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Changing iodine intake and the effect on thyroid disease

The gross iodine deficiency that occurs in some areas leads to cretinism in its various forms, goitre, and hypothyroidism. Another manifestation of iodine deficiency is a general reduction in the intelligence quotient of whole populations. In areas of moderate iodine deficiency, which include much of continental Europe where iodine supplementation is not practised, a different group of disorders is seen. Goitre, initially diffuse and then multinodular, is common, affecting 15% of the population of the Federal Republic of Germany.[1] Autonomous thyroid nodules, toxic nodular goitre, and iodine induced hyperthyroidism are all commoner than in areas with more iodine. More subtle manifestations of minor iodine deficiency include transient neonatal hypothyroidism, especially in the premature,[2] and an alteration in the causes of hyperthyroidism.[3]

Little information is available on the current iodine state in the United Kingdom.[1] Local differences in the prevalence of non-toxic goitre have been described between Sheffield and Ormiston[4] and in East Herrington, County Durham.[5] Appreciable differences have also been reported between different towns in the prevalence of hyperthyroidism.[6] A further study showed a reduced prevalence of thyrotrophin receptor antibodies in older people with hyperthyroidism, who presumably suffered from toxic nodular goitres.[7] This suggested that iodine deficiency had been common in the older age group.

Direct evidence of the prevalence of transient neonatal hypothyroidism in the United Kingdom is not available, but Hulse *et al* found hypothyroidism to be permanent in 31 of 32 infants diagnosed as having neonatal hypothyroidism when treatment was temporarily withdrawn at 1 year.[8] Indirect evidence for an increase in iodine intake in the United Kingdom is seen in the fall in four hour thyroidal uptake measurements of iodine-123 or iodine-131 in hyperthyroid patients seen in both Newcastle and Cardiff over the past two decades. Uptakes in Cardiff have remained almost constant over the past five years. Indirect assessments from dietary questionnaires suggested an average daily iodine intake of about 250 μg,[9] but this method tends to overestimate iodine intake because of losses in preparation; and urinary iodine values of about 100 μg daily in hyperthyroid patients in Cardiff have been reported.[10]

The United Kingdom may be developing into an area of moderate to high iodine intake, not through deliberate iodine supplementation but indirectly through iodine in milk and dairy products. The likely consequences over the next decade are that iodine induced hyperthyroidism may become more common, particularly in those with pre-existing nodular goitres, and that Graves' disease may become more difficult to control with antithyroid drugs. Postoperative hypothyroidism may become rarer and relapse of hyperthyroidism commoner. The dose of radioiodine required to induce euthyroidism and hypothyroidism may increase, and the pattern of thyroid cancer and of hyperthyroidism will alter.

As well as being a substrate for the biosynthesis of thyroid hormone iodine appears to be an immune stimulant,[11] and greater exposure to iodine may increase autoimmune thyroid disease in a population.[12] This finding requires confirmation. Iodine may also affect the response of the thyroid to antithyroid drugs—indirectly by increasing the thyroidal stores of preformed hormone and directly by altering the thyroid's metabolism of carbimazole.[13] Studies are required to assess the effect of iodine state on the response to antithyroid drugs. Patients apparently resistant to carbimazole are a rare but important group, and, although poor compliance is often blamed, evidence for iodine contamination should also be sought.

Few centres in the United Kingdom are able to measure iodine excretion or thyroidal iodine content, and further studies are required of the iodine state of the population and its effect on thyroid physiology and disease.

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Solvent abuse and the heart

The deliberate inhalation of volatile substances, usually halogenated or unsubstituted hydrocarbons, to obtain a "high" is common among teenage boys and causes important morbidity and mortality[1 2]—80 deaths in Britain in 1983.[3] Solvent abuse may cause both acute and chronic cardiotoxicity, and this issue carries three reports of serious cardiac problems associated with exposure to volatile hydrocarbons (pp 727 and 739).

In 1970 Bass reported an epidemic of sudden deaths associated with solvent sniffing in the United States.[4] He proposed that these deaths were caused by an arrhythmia and suggested that volatile hydrocarbons might sensitise the heart to the arrhythmogenic effects of endogenous catecholamines. There is now much evidence to support this hypothesis. Firstly, solvent abuse has been followed by documented ventricular fibrillation.[5-7] Secondly, sudden death related to solvent abuse has occurred often in circumstances associated with intense cardiac sympathetic stimulation—physical exertion, particularly running,[4 8] and various forms of autoerotic behaviour.[3 9] The case of myocardial infarction, possibly caused by coronary artery spasm, and ventricular fibrillation described by Cunningham *et al* (p 739) fits the same pattern. Thirdly, well controlled studies in dogs have shown that adrenaline given after many different inhaled volatile hydrocarbons may produce serious ventricular arrhythmias including ventricular fibrillation.[10 11] Furthermore, ventricular tachycardia has been seen in conscious dogs subjected to a loud noise or made to run on a treadmill after inhaling volatile hydrocarbons.[10 12] Hypoxia,[10] hypokalaemia,[13] and alcohol[14] may all increase the risk of an arrhythmia after solvent abuse, and the cases described by McLeod *et al* (p 727) suggest that there may also be an adverse interaction with halothane.

Studies of acute toxicity have shown that inhaled volatile hydrocarbons can also induce bradyarrhythmias[15 16] and hypotension[17]; these observations were made, however, in animals under general anaesthesia and may not therefore be relevant to those who sniff glue or inhale from aerosol cans.

The relative toxicity of the many chemicals inhaled is not known, largely because we do not know the prevalence of abuse of each agent. Nevertheless, all of these substances may be cardiotoxic. Their effects on the heart are probably caused by non-specific physicochemical actions and seem to occur at doses similar to those that affect the central nervous system.[11] This implies that any sniffer who obtains a "high" also runs the risk of developing an arrhythmia, particularly if he or she then exercises.

Rhabdomyolysis, renal and hepatic damage, and various neurological, psychiatric, and metabolic syndromes have been attributed to habitual solvent abuse.[1 2 13] Now Wiseman and Banim (p 739) and McLeod *et al* (p 727) report three cases of dilated cardiomyopathy associated with chronic solvent abuse or in one case heavy occupational exposure to 1, 1, 1-trichloroethane. Although there are one or two similar case reports,[18 19] the evidence that volatile hydrocarbons may cause a dilated cardiomyopathy is still only anecdotal. Nevertheless, physicians should consider chronic solvent exposure when treating patients with dilated cardiomyopathy, and anaesthetists should be aware of the potential hazards of using halothane or similar agents in patients who may be solvent sniffers.

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Drums begin to beat in the waiting list jungle

Radical change in medical practice is thought to be necessary to shorten the time patients wait for treatment and investigation in the National Health Service. Just last month the government allocated another £25m to help health authorities reduce their waiting lists (28 February, p 590), and last week saw surgeons and managers meeting together at the King's Fund to discuss what should be done (p 783). If quicker is better—and in some cases present treatment delays mean no treatment[1 2]—are doctors willing to alter the referral system and assist their patients to earlier treatment? Will general practitioners refer patients away from the local hospital and named consultant on to others who can see, diagnose, and treat more quickly? Can consultants be persuaded to send their patients on to colleagues within their specialty who can offer quicker treatment?

There is a practical difficulty. Despite modern communications, advances in technology, and the Körner reports on information systems in the NHS, there is no system within