be noted. Only with such detailed information will fair comparisons be possible.

The real differences in surgical mortality that do exist between surgeons and centres may teach important lessons. One study compared mortality between a teaching hospital and its adjacent district hospital and led to a proper intensive care unit being provided in the district hospital.[14] The confidential inquiry into perioperative deaths emphasised the value of regular reviews of mortality for teaching and training and the importance of organisation.[2] Some types of surgery should undoubtedly be concentrated in fewer hands. The development of management protocols for procedures with a high mortality and the mandatory participation of senior medical and surgical staff is also inevitable.[15]

NEIL MORTENSEN

Consultant Surgeon,
John Radcliffe Hospital,
Oxford OX3 9DU

1 Gruer R, Gordon DS, Gunn AA, Ruckley CV. Audit of surgical audit. *Lancet* 1986;i:23-25.
2 Buck N, Devlin HB, Lunn JN. *Report of the confidential enquiry into perioperative deaths.* London: Nuffield Provincial Hospitals Trust and King's Fund for Hospitals, 1987.
3 Anonymous. Second-best prostatectomy. [Editorial]. *Br Med J* 1980;**280**:590.
4 Wennberg JE, Roos N, Sala L, Schovi A, Jaffe R. Use of claims system to evaluate health care outcomes. Mortality and re-operation following prostatectomy. *JAMA* 1987;**257**:933-6.
5 Maevki SC, Luft HS, Hunt SS. Selecting categories of patient for regionalised care. *Med Care* 1986;**24**:148-58.
6 Earlam R. Oesophageal cancer treatment in North East Thames region 1981: medical audit using Hospital Activity Analysis data. *Br Med J* 1984;**288**:1892-4.
7 Matthews HR, Powell DJ, McConkey CC. Effect of surgical experience on the results of resection for oesophageal carcinoma. *Br J Surg* 1986;**73**:621-3.
8 Fielding LP, Stewart-Brown S, Blesovsky L, Blesovsky L, Kearney G. Anastomotic integrity after operations for large-bowel cancer: a multicentre study. *Br Med J* 1980;**281**:411-4.
9 West JG, Trunkey DD, Lim RC. Systems of trauma care. Study of two counties. *Arch Surg* 1979;**114**:455-60.
10 Anderson IA, Woodford M, de Dombal FT, Irving M. Retrospective study of 1000 deaths from injury in England and Wales. *Br Med J* 1988;**296**:1305-8.
11 Brown SCW, Walsh S, Sykes PA. Operative mortality rate and surgery for colorectal cancer. *Br J Surg* 1988;**75**:645-7.
12 Knaus WA, Draper EA, Wagner DP, Zimmerman JE. APACHE II: severity of disease classification system. *Crit Care Med* 1985;**13**:818-29.
13 Schein M. Acute surgical disease and scoring systems in daily surgical practice. *Br J Surg* 1988;**75**:731-2.
14 Gilmore OJA, Griffiths NJ, Connolly JC, *et al.* Surgical audit. Comparison of the workload and results of two hospitals in the same district. *Br Med J* 1980;**281**:1050-2.
15 Jennet B. Variations in surgical practice: welcome diversity or disturbing differences. *Br J Surg* 1988;**75**:630-1.

Rediscovering monoamine oxidase inhibitors

Benefits underestimated, side effects exaggerated

Monoamine oxidase inhibitors were introduced 30 years ago but fell from favour because of their alarming interaction with foods containing tyramine[1,2] and their poor performance in a large trial of antidepressants.[3] Now new American studies are causing psychiatrists to re-evaluate this category of drugs.[4-6]

The results of these studies have confirmed earlier findings that the monoamine oxidase inhibitor phenelzine is superior to placebo[7] and imipramine[8] in agoraphobic patients with panic attacks. Most improvement occurs in measures of phobic anxiety and avoidance, but the frequency of the attacks is reduced. This anxiolytic effect does not depend on the patient also having depressive symptoms.[9]

Monoamine oxidase inhibitors are also effective in depression that is not of the classic endogenous type.[4,10] Electroconvulsive therapy may not be effective in these milder forms of depression, and tricyclic antidepressants may be contraindicated. The patient who is likely to respond to monoamine oxidase inhibitors often reports "atypical" features such as mood reactivity (a transient remission from depressed mood in response to positive environmental factors), overeating, oversleeping, extreme fatigue when depressed, and chronic oversensitivity to rejection. Almost three quarters of patients who show mood reactivity and two or more of the other features will respond to phenelzine (45-90 mg daily), whereas only half respond to impramine (150-300 mg daily)—a response that is not significantly better than placebo.[10] Phenelzine may also produce a good response in patients with panic symptoms, panic attacks, hostility, a self pitying attitude with symptoms of blaming others, irritability, and hypochondriasis[11] and in those with a labile personality, interpersonal sensitivity, and "touchiness."[10]

The efficacy of monoamine oxidase inhibitors in such a wide range of disorders suggests that their actions are not specifically anxiolytic, antiphobic, or antidepressive. This has led Tyrer to conclude that they should be prescribed for agoraphobia, panic episodes, hypochondriasis, irritability, somatic anxiety, and anergia.[12]

Until recently psychiatrists agreed that monoamine oxidase inhibitors do not help patients with neurotic symptoms secondary to a lifelong inadequate personality.[13] But in a recent double blind crossover trial the monoamine oxidase inhibitor tranylcypromine proved to be better than lorazepam, carbamazepine, trifluoperazine, and placebo in helping patients with borderline personality disorder (characterised by prominent rejection sensitivity and problems with impulse control).[14] There was a clear improvement in mood and a less prominent improvement in abnormal behaviour. A subgroup of patients with borderline personality may have features of atypical depression and thus respond preferentially to a monoamine oxidase inhibitor. These findings require replication.

