term treatment of the Zollinger-Ellison syndrome with omeprazole is, however, effective and safe.[22]

The whole range of treatments, from antacids to total inhibition, are needed in gastro-oesophageal reflux disease. Here the acid reflux ranges from a slight increase to severe and prolonged exposure of the oesophageal mucosa to acid[23]—often resistant to conventional treatment. Multiple trials have shown that for the milder types of oesophagitis (grades 1 and 2) the H_2 receptor antagonists combined with antacids are effective in most cases.[24] Treatment, however, often relieves symptoms better than it heals, and commonly has to be continued for two months or more and sometimes indefinitely. Increasing the dose of the H_2 receptor antagonist may improve the healing rate a little.

The more severe the oesophagitis (grade 3, circumferential inflammation; grade 4, deep ulcerations or a stricture) the greater the degree of acid suppression usually needed to achieve healing. The patients do particularly well with omeprazole.[25] Here the difference in efficacy between the H_2 receptor antagonists and omeprazole is sharp, with two month healing rates across the range of oesophagitis for omeprazole reaching more than 85% compared with about 40-60% with H_2 receptor antagonists.[24 26] Patients with severe symptoms or dysphagia should be assessed endoscopically to confirm the diagnosis. High dose H_2 receptor antagonists and alginate antacids may be tried, but if these are not fully effective omeprazole should then be used. Symptoms often improve considerably with omeprazole, and the patient may well press the doctor hard for renewal of the prescription, but it is licensed only for short term use. So at present, after a two month course of omeprazole at a dose of 20 mg (but not all patients respond to 20 mg, and 40 mg may be needed) full dose maintenance with an H_2 receptor antagonist would seem practical, with the course of omeprazole being repeated later if needed. If this approach produces no relief it would be reasonable to treat continuously, especially in the elderly—but only after discussing the implications with the patient. Although omeprazole seems to be safe, however, uncertainty about its long term effects raises a question over this option for younger patients until we have more experience of proton pump inhibitors. Nowadays acid suppression really can be adjusted to suit the patient, but care should be exercised, especially with newer, very powerful compounds.

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Diabetic autonomic neuropathy

A common complication which rarely causes symptoms

Autonomic neuropathy has been recognised as a common complication of diabetes mellitus for many years.[1] Often asymptomatic and diagnosed only on routine screening, it is, indeed, usually defined on the basis of abnormal results of tests of the autonomic nervous system.[1] Using data from cross sectional studies, Ewing and Clarke have suggested that the sequence of autonomic damage begins with loss of sweating in the feet, impotence, and bladder dysfunction, and progresses through abnormalities in the cardiovascular reflexes to a final stage of symptomatic postural hypotension, sweat disturbances of the upper body, gastroparesis, diarrhoea, and bladder atony.[1] This sequence cannot be inevitable, however, as symptomatic patients are so rare.[2] A recent community survey in Britain found a single abnormal cardiovascular autonomic response in 16% of patients with diabetes (both insulin dependent and non-insulin dependent)[2]; this prevalence is similar to reported values from randomly selected, hospital based populations.[3] Although it can affect most body systems, symptomatic autonomic neuropathy is much less well defined[1] and may be more common in insulin dependent patients.[2] The annual incidence is unknown, although abnormal cardiovascular reflexes can be found after only two years of insulin dependent diabetes.[4]

Symptomatic patients have a poor prognosis with up to half of them dying within five years in the most widely quoted series.[5] A recent editorial, however, reported lower mortality figures of 27% for symptomatic patients and 10% for asymptomatic patients after 10 years of follow up.[6] Many of the patients concerned, however, had coexistent nephropathy, and the impact of the autonomic neuropathy alone is not clear. Sudden cardiopulmonary arrest has been described in symptomatic patients, often in association with general anaesthesia,[7] and this may be related to defects such as prolongation of the QT interval[8 9] or to silent myocardial infarction.[10] A prospective study of a group of 17 diabetic patients undergoing eye surgery showed that 35% required treatment with a vasopressor during surgery compared with only 5% of non-diabetic controls, and those with worse autonomic dysfunction tended to need intervention more frequently.[11]

How, then, is autonomic neuropathy diagnosed? The many tests of autonomic function have been extensively reviewed.[1 12] A consensus statement from the American Diabetes Association and the American Academy of Neurology has recommended three tests for routine assessment of autonomic function: firstly, heart rate responses to Valsalva's

manoeuvre, deep breathing, and standing; secondly, the response of blood pressure to standing or tilting; and, thirdly, the sweat response to heat or chemicals such as acetylcholine.[13] The selection of these tests has been questioned, however, largely on the grounds of method and interpretation.[14,15] Some have also criticised the consensus statement for ignoring sympathoadrenal dysfunction,[16] which may precede obvious abnormalities in cardiac reflexes[17] and therefore help in evaluating preventive treatments.

