Sensitivity and specificity of 256 Hz (●) and 512 Hz (○) tuning forks in detecting differences in air and bone conduction of various magnitudes

mask the other ear. The figure compares the sensitivity and specificity of a 256 Hz fork with those of a 512 Hz fork in detecting differences in air and bone conduction (air-bone gaps) by the occurrence of a Rinne negative response with the loudness comparison method. The sensitivity of the 256 Hz fork was significantly superior ($p < 0.05$). Neither the sensitivity nor specificity of either fork was increased by excluding equivocal responses. The sensitivity and specificity were not improved by combining the results of the 256 Hz and 512 Hz forks. The threshold method was inferior to the loudness comparison method in all aspects.

Comment

We concluded that the Rinne test should be carried out with a 256 Hz fork using the loudness comparison method. The figure can be of guidance as once the required magnitude of air-bone gap is decided, the

doctor can work out what percentage of patients will give a false negative or a false positive response. Few doctors would consider detecting an air-bone gap of less than 15 decibels necessary, but gaps of 20 decibels or more would be considered important as patients with such conductive impairments may benefit from an operation. Unfortunately a 256 Hz fork will detect only 69% of such gaps. If a criterion of a 25 decibel gap is set, however, 87% will give a Rinne negative (true conductive deafness) response with the 256 Hz fork. The most encouraging aspect is the test's high specificity. The chance of getting a Rinne negative response in patients without a conductive component to their impairment is less than 10%.

The Rinne test is unnecessary when there is otoscopic evidence of middle ear disease as a conductive impairment can then be assumed and pure tone audiometry will be needed to quantify it. If a patient with impaired hearing and an otoscopically normal tympanic membrane shows a Rinne negative response there is an extremely high possibility that there will be a conduction component to the impairment because of the test's high specificity. If the pure tone audiogram does not indicate a conductive defect then the position should be reviewed. On the other hand, because of the test's fairly low sensitivity in detecting surgically important defects in conduction, a Rinne positive response in these patients should be interpreted with caution.

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Chronic urticaria with angio-oedema controlled by warfarin

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We report a case of severe recurrent angio-oedema that responded to warfarin. This was a chance observation and may have implications for the pathogenesis and treatment of chronic urticaria.

Case report

The patient, a 42 year old man, presented in March 1977 with weekly episodes of severe angio-oedema, which were unresponsive to chlorpheniramine and trimeprazine prescribed by his general practitioner. He also had essential hypertension, familial combined hyperlipidaemia, and ischaemic heart disease, having suffered his first myocardial infarction in 1975.

Treatment with H_1 receptor blocking antihistamines was continued for 18 months, during which time he suffered episodes of urticaria and angio-oedema virtually daily, at times with severe facial swelling necessitating admission to hospital. He was subsequently treated with the H_2 antagonist cimetidine combined with H_1 antagonists, subcutaneous adrenaline, danazol, and systemic steroids. Only the steroids had any effect, and the urticaria eventually became stable when he took prednisolone 12.5 mg on alternate days. Numerous attempts to reduce this dose failed.

In February 1981 he had a further myocardial

infarction, which was complicated by multiple pulmonary emboli. He was treated with intravenous heparin, prednisolone 40 mg to control his angio-oedema, and subsequently oral warfarin. He remained completely free from urticaria throughout this admission and was discharged taking warfarin, prednisolone 12.5 mg on alternate days, cimetidine, and brompheniramine.

The dose of steroid was gradually reduced to 2 mg on alternate days, and the antihistamines were withdrawn with no deterioration. In May that year warfarin was stopped and his angio-oedema returned. The warfarin was restarted and his angio-oedema again came under control. He subsequently reported that he had repeated this procedure with the same result. He continued to take warfarin alone for his urticaria until July 1983, when he was admitted to hospital for further investigation and, without being informed or his consent being obtained, was given placebo warfarin tablets. One week later he developed severe generalised angio-oedema. Warfarin was restarted, and he subsequently had only occasional mild episodes of urticaria without angio-oedema. These episodes affected small areas of the arms, legs, and trunk; were transient, lasting less than two hours; and were usually associated with a fall in the international normalised ratio to below 2.5 (full anticoagulation (international normalised ratio ≥ 2.5) was required for optimal benefit). He had two further small myocardial infarctions.

The following measurements were normal: routine haematological and biochemical variables; concentrations of Clq esterase inhibitor, factor XII, antinuclear factor, antithrombin III (by functional and immunological assay), and protein C (corrected for the effect of warfarin); fibrinolysis induced by factor XII; and results of challenges with oral aspirin, tartrazine, benzoic acid, and yeast.

