 40×10⁹/l and had a low urine output (0·5 ml/kg). Four litres of human plasma protein fraction were transfused over two hours with no change in his central venous pressure (+8 cm H₂O). His urine output returned to normal, and his peripheral perfusion improved. His blood glucose concentration rose to 16 mmol/l (288 mg/100 ml) over six hours, and he required an infusion of insulin and potassium to re-establish a normal concentration.

After 16 hours his neuromuscular blockade was reversed and he was found have regained awareness during insertion of the catheter and to have full recall from that time. He was not, however, distressed and tolerated intubation for another 36 hours until extubation was considered to be safe (due to his slowly resolving oedema).

He recovered completely from immersion in cyanide and the effects of his specific antidote, dicobalt edetate.

Comment

Subsequent review showed that gross oedema after treatment with dicobalt edetate is not unusual,[1] [2] and it has been recommended that dicobalt edetate be used only in severe cases when patients do not respond to repeated doses of thiosulphate and nitrites. When dicobalt edetate is being used its potentially dangerous effect should be borne in mind and facilities for intubation and resuscitation should be immediately available.

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Is cardiac ultrasound mandatory in patients with transient ischaemic attacks?

A cerebral embolus is a much dreaded complication of cardiac disease as it often occurs unexpectedly in patients whose symptoms are few and whose prognosis is otherwise good.[1] Emboli from within the heart are well recognised as a frequent cause of stroke and there is growing evidence that occult structural cardiac abnormalities are a main cause of cerebral and retinal transient ischaemic attacks.[2-4] This has led to the routine use of echocardiography in all such patients. Echocardiography is a non-invasive technique but requires considerable investment in equipment and needs skilled operation and interpretation of results, neither of which may be readily available.

We have examined the diagnostic yield of echocardiography and tried to establish whether this investigation provides new information of importance to the management of these patients.

Patients, methods, and results

Two hundred consecutive patients who had been investigated for cerebral or retinal transient ischaemic attacks (focal neurological episodes lasting less than 24 hours) at the National Heart Hospital non-invasive laboratory between 1982 and 1985 form the basis of this report. Cases were referred from within the hospital (n=78) and from other neurological centres because of symptoms. A clinical history and examination, 12 lead electrocardiogram, and chest radiograph were obtained in each patient. None had known evidence of cerebrovascular disease.

M mode and cross sectional echocardiograms were recorded on Hewlett-Packard phased array apparatus (model 77020A) with a 3·5 or 5·0 mHz transducer. Complete echocardiographic studies using parasternal, subxiphoid, suprasternal, and apical views were routinely obtained and recorded on 12·7 mm video tape for later analysis.

In 106 cases abnormalities were found on physical examination or in the chest radiograph or electrocardiogram or both (group 1) which led to a diagnosis that was confirmed by echocardiography in valvular cases (table). In no case was mitral valve prolapse or valvular disease diagnosed by echocardiography in the absence of the appropriate physical signs elicited by the referring physician. Coronary artery disease was found in 23 patients (history and electrocardiogram), and though two of these had left ventricular thrombus and seven aneurysms, 16 had a normal echocardiogram. The other causes of transient ischaemic attacks included cardiomyopathy in six patients, myxoma in one, endocarditis in six, and valve replacement in 17. One patient in this group had a suspected aortic valve mass as the cause of transient ischaemic attacks; at exploratory thoracotomy, however, no mass was found.

Distribution of abnormal and normal cross sectional echocardiograms from 106 patients with clinical signs suggestive of cardiac source of emboli (group 1) and 94 patients with no clinical signs suggestive of cardiac source of emboli (group 2)

	Abnormal	Normal	Total
Group 1	79*	27	106
Group 2	1	93	94

*Includes one false positive result (see text).

Hypertension was present in 20 patients, nine of whom showed no evidence of left ventricular hypertrophy. The remaining 94 patients showed nothing abnormal on physical examination or in the electrocardiogram or chest radiograph (group 2). In 93 cases the echocardiogram confirmed the absence of structural cardiac disease, but in one the echocardiogram showed a pedunculated mitral valve papilloma, which was confirmed pathologically (table).

Comment

Intracardiac masses and valvular disease are a common and potentially the most easily prevented cause of transient ischaemic attacks and stroke.[2-4] In patients with established valvular disease or valve replacement (with or without infective endocarditis) we found that there was no additional evidence to be gained from echocardiography and that treatment must rest on clinical grounds. When hypertension was the only abnormality the echocardiogram was unhelpful. In patients with coronary artery disease the presence of a left ventricular aneurysm or intracavity thrombus may possibly be the cause of emboli, but in our series this was not an unexpected finding as each patient had previously suffered a myocardial infarction and had cardiomegaly. This was an uncommon problem, however, as these patients represented 23 of 742 with coronary artery disease (without transient ischaemic attacks) studied during the period, of whom 188 had evidence of left ventricular aneurysm or thrombus; this group of patients requires further study. In patients without clinical evidence of cardiac disease the use of echocardiography presents a clinical dilemma. Presumably all patients under 50 with unexplained transient ischaemic attacks should undergo cardiac ultrasound. The diagnostic yield of such investigations is very low (0·5%), however, and, given an estimated unit cost of £200, each unexpected diagnosis costs £40 000, which needs to be weighed against the costs of a completed stroke in a young patient.