Many physicians and general practitioners do not prescribe monoamine oxidase inhibitors because they doubt their safety. The drugs have anticholinergic side effects such as mild sedation, dry mouth, blurred vision, difficulty with micturition, and ejaculatory failure, but these are generally less severe than those produced by tricyclic antidepressants and should not be a deterrent to prescribing.[15] The main fears about the interaction of monoamine oxidase inhibitors with tyramine in foodstuffs have been grossly exaggerated.[16,17] At the height of their use only 17 cases of interaction with food were reported in 10 years. Between January 1975 and December 1983 tranylcypromine was prescribed in 98 000 patient years. Patients should be told, however, to avoid those foods that interact with the drugs—for example, cheese and yeast and beef extracts—and patients should always be given a card to say that they are taking the drugs. Of the monoamine oxidase inhibitors available, tranylcypromine has the greatest propensity to produce these interactions and phenelzine the least. Occasional cases of addiction with tolerance occur, especially with tranylcypromine, and withdrawal symptoms (headache, shivering, paraesthesia, and nightmares) may develop within days after stopping phenelzine and isocarboxazid.[18] For this reason gradual reduction in dosage is the best form of withdrawal.

Monoamine oxidase inhibitors are thus effective in patients with phobic disorders and in those with non-endogenous depression accompanied by somatic anxiety (including panic symptoms) and atypical features. The decision to prescribe the drugs should depend, however, not only on the symptoms but also on factors such as the duration of the illness, the response to previous drugs and other treatments, the patient's

BMJ: first published as 10.1136/bmj.298.6670.345 on 11 February 1989. Downloaded from http://www.bmj.com/ on 24 June 2019 by guest. Protected by copyright.

premorbid personality, and an estimate of the patient's ability to comply with dietary restrictions. The drugs should probably not be used in patients who abuse alcohol and in those with panic and hypochondriacal symptoms who are unable to tolerate even the slightest side effect—for example, dizziness—because it causes further anxiety. For such patients a treatment such as cognitive therapy may be better,[19] but monoamine oxidase inhibitors may be particularly useful in agoraphobics and patients with phobic avoidance who are either unable or unwilling to engage in behavioural treatment.[20]

CHRISTOPHER BASS

Senior Lecturer,
Academic Department of Psychological Medicine,
King's College Hospital,
London SE5 9RS

ROBERT KERWIN

Honorary Lecturer in Psychiatric Neuropathology,
Institute of Psychiatry,
London SE5 8AF

1 Blackwell B, Marley E, Price J, Taylor D. Hypertensive interaction between monoamine oxidase inhibitors and foodstuffs. *Br J Psychiatry* 1967;**113**:349-51.
2 Wright SP. Hazards with monoamine oxidase inhibitors: a persistent problem. *Lancet* 1978;i: 284-5.
3 Medical Research Council. Report of clinical psychiatry committee: clinical trial of the treatment of depressive illness. *Br Med J* 1965;**251**:881-6.
4 Liebowicz MR, Quitkin FM, Stewart JW, *et al*. Antidepressant specificity in atypical depression. *Arch Gen Psychiatry* 1988;**45**:129-37.
5 Janicak PG, Paudey GN, Davis JM, Boshes R, Bresnahan D, Sharma R. Response of psychotic and nonpsychotic depression to phenelzine. *Am J Psychiatry* 1988;**145**:93-5.
6 Quitkin FM, Stewart JW, McGrath PJ, *et al*. Phenelzine versus imipramine in the treatment of probable atypical depression: defining syndrome boundaries of selective MAOI responders. *Am J Psychiatry* 1988;**145**:306-11.
7 Tyrer P, Candy J, Kelly D. Phenelzine in phobic anxiety: a controlled trial. *Psychol Med* 1973;**3**:120-4.
8 Sheehan DG, Ballenger J, Jacobson G. Treatment of endogenous anxiety with phobic, hysterical, and hypchondriacal features. *Arch Gen Psychiatry* 1980;**37**:51-9.
9 Pohl R, Berchou R, Rarney JM. Tricyclic antidepressants and monoamine oxidase inhibitors in the treatment of agoraphobia. *J Clin Psychopharmacol* 1982;**2**:399-407.
10 Liebowicz MR, Quitkin FM, Stewart JW, *et al*. Phenelzine versus imipramine in atypical depression: a preliminary report. *Arch Gen Psychiatry* 1984;**41**:669-77.
11 Kay DWK, Garside RF, Fahy TJ. A double blind trial of phenelzine and amitriptyline in subtypes of out-patient depression. *Br J Psychiatry* 1973;**123**:63-7.
12 Tyrer P. Towards rational therapy with monoamine oxidase inhibitors. *Br J Psychiatry* 1976;**128**:354-60.
13 Pare CMB. The present status of monoamine oxidase inhibitors. *Br J Psychiatry* 1985;**146**:576-84.
14 Cowdry WL, Gardner DL. Pharmacotherapy of borderline personality disorder. *Arch Gen Psychiatry* 1988;**45**:111-9.
15 Rabkin J, Quitkin F, Harrison W, *et al*. Adverse reactions to monoamine oxidase inhibitors. Part 1. A comparative study. *J Clin Psychopharmacol* 1984;**4**:270-8.
16 McGilchrist JM. Interactions with monoamine oxidase inhibitors. *Br Med J* 1975;**271**:591-2.
17 Shaw DM. The practical management of affective disorders. *Br J Psychiatry* 1977;**130**:432-51.
18 Tyrer P. Clinical effects of abrupt withdrawal from tricyclic antidepressants and monoamine oxidase inhibitors after long-term treatment. *J Affect Disord* 1984;**6**:1-7.
19 Clark DM. Cognitive therapy for anxiety. *Behvioural Psychotherapy* 1986;**14**:283-94.
20 Marks IM. *Fears, phobias, and rituals*. Oxford: Oxford University Press, 1987.

