SHORT REPORTS

Quetelet index in diagnosis of anorexia nervosa

The criteria for diagnosing anorexia nervosa vary between authorities,¹⁻³ but a considerable loss of weight or a low body weight is considered an important diagnostic criterion. The body weight criterion for anorexia nervosa is stated, in the absence of any physical or psychological illness, to be a loss of body weight of at least 25% of the original body weight or the weight is less than 80% of average body weight.

The criterion of a loss of at least 25% of the original body weight may be faulty because an obese person losing this proportion of her body weight usually does not have anorexia nervosa. The criterion that the person's weight must be less than 80% of the average body weight for age, sex, and height demands recourse to special tables,⁴ which are not often available in general practice.

We believe that the Quetelet index (weight in kilograms divided by height in metres squared) may be a more appropriate measure of body weight. The index has been used to define the boundaries of various grades of obesity and we have used it to define grades of low body weight.

Patients, methods, and results

We calculated the Quetelet index for a series of women diagnosed as having an eating disorder for at least three years. Twenty three patients had anorexia nervosa (diagnosed using Russell's criteria³); six had anorexia nervosa with bulimia, and 16 had bulimia diagnosed using the criteria of the American Psychiatric Association.¹ The Quetelet index used was that obtained for each patient when at her lowest recorded weight. We also calculated the Quetelet index of 26 student ballet dancers at a national ballet school, again taking the lowest weight reached as the criterion. Ballet dancers fear becoming fat, exercise rigorously, are preoccupied with their weight, and usually have amenorrhoea. Although in these features they resemble patients with anorexia nervosa, most do not develop the illness. The values of the index are as follows: < 15 emaciation, 15-18.9 underweight, 19-24.9 normal weight, 25-29.9 overweight, 30-39.9 obese, ≥ 40 severely (morbidly) obese.

The Quetelet indices of patients with anorexia nervosa, anorexia nervosa with bulimia, and bulimia are shown in the figure. All patients whose

The lowest body weights obtained by patients diagnosed as having eating disorder for three or more years.

Quetelet index was less than 15 had maintained a low body weight for at least 12 months or, if referred in hospital, had returned to a low body weight after discharge. These patients had been diagnosed as having anorexia nervosa; six also fulfilled the criteria for bulimia. All the women diagnosed as having anorexia nervosa and bulimia reached their low body weight transitorily. The lowest body weight of patients diagnosed as having bulimia was also only attained transitorily, and their Quetelet index was above 15. The Quetelet index of each ballet dancer was below 19, which indicates

that by normal standards the women were underweight. Three of the dancers had reached a Quetelet index of under 15. One had been diagnosed as having anorexia nervosa, but the other two increased their weight after counselling and anorexia nervosa was not diagnosed.

Comment

We suggest that the Quetelet index may be as appropriate a measure of body weight in the underweight and emaciated as it is in the overweight and obese. A Quetelet index of less than 15 in the absence of any physical or psychiatric disorder is strongly suggestive of anorexia nervosa. A further advantage of the index is that it is independent of sex, age, or changing fashions in eating behaviour, and possibly culture.

- 1 American Psychiatric Association. *Diagnostic and statistical manual of mental disorders*. 3rd ed. Washington: American Psychiatric Association, 1980.
- 2 Feighner JP, Robins E, Gruze SB, Woodruff RA, Winokur LT, Munoz R. Diagnostic criteria for use in psychiatric research. *Arch Gen Psychiatry* 1972;26:57-63.
- 3 Russell GFM. Anorexia nervosa: its identity as an illness and its treatment. In: Price JH, ed. *Modern trends in psychological medicine*. Vol 2. London: Butterworths, 1970:131-64.
- 4 Society of Actuaries. *Build and blood pressure study*. Vol 1. Chicago: Society of Actuaries, 1959:16.
- 5 Garrow JS. *Treat obesity seriously*. Edinburgh: Churchill Livingstone, 1981.

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Sodium bicarbonate and hyperventilation in treating an infant with severe overdose of tricyclic antidepressant

There is no specific antidote for severe poisoning with tricyclic antidepressants. Treatment is supportive, and death is usually caused by cardiac depression or arrhythmias.¹ I describe here the use of intravenous sodium bicarbonate and hyperventilation to reverse the arrhythmia in a child who had taken a lethal dose of dothiepin.

