

This plus the immediacy of the reaction suggest that his death was unlikely to have been due to toxicity.

Nebulised lignocaine may cause narrowing of the airways in patients with asthma,² but this is rarely of clinical importance especially as nebulised bronchodilators are given before fiberoptic bronchoscopy. Hypersensitivity to lignocaine is uncommon. Urticarial reactions may occur,³ and deaths due to anaphylaxis after small doses of injected local anaesthetic have been reported.^{4,5} We believe that this is the first reported death due to topical lignocaine during fiberoptic bronchoscopy. This procedure, however, remains safe with few complications.

- 1 Credle W, Smidy J, Elliott RC. Complications of fiberoptic bronchoscopy. *Am Rev Respir Dis* 1974;109:67-72.
- 2 Weiss EB, Patwardhan AV. The response to lidocaine in bronchial asthma. *Chest* 1977;72:429-38.
- 3 Waldman HB, Binkley C. Lignocaine hypersensitivity: report of a case. *J Am Dent Assoc* 1967;74:747-9.
- 4 Morisset LM. Fatal anaphylactic reaction to lidocaine. *Armed Forces Medical Journal* 1957;8:740-4.
- 5 Kirkler DM. Allergy to lignocaine. *Lancet* 1962;i:159.

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Minimising bruising in the antecubital fossa after venepuncture

Because of the large number of bruises seen after venepuncture of the antecubital fossa in patients presenting for anaesthesia we investigated the phenomenon.

Methods and results

We recruited 20 volunteers (eight men and 12 women) aged 20-47 with blood pressures of less than 150/95 mm Hg. Each had 5 ml of blood taken from both antecubital fossas by means of a green, 21 gauge Monoject needle and a tourniquet inflated to exactly half the diastolic blood pressure to promote venous congestion. After deflation of the tourniquet haemostasis was promoted by one of two methods based on our observations of current ward practice. In the first method the subjects pressed the contralateral thumb on to a cotton wool ball over the site of venepuncture, keeping the elbow extended. In the second method the subject flexed the elbow, keeping the cotton wool ball over the puncture site. Both methods were used for two minutes, and the site was then inspected for 15 seconds. If bleeding had not stopped the method was repeated for one minute with the same inspection procedure; this was repeated until there was no visible bleeding. An adhesive dressing was then applied.

Subjects were randomised for which arm was to be flexed or extended and thus served as their own controls. All venepunctures were performed by the same investigator. A criterion of the study was that subjects with "difficult veins" and more than one clean puncture of the vein were excluded, as were those who had recently taken aspirin; in practice there were no exclusions. After 48 hours a second investigator, who was unaware of which method of haemostasis had been used, examined the venepuncture sites. Any visible bruising was noted and the area measured with tracing paper and a planimeter; the subject was asked about any symptoms associated with the bruising.

Flexing of the elbow after venepuncture produced visible bruising in 15 subjects compared with only six when the arm was held straight ($p < 0.05$ by McNemar's test for paired data). In addition, the area of bruising was greater in the elbows that were flexed (median 0.55 cm² and maximum 4.88 cm² compared

Bruising 48 hours after venepuncture using two different methods to promote haemostasis

| | Total | No bruised | Median (range) area of bruise (cm ²) |
|-----------------|-------|------------|--|
| Elbow flexed: | | | |
| Left arm | 10 | 8 | 0.56 (0.3-6.2) |
| Right arm | 10 | 7 | 0.39 (0.4-8.8) |
| Elbow straight: | | | |
| Left arm | 10 | 3 | 0 (0.4-8.4) |
| Right arm | 10 | 3 | 0 (0.1-8.4) |

with median 0 cm² and maximum 4.84 cm² in the elbows that were extended; $p < 0.01$ by Wilcoxon's test for paired differences).

Thirty sites showed no visible bleeding after two minutes. One venepuncture site bled for over five minutes, but all the rest had stopped bleeding by 3 minutes 15 seconds. The time for bleeding to stop was unrelated to the size of the subsequent bruise ($p = 0.1$, Spearman's rank correlation coefficient).

Comment

We conclude that the common practice of flexing the elbow after venepuncture is not an efficient way of preventing bruising in the antecubital fossa.

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Huntington's disease: deterioration in clinical state during treatment with angiotensin converting enzyme inhibitor

We report on a woman with Huntington's disease whose condition deteriorated dramatically during treatment with the angiotensin converting enzyme inhibitor captopril and improved on withdrawal of the drug.

Case report

A 47 year old woman who had been diagnosed eight years earlier as suffering from Huntington's disease was admitted to hospital for control of newly diagnosed hypertension (210/120 mm Hg). She had previously been managed at home with haloperidol 1.5 mg thrice daily. On examination she walked without help and had typical features of Huntington's disease, with diffuse involuntary choreiform movements, facial apraxia, and inability to protrude her tongue for longer than five seconds. Her higher mental functions were impaired, although she could communicate and follow simple commands. She was otherwise medically well and had no clinical features of end organ damage due to hypertension.

Haloperidol was continued and captopril 18.75 mg daily in divided doses started. Because her blood pressure was unresponsive the dose was increased and over five days she received a total of 468.75 mg. Her clinical state then deteriorated and she became bedbound and totally unable to communicate and showed generalised rigidity and incontinence. She did not recognise her family and required nasogastric feeding. At no stage had she had any hypotensive episode, metabolic derangement, or fever. Her blood pressure remained raised throughout, with systolic pressure varying between 220 and 180 mm Hg and diastolic pressure varying between 150 and 120 mm Hg. Extensive investigations including lumbar puncture, thyroid function tests, and computed tomography of the brain showed no cause for her altered neurological state. Captopril was stopped and her blood pressure eventually controlled with a combination of nifedipine, atenolol, and a thiazide diuretic. Her neurological state gradually improved, and five days after the captopril was stopped she had recovered her previous functional state. Her blood pressure was controlled, and she was discharged home.

Comment

Altered amounts of various neurotransmitters have been shown in the striatonigral system of patients with Huntington's disease,¹ although the underlying biochemical defect that causes the disease is unknown. In a necropsy study Arregui *et al* showed that the corpus striatum and substantia nigra of patients with Huntington's disease had diminished quantities of angiotensin converting enzyme.² This enzyme has been localised to the striatonigral pathway of rat brains in autoradiographic experiments with tritiated captopril.³

This patient's pronounced deterioration during treatment with captopril for hypertension and her subsequent recovery after withdrawal of the drug suggest that inhibition of angiotensin converting enzyme was the cause of her altered neurological state. This observation may have important