PRACTICE OBSERVED

Sex Problems in Practice

Iatrogenic sex problems

ERIC TRIMMER

Doctors have in the past produced many sex problems by their attitudes and prescriptions. Today this largely unconscious process is still at work but drugs rather than attitudes are often the main offenders. Three main groups of drugs have a well-earned reputation for causing sex problems: (1) psychotropic drugs; (2) anti-hypertensives; (3) anti-depressants and H₂-receptor antagonists. A doctor must take a sexual history before prescribing these drugs or else he will not be able to decide whether the drug or the disease is causing sex problems.

Psychotropic drugs

ANXIOLYTICS

The ultimate anxiolytic, which would subdue the part of the limbic system involved in anxiety while leaving the rest of the brain unaffected, remains regrettably a pharmacological pipe-dream. Despite enthusiastic claims to the contrary, most of our current anxiolytics affect sexual function just like the older central nervous system depressants—that is, barbiturates and alcohol. As the number of prescriptions for anxiolytics exceeds 25 million a year the iatrogenic problems are potentially enormous. Anxiolytics in certain circumstances, however, have a beneficial effect on sexual function. But large doses of barbiturates suppress the release of pituitary gonadotrophins and also by enzyme induction catalyse circulating sex hormones. Epileptic patients with drug-induced sex problems improve if their barbiturate dosage is reduced. When libido and orgasm

return barbiturate dosage can often be further reduced without increasing the frequency of seizures. Misuse and overdose of benzodiazepines occasionally produce disorders of sexual desire and function (particularly erectile failure and "spectatoring"). This is especially likely if they are taken with alcohol. Beta-blockers, which are sometimes used as anxiolytics, only rarely produce sex problems, but they may cause desire disorders because of their depressant action on the central nervous system.

ANTI-DEPRESSANTS

Again when considering the iatrogenic effects of these useful drugs it is important to remember that depression is often complicated by desire and arousal disorders. Treatment may either alleviate these or unwittingly graft sex problems onto an existing disability. The two commonly used groups of anti-depressant drugs have different iatrogenic effects. Monoamine reuptake inhibitors (MARI) are sometimes strongly anti-cholinergic and thus may cause erectile failure. Imipramine, desipramine, and protriptyline can all do this. Other MARI drugs are alpha-adrenoceptor blockers, which delay or abolish ejaculation. Clomipramine and amitriptyline have this effect. In women the iatrogenic effects of MARI compounds are somewhat confused.

Monoamine oxidase inhibitors (MAOI) also inhibit ejaculation and are sometimes implicated in erectile failure. Lithium carbonate has been reported to cause desire disorders, but this is probably a red herring as the increased libido of some manic patients returns to normal with lithium treatment, and yet some patients "blame" this on the drug.

*The term "spectatoring" was coined by Masters and Johnson to describe a condition in which a man directs his activity towards watching evidence of sexual arousal rather than being emotionally involved in a sexual experience.

Lowdwater, Herts 4DE JLE

ERIC TRIMMER, MB, MSc, general practitioner and editor of the *British Journal of Sexual Medicine*

ANTI-PSYCHOTICS

The most likely antipsychotic drugs to cause sex problems are the phenothiazines, which are widely prescribed for schizophrenia. Again problems must be examined against the backdrop of the underlying disease. Perhaps a third of all schizophrenics have sexual desire or arousal problems, and some men experience dysfunction. The remainder have normal or diminished sexual function. The phenothiazines are liable to cause or worsen sex problems in these groups, and because of this patients may stop taking the drugs. Structurally different antipsychotics, such as haloperidol and pimozide, which cause less sexual impairment, are then indicated. The extent to which prolactin-induced sexual problems occur in patients on antipsychotic drugs is unclear. All antipsychotics block dopamine receptors and thus allow the pituitary to secrete more prolactin. It is well known that primary hyperprolactinaemia is associated with impotence and with desire and arousal problems in women, but drug-induced hyperprolactinaemia may not have the same effect.

ANTI-HYPERTENSIVES

The association between the treatment of hypertension and sex problems is complex and confused. The diagnosis of "impotence" always requires qualification (is it primary, secondary, situational, etc.). But if "impotence" is accepted as a rough-and-ready "diagnosis" it is reported to occur in about 7% of men. It occurs in 17% of hypertensive patients, and in about 25% of treated hypertensive patients. (For failure of ejaculation the equivalent figures are 0%, 7%, and 26%.)

