Practice formularies

Towards more rational prescribing

Most prescribing is done in general practice, where patient care is usually shared among different doctors. There are wide variations in prescribing patterns both within and among practices¹ and an ever increasing number of drugs available to choose from. The need to encourage rational, cost effective prescribing is well recognised²⁻³; the challenge now is to find ways to achieve this goal.

The idea of practice specific formularies is not new,⁴ and several practices have developed them and evaluated their use.⁵⁻⁹ The objectives of such formularies are to help doctors select cost effective and acceptable drugs for patients presenting with the most common illnesses seen in general practice and to encourage generic prescribing. Specialist drugs, prescribed by hospital colleagues, and new drugs, not shown to be better than cheaper alternatives, would be excluded.

Those who have developed practice formularies emphasise the educational value to all concerned. Doctors have found it useful to discuss the criteria and selection of drugs with others, including community pharmacists and clinical pharmacologists.¹⁰ This ensures that the most useful and cost effective drugs in each category are included. Good practice formularies have now been developed by the Northern Ireland and South East Scotland faculties of the Royal College of General Practitioners (available from Dr P Reilly, Dunluce Health Centre, Belfast BT9 7HR). These were designed for easy adaptation, and clear guidelines are available on how to do this to meet individual practice requirements (Royal College of General Practitioners, in press).

For a practice formulary to work all partners in the practice need to be committed to and active in its development. Initially, formularies are of greatest value in helping doctors choose drugs for treating acute illnesses. Changing long term drugs is more difficult, but this can be done by discussion with and education of individual patients.

Regular feedback is needed if participation and continuous commitment are to be sustained. Detailed information is now regularly available and includes data on costs, type of drugs used, and the proportion of generic prescribing by each doctor. The effects of introducing a practice formulary on prescribing patterns can be readily assessed. There are other

advantages, however, which are not so easy to measure. The example of the formulary can show trainees how partners may work together to reach agreed policies. Another advantage is to reduce the influence of pharmaceutical representatives and advertising, which are such powerful determinants of drug selection. Formularies must be regularly updated, a task that should be delegated to a specific doctor who will then discuss the proposed changes with the other partners.

By themselves formularies containing limited lists will not lead to more rational treatment. They should incorporate a practice consensus on indications for starting and stopping certain treatments—for example, antihypertensives, antibiotics, digoxin, oral hypoglycaemics, antidepressants, and benzodiazepines. Guidelines should also be provided on possible interactions and which side effects should be discussed with patients.

Such a formulary would provide an up to date rationale for prescribing in general practice. Although reducing drug costs is the main objective, other benefits include sharing ideas and developing joint prescribing policies. These might even lead to shared policies with local hospitals, many of which have their own internal formularies—a development that might affect the pattern of drug prescribing throughout the United Kingdom.

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Foreign bodies stuck in the rectum

The last word

If a doctor, usually a young surgical trainee, encounters a foreign body impacted in the rectum rarely can he or she resist the temptation to share the story with colleagues. Doctors encountering two such hapless patients in quick succession feel an uncontrollable desire to report on them in the medical journals, the ambitious ones embellishing the report with a review of the literature. I suspect that editors hoping to provide a little light relief are inclined to give such reports higher priority than they deserve. No one can claim that much good is done by these publications other than possibly bolstering the trainee's curriculum vitae and providing a talking point for future interviews.

If these reports do little to help the unfortunate patient or enlighten the medical profession then perhaps there should be a moratorium on such publications. Perhaps some prize should be awarded to those who have encountered many such cases and resisted the temptation to publish. I can claim only four, but I am sure that this is not a record.

Among the latest to succumb to temptation are Busch and Starling from Wisconsin.¹ They report on two men who had inserted a foreign body into the rectum for sexual gratification. One of them admitted to having performed the same act many times and called for medical help only when he could not remove it. What is perhaps indicative of the attitude of the

medical profession towards such acts is that these patients were offered psychological counselling. They refused, I expect to no one's surprise. There must be a few patients who have inserted foreign bodies unintentionally or by accident—they are the more unfortunate victims because no one will ever believe their story.

Presumably foreign bodies that become stuck in the rectum represent what might be aptly described as the "tip of an iceberg." I can only conjecture that many foreign bodies are inserted and removed from rectums for various reasons, mostly sensual. No physical harm is done so they never reach medical attention.