Case report

An 11 month old girl weighing 9.7 kg had been given about 13 dothiepin 75 mg tablets (100 mg/kg) by her sister. She had become drowsy, her limbs started to twitch, and she had a generalised convulsion. On admission to hospital one and a half hours later she was comatose and convulsing. Her pulse was 160 beats/min, blood pressure 80/50 mm Hg, respirations regular, and pupils fixed and dilated. Electrocardiography showed sinus tachycardia. Her convulsions were controlled with intravenous diazepam 10 mg and intramuscular paraldehyde 4 ml. She was electively intubated and her stomach emptied.

An hour after admission she suddenly became bradycardic (pulse 50 beats/min, blood pressure unrecordable). The electrocardiogram showed wide QRS complexes (see figure (a)). Cardiac massage and assisted ventilation were started. She did not respond to intravenous atropine 0.3 mg. She was given 5 mmol intravenous sodium bicarbonate and 50 ml plasma protein fraction. She required intravenous diazepam 15 mg to control a further convulsion. Blood gas analysis showed hypoxia with metabolic and respiratory acidosis (pH 7.14, carbon dioxide partial pressure 5.7 kPa (42.8 mm Hg), oxygen partial pressure 5.8 kPa (43.5 mm Hg), base excess -14.5 mmol(mEq)/l, and bicarbonate 13 mmol(mEq)/l).

Intravenous sodium bicarbonate (10 mmol immediately, 20 mmol over the following hour) was given and hyperventilation started. A further dose

Electrocardiographs after overdose of tricyclic antidepressants: (a) 2½ hours after ingestion (pulse 50 beats/min); (b) 4 hours after ingestion (pulse 104 beats/min, systolic blood pressure 75 mm Hg); and (c) 5 hours after ingestion (pulse 150 beats/min, systolic blood pressure 75 mm Hg).

of atropine 0.3 mg intravenously was ineffective. She was given 10% mannitol 10 ml for any cerebral oedema and activated charcoal via the nasogastric tube. One hour later blood gas results showed correction of her acidosis (pH 7.5, carbon dioxide partial pressure 3.5 kPa (26.3 mm Hg), oxygen partial pressure 20 kPa (150 mm Hg), base excess -1.5 mmol/l, and bicarbonate 23 mmol/l). Shortly after this her pulse suddenly returned to 104 beats/min, and her blood pressure was 75 mm Hg systolic. Her electrocardiogram still showed widened QRS complexes (figure (b)), but there was a gradual improvement over the next few hours with narrowing of the QRS complexes, some ST depression, and ventricular ectopic beats (figure (c)).

Ventilation was continued to maintain her respiratory alkalosis, and her electrocardiogram was monitored. Ten hours after ingestion of the tablets she responded to pain and showed some spontaneous limb movements. She was extubated after 48 hours and made an uneventful recovery. There were no residual symptoms or signs three months later.

Comment

Our patient presented with the most serious cardiac manifestations of overdose with tricyclic antidepressants, cardiac arrhythmia and hypotension. Her plasma dothiepin concentration (2.6 mg/l) was well above the toxic concentration (1 mg/l), although symptoms of toxicity are not always related to dose.

A series of experiments with puppies and the successful treatment of nine out of 12 children with arrhythmias caused by tricyclic antidepressants have shown the beneficial effect of sodium bicarbonate in treating such arrhythmia.² Correcting the combined respiratory and metabolic acidosis increases the protein binding of the drugs and decreases the risk of arrhythmias and myocardial depression from acidosis. Deliberate artificial hyperventilation and sodium bicarbonate have reversed arrhythmias resistant to treatment with physostigmine and lignocaine.³ Unexpected late deaths after ingestion of tricyclic antidepressants have been reported, and 24 hours' continuous electrocardiographic monitoring after resolution of signs of toxicity is recommended for all patients.⁴ A review of accidental poisoning in 1974-80 showed that tricyclic antidepressants caused the deaths of 26 out of 79 children under 5. Most prescriptions (for 33 out of 57 drugs) were for the mothers.⁵

In the light of this case report, repeat arterial pH and blood gas measurements should be undertaken at least hourly in those severely poisoned, and sodium bicarbonate and hyperventilation should be considered early in management.

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¹ Thorstrand C. Cardiovascular effects of poisoning with tricyclic antidepressants. *Acta Med Scand* 1974;195:505-14.

² Brown TCK. Sodium bicarbonate treatment for tricyclic antidepressant arrhythmias in children. *Med J Aust* 1976;2:380-2.