Considerable differences in iatrogenic effects are reported for different antihypertensives, but unfortunately the same criteria have not been applied in all studies. It has been stated that about 5% of men chronically using the thiazide diuretics, ethacrynic acid, and frusemide experience disorders of potency attributable to the drug, but that potassium supplementation ameliorates the condition. This is not universally accepted and the incidence of impotence in patients taking diuretics alone has been reported elsewhere to be as high as 32%, and the incidence of ejaculation failure 14%. With such discrepancies other complicating factors may well be operating—for example, the presence or absence of pelvic arterial disease.

The adrenoceptor-blocking agents are universally admitted to cause sex problems. About 40-60% of hypertensive patients treated with guanethidine report "erection difficulties," and about 70% report impaired ejaculation. When bethanidine is prescribed the figures are 65% and 40%. Problems caused by methyldopa seem to be dose-related; if less than 1 g it is taken daily then drug-induced problems of impotence or impaired arousal are reported in 10-15%, of patients (1-1.5 g a day produces a figure of 20-25%, and over 2 g daily 50%). Similarly spiroxolone's effects are dose-related; if less than 100 mg a day is taken then problems do not seem to occur, but doses of 400 mg produced "reduced libido in 22%" of healthy young men in one survey.

Clonidine (thought to act by diminishing sympathetic outflow) was once thought to be free from sexual side effects, but more recently the drug has been said to produce impotence in 10-25% of patients. Beta-blockers were greeted enthusiastically because of their supposed lack of sexual side effects—and with some justification. Now, however, at least one such drug—propranolol—has been shown to produce erectile failure sometimes, which is presumably because it can cross the blood-brain barrier. Another similar drug, acebutolol, which does not cross the blood-brain barrier, allows beta-blockade to continue without such problems. One possible but rare iatrogenic effect of beta-blockade is Pheochromocytoma (induration of the corpora cavernosa of the penis). Clearly the assessment of iatrogenic factors in treating hypertension demands an adequate pretreatment assessment of sexual functioning if any sense is to be made of this complicated subject.

The anti-hypertensives may, however, have one sexual side effect that might be considered a bonus for sufferers. Treatment with prazosin is said to be associated with an incidence of impotence of less than 1%; this raises the possibility of "iatrogenic" erections, which has encouraged some researchers.

Antidyspeptic drugs and H₂-receptor antagonists

Several new drugs for treating peptic ulceration have appeared recently. New drugs usually mean unexpected as well as expected side effects. Extracts of liquorice used for many years to treat dyspepsia are now enjoying fresh interest. Unfortunately they have many side effects, and impotence and desire and arousal problems have been reported. Metoclopramide has several gastrointestinal uses, but it is a potent dopamine-receptor antagonist and so induces hyperprolactinaemia. Although, as I previously mentioned, secondary hyperprolactinaemia is less worrying than primary, four out of five patients taking metoclopramide in one study complained of loss of libido.

The competitive H₂-receptor antagonists are the newest drugs and their introduction produced a new crop of sex problems. Animal experiments have shown that cimetidine has weak antiandrogenic activity, and plasma concentrations of cimetidine during treatment in man are high enough to inhibit and dihydrotestosterone binding. The drug has an effect on the hypothalamic-pituitary-gonadal axis; it also increases plasma prolactin, and gynaecomastia has been reported during treatment. Finally histamine H₂ and H₁ receptors are present in the corpora cavernosa, although what part histamine plays in the complicated physiological mechanism of erection is not yet clear.

Surgery

Surgery has been thought to cause many sexual problems, but often unjustifiably. Many operations on the abdomen or pelvis have been blamed for producing subsequent impotence, when the men operated on have had very low desire for many years previously. Certain operations do, however, produce iatrogenic problems.

PROSTATECTOMY AND ORCHIDECTOMY

Prostatectomy was for years believed to produce a high incidence of erectile failure despite observations of many spontaneous erections after the operation. Transurethral and retropubic prostatectomy probably produce impotence in 5% of patients, and in most cases this impotence is psychogenic. Most men do not ejaculate externally after prostatectomy but retrograde ejaculation occurs into the bladder. Perineal prostatectomy for benign prostatic hyperplasia does, however, have a high (40-50%) chance of producing erectile failure, and when the wider dissection necessary for cancer is carried out the impotence rate approaches 100%.

