SHORT REPORTS

Effects of cooking on serum glucose and insulin responses to starch

Several studies have shown that the response of glucose or insulin, or both, to various carbohydrates or carbohydrate-rich foodstuffs varies,¹⁻⁴ even though the amount of carbohydrate ingested remains constant. Certain dietary fibres reduce the response of glucose and insulin to a carbohydrate load, and it has been suggested that this may be due to altered viscosity.⁵

We studied the responses of serum glucose and insulin to both cooked and raw starch. Cooking not only increases the viscosity but also splits the starch granules, thereby increasing the availability of the starch to amylase.

Subjects, methods, and results

Eleven healthy male medical students were given, in random order and after an overnight fast, 1 g of glucose equivalent per kg body weight as either raw or cooked corn starch (0.91 g/kg body weight; Boots Co Ltd) or glucose monohydrate (1.1 g/kg body weight) in 4 ml water/kg body weight. Cooking entailed heating the raw starch and water to 68-70°C and stirring until

Mean (±SE) serum insulin and glucose concentrations after ingestion of glucose, raw starch, and cooked starch.
Conversion: SI to traditional units—Glucose: 1 mmol/l ≈ 18 mg/100 ml.

complete gel formation. The raw starch and water were ingested as a slurry³ and the glucose monohydrate as a solution. Blood samples were taken before and 15, 30, 60, and 90 minutes after ingestion of the test meal. Serum glucose concentration was estimated with a glucose oxidase method, and insulin values by a double antibody radioimmunoassay technique. Student's *t* test was used for statistical comparisons.

The responses of serum glucose to glucose monohydrate and cooked starch were closely similar, while that to raw starch was significantly less (figure). The serum insulin response was greatest with the glucose monohydrate meal, and the area under this response curve was significantly greater than that after the cooked starch meal ($p < 0.01$), which in turn was significantly greater than that after the raw starch meal ($p < 0.01$) (figure).

Comment

In any comparison of response to various dietary carbohydrates the effects of cooking the carbohydrates may be important. The similar pattern of serum glucose response to glucose monohydrate and cooked starch compared with the simultaneous significant difference in serum insulin responses again exemplifies the limited value of the response of serum glucose as a measure of the rate and extent of glucose absorption. The different nature of serum glucose response to the raw and cooked starch may theoretically have been due either to reduced availability of the raw starch to amylase or to the difference in viscosity of the two test meals. The smaller responses of serum glucose and insulin to the raw starch, however, suggest, that an alteration in viscosity was not responsible.

Serum insulin values rose less after starch, whether raw or cooked, than after an equivalent load of glucose. That the response of insulin after cooked starch was less than after glucose suggests that viscosity of the meal was playing a part, whereas the greater reduction in the response of insulin to raw starch was unlikely to have had a viscosity component. It seems that the ability to alter the response of glucose or insulin, or both, to a carbohydrate meal is not peculiar to certain types of dietary fibre but can also be brought about by the cook.

¹ Coulston RD, Greenfield M, Kraemer F, Tobey T, Reaven G. Effect of source of dietary carbohydrate on plasma glucose and insulin responses to test meals in normal subjects. *Am J Clin Nutr* 1980;**33**:1279-82.

² O'Dea K, Nestel PJ, Antonoff L. Physical factors influencing postprandial glucose and insulin responses to starch. *Am J Clin Nutr* 1980;**33**:760-5.

³ Vaaler S, Hanssen KF, Aagenoes O. Plasma glucose and insulin responses to orally administered carbohydrate-rich foodstuffs. *Nutr Metab* 1980;**24**:168-75.

⁴ Jenkins SJA, Wolever TMS, Taylor RH, Barker HM, Fielden H. Exceptionally low blood glucose response to dried beans: comparison with other carbohydrate foods. *Br Med J* 1980;**281**:578-81.

⁵ Jenkins DJA, Wolever TMS, Leeds AR, et al. Dietary fibres, fibre analogues and glucose-tolerance: importance of viscosity. *Br Med J* 1978;**ii**:1392-4.

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Compression of oesophagus by tortuous dilated aorta

Although compression of the oesophagus by aortic aneurysm or great-vessel anomalies—namely, dysphagia lusoria¹—is well recognised, compression by a dilated and tortuous aorta has been reported in only 15 patients,²⁻⁵ most being elderly women. We report six cases seen in this hospital and suggest that the condition may be more common than has been supposed. We also report kinking obstruction of the elongated oesophagus proximal to the compression, which has not been described.

Case reports

Case 1—An 89-year-old woman admitted in left ventricular failure also complained of longstanding anorexia, vomiting, and weight loss of 6 kg. Pronounced kyphoscoliosis and left ventricular hypertrophy were noted. She was normotensive. Chest radiography showed cardiomegaly with left ventricular enlargement, and an unfolded aorta, basal lung changes consistent with aspiration, and a mediastinal fluid level. The descending aorta had a reversed S configuration.³ Barium swallow showed oesophageal elongation and secondary kinking obstruction (figure). Even after 20 minutes only a small amount of barium had entered the stomach. The reason for the oesophageal compression was apparent but had not been recognised when barium swallow had been performed elsewhere five years previously.

Case 2—Another woman, aged 73, had longstanding severe osteoporosis. She also complained of dysphagia and dyspepsia. Radiological investigation showed multiple compressed vertebrae, appreciable kyphosis, and profound osteoporosis and also noticeable hold-up of barium in the lower oesophagus owing to a tortuous aorta with the reversed C configuration.³ There was mild left ventricular enlargement. She was normotensive.