³ Kingston M. Hyperventilation in tricyclic antidepressant poisoning. *Crit Care Med* 1979;7:550-1.

⁴ Pentel B, Sioris L. Incidence of late arrhythmias following tricyclic antidepressant overdose. *Clin Toxicol* 1981;18:543-8.

⁵ Craft AW. Circumstances surrounding deaths from accidental poisoning 1974-80. *Arch Dis Child* 1983;58:544-6.

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Active and passive smoking in married couples: results of 25 year follow up

Smoking is known to reduce a man's life span, but there are indications that in women smoking may be less harmful.¹ A further debate concerns the extent to which passive smoking in women is related to disease and premature death.^{2,3} We studied the relations between smoking and mortality in a 25 year follow up of 1007 married couples.

Subjects, methods, and results

The 1007 married couples were a subset of a cohort of 1583 men and 1508 women, civil servants and spouses of civil servants of the city of Amsterdam, who had participated in a general health examination in 1953-4 when they were aged between 40 and 65. Among the participants there were 1070 married couples; the other subjects had presented singly to the examination. The original aim of the health examination had been to study the feasibility of the early detection of chronic diseases.⁴ As part of each patient's medical history a smoking history had been obtained. This subsequently enabled us to categorise all participants according to smoking habit. The categories comprised non-current smokers (those who had stopped smoking, never smoked, or smoked less than one item a day), current cigarette smokers (subclassified according to number of cigarettes smoked; occasional pipe or cigar smoking disregarded), and pipe or cigar smokers (occasional cigarette smoking disregarded).

The survival of these subjects was followed up through the civil servants' pension offices, which held only information about total mortality (all causes of death). By 1981 the follow up was complete in 1571 men (99.2%) and 1504 women (99.7%); data were complete for 1007 married couples. Further details of the study will be published elsewhere.⁵ We analysed 15 and 25 year mortality according to smoking habit. The cut off at 15 years was chosen because about half of the observed deaths had occurred by that time. Figures for mortality were standardised for age by the direct method. All women who were active smokers were excluded from the analysis of passive smoking. Women who were not smokers were classified according to the smoking habits of their husbands.

The table shows the percentage total mortality, together with its standard error, at 15 and 25 years' follow up in husbands who were active smokers, wives who were active smokers, and wives who were passive smokers.

Mortality after 15 and 25 years' follow up according to smoking habit in 1007 married couples

		Percent (SE) mortality* after:	
Daily smoking habit†	No	15 years	25 years
<i>Husbands who smoked actively</i>			
Non-current	129	15.3 (3.1)	42.6 (4.0)
1-10 cigarettes	329	18.3 (2.0)	48.9 (2.6)
11-20 cigarettes	283	24.9 (2.6)	55.2 (2.8)
> 20 cigarettes	130	30.0 (4.0)	60.2 (3.7)
Pipe or cigar	136	24.3 (3.6)	57.7 (4.2)
<i>Wives who smoked actively</i>			
Non-current	803	10.1 (1.0)	29.1 (1.4)
1-5 cigarettes	121	8.8 (2.7)	28.2 (4.1)
> 5 cigarettes	83	5.1 (3.4)	41.1 (5.0)
<i>Wives who smoked passively</i>			
Non-current	113	11.6 (3.0)	38.1 (4.1)
1-10 cigarettes	266	8.6 (1.6)	28.5 (2.5)
11-20 cigarettes	223	9.3 (2.0)	27.8 (2.6)
> 20 cigarettes	90	12.3 (3.1)	34.6 (5.3)
Pipe or cigar	111	14.6 (3.1)	34.4 (3.6)

*Standardised for age. †In women who smoked passively daily smoking habit is daily smoking habit of husband.

Comment

Active smoking in men showed its known gradient of risk. Active smoking in women carried less risk; only after 25 years of follow up could a slight gradient in risk be discerned. Women, however, smoked considerably less than men; few women smoked more than five cigarettes a day, which was reflected in a large standard error. Other differences in the smoking habits of women, such as the pattern of inhalation, might partly account for the difference in risk.¹ Lastly, passive smoking was not associated with an increase in total mortality.

This finding does not necessarily contradict those of other studies that showed an association between passive smoking and lung cancer in women.^{2,3} Lung cancer is a rare disease in women, and even a severalfold increase in its prevalence would be unlikely to affect total