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Relation between intake and plasma concentration of vitamin C in elderly women

The plasma concentration of vitamin C is relatively low in the elderly, particularly those in long stay hospitals,¹ but it is not clear to what extent these low concentrations are due to low intake. We examined the relation between intake and plasma concentrations of vitamin C in elderly women living at home and in long stay hospitals.

Patients, methods, and results

We studied 101 elderly women: 24 were healthy and living at home (mean age 74), and 77 were long stay patients (mean age 83) in two geriatric hospitals and one psychiatric hospital. Food intake was weighed and noted for five days in the case of the elderly women in hospital and for seven days in the case of those at home. Vitamin C intake was calculated from standard food tables except in the case of vegetables and potatoes delivered to the wards, which we analysed for vitamin C. Each subject's plasma concentration of vitamin C was measured at the end of the period during which intake of food was measured.²

The figure shows the relation between plasma concentration and intake of vitamin C. The relation was sigmoidal, not linear, the curve being constructed by plotting the median concentration of vitamin C at the median point for each decile of intake. First and last deciles were split again to give greater detail. When the intake of vitamin C was above 60 mg a day the median plasma concentration was 74 $\mu\text{mol/l}$ (1.3 mg/100 ml), and all but one of the subjects had a plasma concentration above 20 $\mu\text{mol/l}$ (0.35 mg/100 ml). There was a large decrease in the median plasma concentration of vitamin C as the median intake decreased from 60 to 30 mg a day, and most patients with an intake of less than 30 mg a day had a plasma concentration of less than 20 $\mu\text{mol/l}$ (0.35 mg/100 ml).

Median plasma concentration of vitamin C at median point for each decile of daily intake of vitamin C (percentiles indicated by dotted lines on horizontal axis).

Conversion: SI to traditional units—Plasma vitamin C: 1 $\mu\text{mol/l} \approx 17.6 \mu\text{g}/100 \text{ ml}$.

Comment

The sigmoidal relation between intake and plasma concentration of vitamin C has not been reported before. Garry *et al*³ showed that the relation was not linear, but their study did not include elderly people with low vitamin C intakes. We found that as intake increased from 10 to 30 mg a day the plasma concentration of vitamin C rose only slowly. The rapid change in plasma concentration as the intake increased from 30 to 60 mg a day suggested that a pathway of metabolic utilisation was being saturated. The much slower increase with intakes above 60 mg a day may have been due to a large loss of vitamin C in the urine when the renal threshold of a plasma vitamin C concentration of 51-57 $\mu\text{mol/l}$ (0.9-1.0 mg/100 ml) was exceeded.⁴ When we gave a similar group of elderly patients 1 g vitamin C a day for two months their mean plasma concentration reached only 78 $\mu\text{mol/l}$ (1.37 mg/100 ml).⁵

Evidence shows that plasma concentrations of vitamin C should be maintained above 20 $\mu\text{mol/l}$ (0.35 mg/100 ml) to ensure against impairment of health.⁴ The results presented here suggest that this may be achieved, even in the elderly, if intake of vitamin C is kept above 60 mg a day.

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Protracted survival in patients with Down's syndrome

The life expectancy of patients with trisomy 21 remains less than that of the general population,¹ but there seems little doubt that it is steadily increasing.^{2,3} We report on an old man who suffered from Down's syndrome, coeliac disease, and oesophageal carcinoma.

Case report

A 50 year old man with Down's syndrome presented in 1962 with a three year history of diarrhoea. Investigation showed megaloblastic anaemia, and a jejunal biopsy specimen showed villous atrophy. A gluten free diet was started, with excellent results. Haemoglobin concentrations returned to normal over the following weeks and diarrhoea stopped. Coeliac disease was diagnosed.

Down's syndrome was diagnosed on the clinical features, including handprint and footprint studies, and was confirmed by chromosome analysis of 40 fibroblasts cultured from a skin biopsy specimen. Trisomy 21 occurred in 39 of them, which made a diagnosis of mosaicism unlikely.

The patient lived at home with an older sister and remained well for a further 20 years. He presented again, when his sister could no longer look after him, with a six month history of weight loss and anorexia. Investigation showed almost complete obstruction of the oesophageal lumen by a 3 cm long annular squamous cell carcinoma. He died three weeks later. Repeat chromosome analysis had confirmed trisomy 21.