A compromise solution that would achieve standardisation and satisfy diagnostic requirements without loss of sensitivity would require a slight modification of the battery of tests proposed by Ewing and Clarke[18] by using a single Valsalva manoeuvre instead of three, three deep breaths instead of six, and age adjusted normal ranges. Results from these tests performed in different centres show comparable and stable coefficients of variation with time,[19] and computer assisted systems for data collection and analysis are now widely available.[14,20] Despite differences in the sensitivity of tests careful studies have shown that the distribution of autonomic damage is usually not patchy, that it affects sympathetic and parasympathetic function equally, and that there is a good correlation between cardiovascular reflex abnormalities and damage in other systems.[1] Newer tests that use a pulse oximeter[21] or modifications of older ones such as the diving reflex[22] have yet to be fully evaluated in diabetic patients. The increasingly widespread use of pulse oximetry in anaesthesia may make for easier diagnosis in patients about to undergo general anaesthesia, and this technique probably ought to be evaluated by a prospective study.

What is the pathological basis of autonomic neuropathy? Studies have shown a mixture of peripheral axonal loss and segmental demyelination with preservation of cell numbers in the spinal cord.[23] Inflammatory changes have been seen in autonomic ganglia and have suggested an immunological aetiology to some authors,[24] although a metabolic causation is more likely, possibly by activation of the sorbitol pathway.[25] Ischaemia secondary to microvascular damage has also been proposed as a mechanism.[26]

Can the process be affected by treatment? Patients in whom blood sugar concentration has been restored towards normal by either continuous subcutaneous insulin infusion or transplantation of the pancreas have shown either slight[27] or no improvement[28,29] in cardiovascular autonomic neuropathy after two or more years. In the most recent study a tendency for stabilisation was seen 42 months after transplantation of the pancreas while the condition of control patients continued to deteriorate.[30] Cardiovascular reflexes have been shown to be impaired in newly diagnosed insulin dependent patients after two years of poor control but to remain normal in those with good control.[4] Prevention of rather than reversal of autonomic neuropathy may thus be more easily attainable. The effects of aldose reductase inhibitors in humans have so far been disappointing.[31]

Before treatment of the symptoms of neuropathy is considered any potentially exacerbating factors should be excluded, particularly possible side effects from concurrent medications. Impotence is the most commonly reported symptom but has multiple aetiologies, and careful evaluation of the individual patient is necessary before ascribing its cause to autonomic neuropathy.[32,33] Symptomatic postural hypotension may sometimes be improved by simple measures such as raising the head of the patient's bed, but drug treatment is often required, with fludrocortisone as the agent of first choice.[1,12] Its use is often limited by side effects, however, in which case prostaglandin synthase inhibitors or domperidone[34] may be tried. β Blockers are now thought not to help.[35]

There is a poor correlation between gastrointestinal symptoms and objective evidence of neuropathy,[36] and some dispute whether diarrhoea is a true manifestation of autonomic dysfunction,[37] largely because it often responds to antibiotics. If these fail loperamide or clonidine may be tried.[1,12,37] Constipation is a more common gastrointestinal symptom but has an even less well understood aetiology: it may respond to metoclopramide or domperidone.[1,12] These drugs are also used in patients with symptomatic gastroparesis, and a recent study of treatment with intravenous erythromycin has shown improved gastric emptying in 10 patients with proved gastroparesis, probably as a result of its motilin-like activity.[38] It was much less effective, however, when taken orally. Pregnancy may exacerbate symptomatic autonomic neuropathy, particularly gastroparesis and postural hypotension, and poses therapeutic limitations because of considerations for the fetus. Patients need careful monitoring, particularly in early pregnancy, when vomiting may be a serious problem.[39,40]

In summary, abnormalities in autonomic reflexes are common in diabetic patients, but few have symptoms. Though universal screening is probably not indicated, diabetic patients need particularly careful cardiovascular monitoring during the induction and maintenance of general anaesthesia, and routine preoperative evaluation of the autonomic nervous system should perhaps be considered.