Surgery for constipation

Sometimes justified for the idiopathic slow transit type

Surgery can usually produce a good functional result in dysfunction of the colon secondary to lesions such as aganglionosis or cancer. But its role in treating idiopathic constipation is more controversial and difficult. Patients with idiopathic constipation, particularly young women, are greatly distressed by their symptoms and in some the features are appreciably improved after operation—so we have to ask which patients should be operated on and by what procedure.

Initially, constipation was thought to be due to a colonic disorder in which the transit of faeces through the large bowel was delayed[1]; such patients might be expected to improve after colectomy. Nevertheless, later it was found that in some patients constipation was a disorder of defecation related to dysfunction in the pelvic floor musculature.[2] Such patients might be expected to benefit from pelvic floor surgery. Now the clinical picture is known to be further complicated because some patients may have both disorders.

Partial colectomy gives a poor clinical result in patients with slow transit constipation.[3] On the other hand, subtotal colectomy and ileorectal anastomosis greatly improve bowel function,[3,4] though caecorectal anastomosis is less effective.

Pelvic outlet obstruction (anismus) may result either from dysfunction of the internal sphincter (hypertonia) or from failure of the puborectalis muscle to relax normally during defecation. The first abnormality has been treated with anorectal myectomy, with mixed results,[5,6] and the second with lateral or posterior division of the puborectalis with disappointing results.[7,8]

Hence there are no clear answers to the two questions asked at the beginning. Surgery cannot be advocated for pelvic outlet obstruction at present, at least until the value of anorectal myectomy has been studied. On the other hand, patients with idiopathic slow transit constipation can be offered subtotal colectomy with ileorectal anastomosis. The decision to proceed to surgery should be made only when all medical measures have failed and preferably after the patient has had a psychiatric assessment. Surgery can restore reasonable bowel function but at a considerable cost. Many patients develop severe diarrhoea, which in turn may lead to disabling faecal incontinence, and over two thirds of them continue to complain of abdominal pain postoperatively.[4]

M M HENRY

Consultant Surgeon,
Central Middlesex Hospital,
London NW10

1 Hinton JM, Jones JEL. Constipation: definition and classification. *Postgrad Med J* 1968;**44**:720-3.
2 Preston DM, Jones JEL. Anismus in chronic constipation. *Dig Dis Sci* 1985;**30**:413-8.
3 Preston DM, Hawley PR, Jones JEL, Todd IP. Results of colectomy for severe constipation in women (Arbuthnot Lane's disease). *Br J Surg* 1984;**71**:547-52.
4 Kamm MA, Hawley PR, Jones JEL. Outcome of colectomy for severe idiopathic constipation. *Gut* 1988;**29**:969-73.
5 Martelli H, Devroede G, Arhan P, Duguay C. Mechanisms of idiopathic constipation: outlet obstruction. *Gastroenterology* 1978;**75**:623-31.
6 Yoshioka K, Keighley MRB. Anorectal myectomy for outlet obstruction. *Br J Surg* 1987;**74**:373-6.
7 Barnes PRH, Hawley PRH, Preston DM, Jones JEL. Experience of posterior division of the puborectalis muscle in the management of chronic constipation. *Br J Surg* 1985;**72**:475-7.
8 Kamm MA, Hawley PR, Jones JEL. Lateral division of the puborectalis muscle in the management of severe constipation. *Br J Surg* 1988;**75**:661-3.

BMJ: first published as 10.1136/bmj.298.6670.345 on 11 February 1989. Downloaded from http://www.bmj.com/ on 24 June 2019 by guest. Protected by copyright.