Orchidectomy and postoperative radiotherapy for testicular tumours produce decreased sexual function in about 3% of men.

GYNAECOLOGICAL SURGERY

Gynaecological surgery may be followed by dyspareunia (especially after vaginal reposition operations), anorgasmia, and lack of libido when malignant disease of the vulva, vagina, or uterus is treated by radiotherapy or surgery, or both. Breast surgery, including mastectomy, is not generally complicated by sexual problems unless the woman's body image is badly distorted by her operation. Counselling both before and after the operation will usually prevent this happening. After a mastectomy about 50% of women resume intercourse within a

month of leaving hospital. The frequency of intercourse is, however, usually reduced.

RECTAL SURGERY

The effects of rectal surgery on sexual functioning depend on the type of operation carried out and on the sex of the patient. An anterior rectal resection, in which the distal rectum is left, has little or no effect on sexual functioning. Abdominoperineal resections, however, have profound effects on men, rendering them impotent. Women may participate in coitus without difficulty and experience orgasm after similar surgery. For either sex a colostomy has little effect on sexual functioning provided the patient receives counselling before and after the operation.

CONTRACEPTION

Vasectomy has adverse iatrogenic effects on sexual functioning in men only if they see the operation as castrating or if there has been previous disagreement with the partner about having the operation. The effect of the contraceptive pill on sexual functioning has been widely and unsatisfactorily debated. It seems reasonable to state that it enhances sexuality for about as many women as it inhibits sexual functioning. Intrauterine devices may inhibit sexual response if pelvic inflammatory disease, dyspareunia, or menorrhagia complicate usage. Tubal procedures carried out for contraceptive purposes are said to be remarkably free from adverse sexual effects, although some women develop diminished libido and depressed arousalability, usually for psychological reasons.

Trainees' Corner: Managing Chronic Disease

Chronic bronchitis

I: Diagnosis and investigations

LEONARD ROGERS, S E JOSSE

This article is based on an audiotaped presentation made for vocational trainees in general practice by the MSD Foundation. Further information about the tape-slide programme on which this series is based is available from the MSD Foundation, Taviton House, Taviton Square, London WC1.

Chronic bronchitis is common and has been extensively studied, and it is surprising therefore that there are many aspects of its aetiology about which we are still ignorant. Although there is evidence that the impact of chronic bronchitis, judged by its mortality rate, has lessened slightly over the past 20 years the morbidity rate has not decreased.

Every general practitioner still has many bronchitic patients in his practice. Many GPs, however, find chronic bronchitis a depressing disease to treat: one survey showed that they find treatment relatively ineffective; they have difficulty arranging hospital admission; and find it hard to stop patients smoking. But the same survey showed that a strong relationship between the GP and his patient leads to the patient having a much higher morale—even when he is physically deteriorating.

A bronchitic patient

Arthur Cook is 53 and married with one son. His job is on the shop-floor in the stores of a heavy engineering works. You have not seen him before, but he has been attending your practice since moving

Buntingford, Hertfordshire

LEONARD ROGERS, formerly deputy director, MSD Foundation

London N11 2RD

S E JOSSE, MB, FRCP, general practitioner

to the area about seven years ago. He smokes about 20 cigarettes a day, the first with his early morning cup of tea. He no longer smokes them down to the butt, having seen all the publicity about cancer.

Before we consider management we want to look at the disease itself, not just to refresh your memory but also to give some perspective to what follows. Firstly, what do we mean by the term "chronic bronchitis"?

Definition

The definition of chronic bronchitis that is generally accepted is based on the symptom of persistent expectoration and includes no reference to any disability. This definition was originally adopted mainly for epidemiological rather than for clinical purposes. Undoubtedly the principal cause of mucus hypersecretion is prolonged irritation, particularly from cigarette smoking, dust, fumes, and atmospheric pollution. The hypertrophy of mucous glands in large airways occurring as a result of irritation does not, however, necessarily lead to airflow obstruction. Almost all the sputum expectorated by the patient with chronic bronchitis comes from these large airways, and there may be no correlation between the amount of sputum regularly expectorated and the severity of airflow obstruction. Consequently a patient with a productive cough (who is by the conventional definition a chronic bronchitic) may have minimal airways obstruction, while, conversely, a patient with an only minimally productive cough may have airways obstruction that is severe.