The doctors from Wisconsin embellish their report with a detailed analysis of some 700 identifiable objects that have been removed from the rectums of about 200 patients. They have analysed them according to criteria such as animal, vegetable, and mineral; homicide, leisure, or pleasure; and so

on. Their list includes plantain (with condom), an ice pick, and a frozen pig's tail: shouldn't we be prepared to accept it as the last word on the subject?

Many doctors seem to have become more open minded in their attitudes towards the vagaries of human behaviour since the emergence of AIDS. Surely we should not be telling patients not to do things that we know they will continue to do but telling them how to do them safely. Should objects such as dildos carry government health warnings?

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Faint heart

Opioids have a role in circulatory collapse due to acute blood loss

During the second world war researchers on both sides of the Atlantic studied the circulatory responses to acute blood loss in human volunteers with the new technique of right atrial catheterisation to measure cardiac output.^{1,2} They observed two phases in the haemodynamic response to venesection. In the first, arterial blood pressure fell very little as peripheral resistance rose progressively to counteract the fall in cardiac output. This compensatory haemodynamic response may also be seen when haemorrhage is simulated by foot down tilting or applying negative pressure to the lower half of the body.^{3,4} It is associated with an increase in sympathetic vasoconstrictor drive as measured directly by microneurography⁵ or indirectly by an increase in plasma noradrenaline concentration.³ All these observations are consistent with the notion that arterial blood pressure is maintained during acute hypovolaemia by a reflex response to the unloading of arterial baroreceptors and cardiopulmonary receptors.⁶

These studies found that a second phase supervened when blood loss, either true or simulated, exceeded about a quarter of the blood volume. At the beginning of the second phase the heart rate, peripheral resistance, and arterial pressure fall profoundly, and the subject usually faints. Sympathetic vasoconstrictor drive stops abruptly.^{7,8} The cardiovascular reflexes that maintained constant blood pressure despite the fall in cardiac output seem suddenly to have been switched off. At the same time another vasoconstrictor mechanism is switched on: there is a massive outpouring of antidiuretic hormone into the bloodstream.^{3,9}

Why this sudden and life threatening failure of cardiovascular homeostasis? Only recently have some answers to this question been found, with studies in unanaesthetised rabbits and dogs, which exhibit a biphasic response to haemorrhage similar to that occurring in human volunteers.¹⁰⁻¹⁴ Barcroft *et al* suggested that the signal for circulatory decompensation might come from the heart,¹ and it was later shown that sensory receptors in the left ventricle fire off during an acute reduction in central blood volume.¹⁴ Blocking the cardiac nerves in conscious rabbits prevents the second phase of the response to haemorrhage,^{15,16} though the influence of cardiac receptors is not as apparent in conscious dogs.^{13,17}

How does this putative signal from the heart obtund the compensatory baroreflexes? Good evidence now exists that it does so through endogenous opioids located in the brain rather than peripherally¹⁶ and governed by opiate receptors of the δ subtype.¹⁸ The signal for releasing antidiuretic hormone also comes from cardiac receptors,¹⁹ probably located in the ventricles,¹⁷ though this signal travels to the hypothalamus through pathways that do not interact directly with the arterial baroreflex mechanism.²⁰

This new work provides a much better understanding of what happens to conscious humans or animals during acute blood loss. In conscious animals blood pressure falls during acute haemorrhage not because the cardiovascular reflexes can no longer cope but because they are switched off. Do we have any evidence of this occurring clinically? There is a dearth of systematic observations bearing on this question, but patients hypotensive from acute blood loss have a relative bradycardia, and transfusion not only restores blood pressure but also raises the heart rate.²¹ This suggests that once central blood volume is restored to a critical level the cardiovascular reflexes are switched on again.

Do opiate antagonists have a therapeutic role in acute blood loss? In one study naloxone in a dose of 0.1 mg/kg failed to prevent circulatory decompensation during simulated hypovolaemia in volunteers.²² This negative result is not surprising—the dose of naloxone is 30-50 times less than that effective in animals. It would be wrong, however, to contemplate giving naloxone to humans in doses of 3-5 mg/kg. Not only would this be dangerous but the outcome would have no clinical application because the powerful affinity of naloxone for opiate receptors of the μ subtype would render narcotic analgesics ineffective. If opioid antagonism is to have any place in the treatment of acute blood loss—for instance, as a form of first aid when the blood volumes cannot be restored immediately—it may be through giving selective δ receptor antagonists.

Until recently the only δ selective antagonists available were peptides that do not cross the blood-brain barrier, but at the conference on international narcotics research in 1988 Portuguese *et al* announced the development of a non-peptide δ receptor antagonist, naltrindole.²³ This may be a useful