The remaining four patients all complained of dysphagia, and similar hold-up of barium by a tortuous aorta was noted on radiological investigation. In addition three of the four showed kinking obstruction similar to that seen in case 1; in one patient this was more severe than the original compressive obstruction. Three of the four patients had evidence of left ventricular enlargement. Two were hypertensive. One patient died from inanition owing to complete oesophageal obstruction. Oesophageal compression by the aorta was confirmed as the only abnormality post mortem.

X-ray film obtained on barium swallow (case 1) showing reversed S configuration of aorta compressing mid and distal oesophagus and also secondary kinking obstruction of upper oesophagus.

Comment

Twenty-one patients, including our own, have now been described with this condition. Most are elderly women with a long history of dysphagia or anorexia and often weight loss.

The aortic unfolding that causes oesophageal compression is seen on barium studies to fall into two clear categories—that is, the reversed S and reversed C configurations.³ The reversed S aorta is likely to compress the oesophagus in its mid and lower thirds and the reversed C only in the lower third. Four of our patients showed the reversed C and two the reversed S aorta.

Because of the oesophageal compression oesophageal dilatation and then elongation occur. The elongation introduces a kinking obstruction which usually occurs just below the aortic arch and may be more severe than the original compressive obstruction.

Why this condition predominates in women and whether aortic unfolding is more common in women are not known; while kyphosis (which may contribute to the condition) resulting from osteoporosis is more common in women, it was not present in all of our patients. Hanna and Derrick suggested that the oesophageal obstruction is due to compression between the unfolded aorta and an enlarged left ventricle⁴; left ventricular enlargement may well aggravate the condition but is clearly not an essential feature since it was absent in one of our patients.

Prognosis and treatment of the condition vary. One of our patients was subsequently untroubled by symptoms, one was satisfactorily treated with and one was trying a semi-fluid diet, and another died of inanition. The fate of the other two was not known. The course in previously reported patients has also varied.²⁻⁵ Patients have been asymptomatic, had slight or intermittent dysphagia, or shown severe oesophageal obstruction often with accompanying oesophagitis. Treatment for the condition varies with severity and includes a semi-fluid diet and oesophagoscopy or bougienage, or both. In the most severe cases surgical repositioning of the oesophagus has been reported to relieve the compression and symptoms completely.⁵

Recognising this condition is important as it is a remediable cause of dysphagia, anorexia, and weight loss and, though usually more likely to cause mild rather than severe symptoms,⁵ is nevertheless potentially fatal, causing aspiration pneumonia and inanition. We suggest that it is not uncommon in elderly women and if it was recognised more widely than at present many of the ensuing symptoms might be avoided by using a fluid or semi-fluid diet and resorting to bougienage or surgical relief of the compression in those unhelped by diet.⁵

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⁵ McMillan IKR, Hyde I. Compression of the oesophagus by the aorta. *Thorax* 1969;**24**:32-8.

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Multisystem disorder after exposure to paint stripper (Nitromors)

Nitromors is a household and industrial paint stripper whose active ingredient is methylene chloride (CH_2Cl_2). It is reputedly harmless in ordinary use, but methylene chloride is a halogenated hydrocarbon and these compounds are a recognised cause of hepatic and renal damage.¹ Inhalation of methylene chloride vapour may cause pulmonary oedema² and depression of the central nervous system.³ We report a serious multisystem disorder that followed exposure to Nitromors and was complicated by the development of temporary diabetes mellitus.

Case history

A previously fit 25-year-old accountant used Nitromors in a confined space for three to four hours, taking the recommended precautions to avoid skin contact. He suffered no immediate side effects except for mild conjunctival irritation, and returned to work the next day. Forty-eight hours after exposure he felt generally unwell, with swollen and stiff ankles, knee, and wrist joints, and over the next 24 hours he developed a pink, profuse, non-itchy rash. During the next eight days he noticed increasing difficulty with simple intellectual tasks and an inability to concentrate and his work deteriorated; he developed swelling of the face and shortness of breath on minimal exertion. Ten days after exposure he became increasingly thirsty and complained of nocturia, polyuria, and weight loss of 2.7 kg.

On admission he had stiffness of the fingers, wrists, and ankles and a profuse pink nodular rash, parafollicular in distribution, limited to the upper half of the body. Although he was conscious and fully orientated, his concentration was poor and mental arithmetic impaired; his long-term memory was intact but immediate recall poor. There was pitting oedema of both ankles. Jugular venous pressure was raised by 2 cm H_2O , pulse was 98 beats/min and blood pressure 160/110 mm Hg, and a third heart sound was present. Urinary examination showed 2% glycosuria and a few red blood cells per high-power field on microscopy. Laboratory results included normal urea and electrolyte concentrations, a random blood glucose concentration of 12.1 mmol/l (218 mg/100 ml), bilirubin 27 nmol/l (1.6 $\mu\text{g}/100$ ml), alkaline phosphatase 170 King Armstrong units/l, alanine aminotransferase 47 IU/l, and normal serum protein, creatinine, and calcium concentrations and creatinine clearance. Electrocardiography showed widespread T-wave inversion, and a chest x-ray film showed cardiomegaly with upper-lobe blood-vessel diversion. Virological studies were negative and coxsackie B antibodies not detected.