A far more important cause of airways obstruction is hypersecretion of mucus in the small airways, where the epithelium undergoes metaplasia to goblet cells. The obstruction may be temporary if the mucus is removed by ciliary action, but hypersecretion may lead to the small airways being permanently damaged and eventually obliterated. Recurrent or persistent

infection, especially affecting the small airways, has long been thought to be the principal cause of their damage and hence of progressive airways obstruction, but there is as yet no definite proof of this, and it remains one of the most controversial aspects of the disease.

Pathogenesis

Haemophilus influenzae and to a lesser extent *Streptococcus pneumoniae* are the commonest organisms isolated from sputum during acute attacks. The haemophilus is found as a commensal in the upper respiratory tract, but in favourable circumstances it can invade the lower respiratory tract, and once established in the bronchi may never be eradicated. It has the ability to produce toxins that not only inhibit ciliary action but also damage epithelial cells, releasing factors that potentiate the growth of the haemophilus—a vicious circle.

Bronchoscopic studies have shown that there is often a latent bacterial infection in the lower respiratory tract that can be activated by various insults; probably the commonest is an acute exacerbation due to a virus, which is often described by the patient as a "cold going down to the chest." Several studies have indicated that rhinoviruses are numerically the most important viruses to precipitate exacerbations, probably due to the large number of distinct serotypes. About 100 have been identified already, and infection by one confers no cross-immunity against others. Fig 1 shows an experimental tissue

culture section of a normal bronchus, and fig 2 the effect of five days' exposure to rhinovirus. Fig 3 shows the bronchus after 10 days when the mucous membrane has been considerably thinned and the cilia virtually obliterated.

The two types of influenza virus, A and B, although less frequently seen, are highly virulent and can cause a severe bronchitis in patients who have pre-existing damage to the bronchi. This is almost always accompanied by a secondary bacterial infection, of which perhaps the most serious is staphylococcal. Epidemics of influenza always produce definite peaks in mortality rates from chronic bronchitis.

Up to now research on pathogenesis has tended to concentrate on infection and environmental factors, notably irritants such as smoking and atmospheric pollution. A genetic predisposition to chronic bronchitis has been suggested, however, as well as to some forms of emphysema. Although it is not yet possible to identify the people who will succumb to deteriorating respiratory function and eventual disability, there are certain guidelines which can give some help: firstly, the patient's family history—for instance, a high incidence of chest infections; and secondly, a review of his own history—in particular, the frequency of chest infections in childhood and his immediate past (two or three episodes in the past year suggest susceptibility). Also at risk is the middle-aged smoker who may be free from complaints at the time, but who may have a degree of airflow obstruction that can only be shown by lung function tests.

Arthur again

Let's now take up the problem of Arthur Cook. He arrives at your surgery one winter morning because his cough is playing him up, particularly in the early morning on his way to work. He does not volunteer that his cough may be related to his smoking, apart from helping to "clear his chest" to start the day off. He says he has had several colds going to his chest recently and is a bit worried about being breathless climbing stairs and wonders if it is his heart. His past notes (which are all that were recorded at the time) are given in the table. What signs would you look for and what tests would you carry out to decide on your diagnosis?

Arthur Cook Age 53 Married, 1 son Occupation: general deputy stores Manager, 1 son	Medical record new to list Chest X-ray BP 125/90 NAD
Date 1975	Cough about three days NAD
1977	"flu" lost 10 lb No breath Rx Berlyn
14 December 1979	visit cough 10 fever 38.5 yellow phlegm Rx Amoxi 250 mg i.d.s. 7 days
26 March 1980	much better chest X-ray final certificate
3 April 1980	c/o cold congestion no appetite Rx Aspirin
20 November 1980	c/o cold congestion no appetite Rx Aspirin

C/o = complaining of. NAD = no abnormality detected. Rx = treatment. i.d.s. = three times a day.

Examination

When examining Mr Cook, as well as looking at his fingers for clubbing—a feature of other lung diseases including bronchiectasis

Fig 2—Section of a bronchus after five days of exposure to rhinovirus. Fig 3—Section of a bronchus after 10 days shows that the mucous membrane is very thin and the cilia have nearly disappeared.