clear on the request form. Knowing that treatment is indicated, the laboratory should provide information on antibacterial sensitivities as soon as possible.

Good communication between microbiology laboratory staff and general practitioners is essential if the correct balance between advantage and disadvantage of the use of antibacterial agents in the treatment of urinary tract infection is to be achieved.

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# Do therapeutic interventions stand the test of time?

MANY therapeutic interventions demonstrate a pendulum of usage. After 30 years, anticoagulant drugs are again popular for a variety of indications while the use of lignocaine, still controversial in treating acute myocardial infarction, appears to be on the wane. The vicissitudes of digitalis are evident in the pages of medical journals, the pros and cons punctuated by an occasional outburst of enthusiasm about its use for congestive cardiac failure.[1] Finally, in the hands of plastic surgeons, the leech is enjoying a modest comeback.[2]

An upswing in usage is easy to understand and may be the result of fresh information or a serendipitous discovery extending the scope of a drug or procedure. Beta blockers were employed for nearly a decade for angina and for supraventricular arrhythmias before they came into widespread use as a first line therapy for lowering blood pressure. More recent indications include migraine headache, thyroid disease and portal hypertension. Coronary bypass surgery was originally performed for intractable ischaemic pain that interfered to an intolerable extent with the patient's lifestyle. Today, it is often used for averting sudden death in people with extensive coronary heart disease who are only mildly symptomatic.

The downswing of the therapeutic pendulum is harder to comprehend. What is the reason for the apparent waning of efficacy over time, a phenomenon that might be called herd tachyphylaxis? First, there is the loss of placebo influence, a process of familiarity breeding contempt. This is reinforced by state-of-the-art knowledge imparted to doctors by visiting representatives for drug manufacturers who are always abreast of the failings of other companies' products. Thus, the enthusiasm with which the preparation is dispensed may lessen, and with it the power of suggestion.

Secondly, there is the process that could be referred to as dilution of indications which is the reverse of expansion of scope. Many people are now experiencing chest pain within a few years of having coronary bypass surgery.[3] As a result, the operation appears less successful than was originally hoped. However, our protocol for investigating patients who have sustained a myocardial infarction has grown ever more aggressive and some patients are referred to a surgeon when asymptomatic or with only mild symptoms. Thus, prevention of a further infarct has become the outcome measure rather than relief of chest pain, the recurrence of which may wrongly suggest failure. The same applies to propranolol, as its uses proliferate and the diagnoses for which it is prescribed become further removed from its best known pharmacological activity. Propranolol is prescribed for both angina pectoris and stage fright; if the drug is ultimately shown to be less effective for the latter than initially thought, its reputation regarding the former may be tarnished too.

Thirdly, there is the question of what we are treating and the extent to which the diagnosis has been refined. Nitrates are prescribed when the patient gives a history consistent with angina pectoris. A stress test or angiogram are not carried out since these can only support or diminish the likelihood that angina is present; the report of symptoms is the decisive factor.[4] In effect, the response to a nitrate may be looked upon as furthering the diagnosis. Similarly, if a patient presents with symptoms suggesting a peptic ulcer, a therapeutic trial is regarded as a cost effective approach, with gastroscopy reserved for those for whom drug treatment is ineffective.[5] In both cases, there is no gold standard for the diagnosis. A normal angiogram does not exclude spasm as a possible cause of angina and the correlation between gastroscopic findings and report of pain in patients with a peptic ulcer is known to be poor.[6] Thus, a precise diagnosis is virtually impossible and there always exists the possibility that a patient with chest pain who responds to nitrates or one whose dyspepsia is alleviated by $H_2$-receptor blockers may have another condition which these drugs may improve.

Urinary tract infection is a self-limiting disorder, and thus treatment with antibiotics must be considered palliative. A respected textbook of primary care states that antibiotics may

be prescribed for dysuria of acute onset, and that urinalysis and culture should be performed 'when indicated'.[7] There is little doubt that cystitis is often a viral disorder and it may sometimes be induced by drugs. When these aetiologies are grouped together with bacterial infection in a single algorithm, the apparent outcome is good since the condition usually clears within a few days whether or not it is treated.[8] However, should the time come when we can be certain of the causal agent, the question of resistance to antibiotics will arise and preparations such as amoxycillin or cotrimoxazole will appear less effective. Lastly, a drug's best known mechanism of action may not be apposite to any particular result. For example, the apparent analgesic effect of amitriptyline may really derive from the production of basically undesirable pharmacologic results distracting patients from their misery.

Fourthly, the putative waning of efficacy can only be measured if there exists a clearly defined, preferably quantifiable, therapeutic objective. This brings us to outcome evaluation and whose estimate to accept, the doctor's or the patient's. The success of treatment is determined in different ways by the doctor and the patient, with the patient more likely to use psychosocial criteria such as the effect of the intervention on lifestyle, work, sex, sleeping habits and the family.[9] For the doctor, the matter is complicated by there being both a 'what' and a 'when' to outcome. For example, when treating otitis media, it would not be unreasonable to demand defervescence and the disappearance of pain as standards for success. These, in turn, might lead to children returning sooner to school allowing their parents to return to work. On the other hand, if the criterion adopted is the absence of a middle ear effusion two months after the acute episode, then the mode of treatment may make no difference.[10]

Finally, Balint maintained that the physician is a pharmacological agent and, as such, he or she can have synergistic or antagonistic interactions with other therapeutic modalities.[11] The influence of the doctor–patient interaction has been convincingly demonstrated in studies of headache,[12] duodenal ulcer[13] and even streptococcal pharyngitis.[14]

To summarize, the decline in the effectiveness of therapeutic interventions may operate by several mechanisms. These include a waning of the placebo effect, dilution of indications, difficulties in evaluating outcome and the fact that our diagnostic skills usually lag behind our knowledge of therapeutics.

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