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Detecting bladder cancer

Screening is of doubtful benefit, but all patients with haematuria should be investigated

Screening for bladder cancer was probably the first screening programme for any tumour. In low risk groups dipstick assessment of urine for microscopic haematuria is the main test used in screening for occult lesions of the urinary tract and the finding of blood in the sample has high predictive accuracy.[1] In high risk groups cytological examination of multiple urine samples remains the first line technique for detecting malignant lesions.[2] The sensitivity of cytological tests varies widely, but flow cytometry of urine seems to be the most sensitive method of detecting malignant bladder cells, though this may be because it relies on bladder irrigation samples.[2] Unfortunately, regular widespread sampling is impracticable and the test is expensive.

Studies of screening in the general population (low risk) have shown large variations in the numbers of patients with occult haematuria; in general screening identifies only a few people with occult malignancies. One study found positive dipstick haematuria in 255 out of 10 050 (2·5%) men aged 21-72 years,[3] whereas another found that 132 out of 578 (23%) men aged 60-85 gave positive results.[4] Seventy six of the 255 (30%) patients with positive results in the first study were known to have undergone further urological investigations, and 21 (28%) had urinary tract abnormalities, two of them bladder tumours. Investigations in 87 of the 132 (66%) patients with positive results in the second study showed urological disease in 45 (52%), four of whom had bladder tumours.

The most important issue in any screening programme is whether there is irrefutable evidence that the results of screening tests have prevented or postponed either the onset of the disease or early death due to that disease.[5] Is there, therefore, any benefit in early diagnosis, and can the natural course of bladder cancer be altered by early diagnosis?

In high risk groups, one recent study showed that there was no excess in mortality from bladder cancer in men working in the rubber industry compared with matched controls in the general population.[6] There is, indeed, little evidence to support earlier claims of an improvement in life expectancy in high risk patients in whom transitional cell carcinoma was diagnosed on routine screening.[7] The preliminary conclusion of a recent international meeting in Cincinnati on screening for bladder cancer was that existing data on both high and low risk groups could neither confirm nor refute any positive benefit in life expectancy from early diagnosis (P A Schulte, personal communication). Controlled large scale prospective studies have not been undertaken and are impracticable.

Views differ, too, on the cost effectiveness of screening for bladder cancer. Fraser *et al* found that their screening programme added to hospital costs without benefit to the patient.[5] Using a mathematical model, however, Ellwein and Farrow suggested that cytological screening—with its predilection for identifying high grade, aggressive tumours—would be cost effective in patients in their 60s and 70s and would yield a projected increase in life expectancy of three years or more in patients without symptoms who were truly positive for tumours.[8] In terms of increased life expectancy from early diagnosis of tumours, continuing cytological screening in high risk groups is therefore of doubtful benefit. Why, then, do we continue to screen? Certain screening programmes in high risk groups are required by statute.[9] While uncertainty of benefit remains and industrial compensation is at issue careful surveillance should continue in new high risk groups such as people working with plastic mouldings who handle 4,4′ diamino-3,3′ dichlorodiphenylmethane (MBOCA).[10]

Long term screening programmes may serve to alleviate patients' anxieties. Conversely, regular surveillance may promote anxieties.[11] It is too early to know whether regular infusion of agents such as BCG into the bladder is effective in reducing the progression of dysplasia or carcinoma in situ.

The benefit of dipstick screening for haematuria to detect carcinoma of the bladder in the general population also remains unproved. When the patients with positive results are investigated only a few have tumours. Although larger numbers are found to have other urological disorders, probably less than half benefit from early treatment. At present, all sorts of health prevention programmes, including screening, are being promoted by private and government institutions alike—often without good evidence of their value. Furthermore, even those patients found to have microscopic haematuria on screening may not be referred by their general practitioners for further investigations.[3 5]

The overall picture, then, is confused and unsatisfactory. For the present it seems unnecessary to screen the general population at all ages for bladder cancer, even though bladder tumours may occur in patients aged under 40.[3] Yet despite screening being of doubtful benefit it remains imperative to investigate any incidental finding of haematuria by dipstick analysis, particularly in older people, even in those with a trace of blood. Doctors should be aware that haematuria, even in the presence of identifiable disease, may occur intermittently.[4] With the increasing accuracy of ultrasonography and availability of outpatient flexible cystoscopy examination, each patient can be thoroughly investigated for roughly £50. Integrated, single visit haematuria clinics may soon be commonplace.

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