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Comment

A previous report of two similar cases of urticaria controlled by warfarin did not include a placebo challenge.¹ Our case differed from those cases in that we had to keep the prothrombin time within the therapeutic range.

The mechanism of this response to warfarin is obscure, but there may be a link between urticaria and the coagulation system.^{2,5} Ryan proposed that plasmin may contribute to urticaria through activating the complement cascade, activating kallikrein, and producing degradation products of fibrin which also promote inflammation.³ There are also several reports of chronic urticaria and angio-oedema responding to the plasmin inhibitors tranexamic acid^{3,4} and aprotinin.⁵ Warfarin could affect plasmin activity in two ways: firstly, by reducing the formation of fibrin

and, secondly, by reducing protein C concentrations. Reduced formation of fibrin may also be important in itself as fibrin may perpetuate inflammation in chronic urticaria.²

We suggest that further investigation of the role of the coagulation system and the use of warfarin in chronic urticaria would be of value.

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Anaesthetic complications of a weight reducing regimen

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Hypokalaemia during anaesthesia may cause cardiovascular instability and arrhythmias and adversely affect muscle activity. I report on a 16 year old girl who required emergency appendectomy but was found to be hypokalaemic as a result of a weight reducing regimen that included thiazide diuretics.

Case report

A 16 year old girl was admitted with acute appendicitis. She did not have a history of diarrhoea or vomiting. General questioning elicited that eight months before admission, and without her general practitioner's knowledge she had started taking a strict reducing diet and treatment (phentermine 15 mg daily and an unlabelled white tablet for "water") prescribed by a private clinic. Her weight had decreased from 69 kg to 53 kg in two months but returned to 69 kg after school examinations and parties caused a break in the regimen. Two weeks before admission she had restarted the regimen, and on admission her weight was 59 kg.

Routine blood investigations showed hypokalaemia (serum potassium concentration 2.9 mmol/l; normal range 3.5-5.0), and operation was deferred owing to the possibility that the anaesthetic might precipitate a dangerous cardiac arrhythmia. To improve the serum potassium concentration in the short term 27 mmol potassium chloride was infused intravenously over 90 minutes under electrocardiographic monitoring, and this raised her serum potassium concentration to 3.5 mmol/l. A further 13.5 mmol was given before anaesthesia, and 5 mmol potassium in one litre of compound sodium lactate intravenous solution was given during the operation.

Anaesthesia was induced with thiopentone sodium; maintained with nitrous oxide, oxygen, and isoflurane; and supplemented with fentanyl. Muscle relaxation was by suxamethonium chloride and vecuronium. Anaesthesia was uneventful, no cardiac arrhythmias being recorded during continuous monitoring.

The serum potassium concentration two hours after operation was 3.2 mmol/l. A further 27 mmol potassium chloride was infused over eight hours, after which the serum concentration was 3.1 mmol/l. Intravenous supplements were continued until oral potassium salts could be started. Before discharge a

serum potassium concentration of 4.2 mmol/l was maintained without supplementation, indicating that most of the total potassium in the body had been restored.

Comment

Patients with hypokalaemia may require an increased dose of depolarising muscle relaxant during anaesthesia,¹ while the action of non-depolarising relaxants is enhanced. Reversal with neostigmine is less predictable,^{1,2} and the risk of inadequate reversal or recurarisation is compounded in the out of hours emergencies. Intermittent positive pressure ventilation during anaesthesia increases intrathoracic pressure and causes a drop in blood pressure, which normally recovers with the resumption of spontaneous breathing. In hypokalaemic patients, however, this return to normal may not occur. The patients are also more likely to develop and less able to deal with postoperative hypotension.³

In hypokalaemia the myocardium is thought to become more susceptible to arrhythmias owing to increases in automaticity, conductivity, and excitability. It is unclear at what degree of hypokalaemia this becomes important, and the only prospective study in humans shows no evidence of chronic hypokalaemia predisposing to arrhythmias during operation.⁴ Hypokalaemia may, however, increase the mortality associated with a serious arrhythmia.⁵

This patient's own doctor was not informed of her self referral to an obesity clinic or of her drug treatment, and so he was unable to supply this information to the hospital. No blood tests had been done by the clinic. Drugs were dispensed unlabelled, so possible drug interactions with anaesthetic agents and with antibiotics could only be guessed.

Diuretics cause hypokalaemia and should not be dispensed without regular monitoring of serum potassium concentrations. Communication between members of the medical profession is important, particularly on the part of doctors who provide treatment in clinics to which patients refer themselves.

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