Comment

At 71 our patient was slightly older than any previously reported patient with Down's syndrome. His remarkable longevity reflects both the attention he received from his sister, avoiding the hazards of institutional care, and the general improvement in the long term survival of patients with Down's syndrome over the past 20-30 years.¹⁻³ In Japan the life expectancy of a cohort with Down's syndrome born between 1966 and 1975 has been estimated to be 50 years, although in other reports the improvement in survival has been much less dramatic. It is of enormous importance in the "costing" (in human and social terms) of this important syndrome that patients such as ours are now living to 70 and beyond. Read recently claimed that the incidence of Down's syndrome is increasing and suggested that this may be related to the use of oral contraceptives,⁴ although most workers agree that the incidence of the syndrome has decreased in recent years. This decrease, however, will not be accompanied by an equal fall in prevalence, which in some areas is actually expected to rise.³ This almost certainly results from a combination of improved medical and other care for infants and children, particularly in institutions for the mentally handicapped, where patients have tended to do less well than at home, and of improved cardiac surgery, as cardiac lesions have been responsible for a large proportion of the early mortality in Down's syndrome.

An increased incidence of oesophageal carcinoma is fairly well established in patients with coeliac disease. Such carcinoma may develop many years after the symptoms of gluten sensitivity first occur, and this tendency to malignant change may not be affected by adherence to a gluten free diet.

To our knowledge oesophageal carcinoma has not been reported in Down's syndrome. The association of Down's syndrome and coeliac disease has been reported once previously.⁵

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Myopotential inhibition of unipolar pacemakers

Myopotential inhibition of pacemakers is an important cause of recurrent dizziness and syncope in patients with permanent pacemakers.¹ Muscle potentials from the adjacent pectoralis major activate the sensing amplifier of the pacemaker, causing inhibition and recycling.^{2,3} Repeated sensing and recycling lead to protracted inactivity of the pacemaker and recurrence of the patient's original symptoms. We report the clinical presentation of myopotential inhibition, useful tests for confirming the diagnosis, and the various techniques available for its management.

Patients, methods, and results

Thirteen men aged 39-87 (mean 65 (SD 14.5)) and three women aged 47-65 (mean 58 (10)) presented during 1982 with recurrent dizziness or syncope after implantation of permanent pacemakers. All pacemakers were of the unipolar ventricular demand type and were sited subcutaneously anterior to one or other pectoralis major muscle. Models included Teletronics 150L, 150B, 161, Optima, Optima MP; Cardiac Pacemaker 0521, 0523; and Vitatron C2121.⁴

Symptoms appeared one to 36 months (mean 11.4 (9.6) months after implantation. All were associated with movements of the arms that involved the pectoralis major underlying the pacemaker. These movements included washing, drying, and scratching under the opposite axilla; digging, hoeing, and raking soil; cutting a hedge with garden shears; sweeping leaves; shovelling snow; removing a pullover; getting out of a chair; lifting objects such as a pile of books, a car battery, a full kettle; washing and combing hair; peeling vegetables; turning a steering wheel; pushing and pulling a glass swing door; and hanging wallpaper. One man experienced near syncope on hugging his wife, and a university lecturer complained of dizziness when gesturing or drawing with his left hand. A traffic policeman, whose pacemaker was implanted in the right pectoral region, collapsed unconscious while removing his notebook from his left breast pocket at the scene of a traffic offence. Walking while wearing his heavy overcoat also produced dizziness.

Electrocardiograms were taken while the patients repeated those movements of the arms that were associated with symptoms or performed isometric exercises using the arm muscles on the same side of the body as the pacemaker. Characteristically, the electrocardiograms showed loss of the pacing artefact and ventricular asystole or a return to the non-paced cardiac rhythm (figure). The skeletal myopotentials responsible were usually seen

Electrocardiogram showing myopotential inhibition caused by washing under the left axilla. The muscle potentials (arrow) lead to recycling of the timing circuit, loss of the pacing artefact, extreme bradycardia, and dizziness.

on the electrocardiogram during the period when the pacemaker was inactive. Tracings of diagnostic quality were best obtained with a recorder with three channels and by attaching the limb leads to the patient's back with adhesive electrodes.

Six patients chose to try and avoid the precipitating movements. Six had their pacemakers changed to multiprogrammable units; these patients, together with two already possessing such units, had their symptoms abolished by an asynchronous pacing programme. One patient was cured by a low sensitivity programme and one by changing to a ventricular triggered unit.

Comment

We have found that myopotential inhibition occurs in up to 40% of patients dependent on pacemakers, 15% of whom are symptomatic, one half seriously enough to warrant corrective intervention. The clinical presentation and diagnostic tests should be appreciated by all those responsible for patients with pacemakers. Holter monitoring may be useful but is not routinely necessary.⁵ Failure to recognise the symptoms can lead to delay in diagnosis and requests for inappropriate and expensive investigations.

For symptomatic patients avoiding the movements responsible may be sufficient. Alternative techniques include reducing the sensitivity of the pacemaker or reprogramming the pacemaker from a ventricular demand to ventricular triggered or asynchronous mode. Replacing the unipolar system by a bipolar system that will not sense local myopotentials is a further option. Implantation of pacemakers with such multiprogrammable capability would obviate the need for further surgical intervention in patients with serious myopotential inhibition of the pacemaker.

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