The future of cancer chemotherapy lies in increasing the specificity of treatment and diminishing its toxicity. The lack of specific treatment for many cancers emphasises the importance of the clinical trial in assessing treatment: this will jettison irrelevant or harmful measures, preserving only those of proved value. We must now put similar efforts into identifying those factors, including antiemetics, that can best reduce the one consequence of treatment most often raising doubts in the patient's mind about whether the cure is really worth the discomfort of treatment, however temporary this may be.

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Tardive dyskinesia

If anyone doubts the continuing truth of Voltaire's claim that doctors pour drugs of which they know little into patients of whom they know even less, the phenomenon of tardive dyskinesia should convince them. Though it was first reported[1] in 1957, for many years psychiatrists were reluctant to accept that tardive dyskinesia was a side effect—all too often an enduring one—of the neuroleptic drugs[2 3] that are their principal weapons against schizophrenia. The syndrome is chiefly characterised by repetitive pouting of the lips and protrusion of the tongue, often accompanied by bizarre facial grimacing and sometimes more widespread choreiform movements. Gibson[4] found that in chronic schizophrenic patients receiving depot neuroleptics the syndrome appeared with steadily increasing frequency and had affected about a quarter of them after three years. Nor is it only schizophrenic patients who may be affected. Paulson[2] reported 14 sufferers who were neither institutionalised nor psychotic but who had all received neuroleptics, some of them for many years.

When many different treatments are recommended for a condition usually none of them is particularly effective. This is certainly the case with tardive dyskinesia. For example, though a recent well-controlled trial[5] showed that muscimol (a structural analogue of γ-aminobutyric acid, GABA) improved symptoms in tardive dyskinesia unfortunately it also caused an increase in psychotic symptoms.

Because treatment is so unsatisfactory attempts have been made to detect patients who may be especially likely to develop tardive dyskinesia. Those with brain damage and the elderly (not necessarily the same people) are known to be vulnerable. Wegner *et al*[6] have recently reported the presence of a characteristic pattern in the electroencephalogram in 95% of patients with tardive dyskinesia, whereas it is found in only 33% of controls; but whether this finding is a precursor or a result of tardive dyskinesia is not yet known.

In our present state of knowledge, the best hope of prevention of tardive dyskinesia remains care and discrimination in the use of neuroleptics. Not all schizophrenic patients require medication, and in those who do short "drug holidays" may reduce the likelihood of tardive dyskinesia—though there is no proof as yet. Most reports suggest that routine anticholinergic medication increases the risk of tardive dyskinesia, though Gibson[4] disagrees. Clearly, neuroleptics should be prescribed for patients with conditions other than schizophrenia only after most careful consideration of the alternatives, especially where long-term use is likely. Early diagnosis of tardive dyskinesia may be important in arresting its progress, but it may also be difficult in a condition which, perhaps surprisingly, often seems to trouble the patients less than it troubles their physicians.[2] In established cases, a gradual reduction or withdrawal of neuroleptic drugs may be helpful, though this sometimes leads to an initial worsening of the dyskinesia. Such a policy may not be practicable if it results in a recurrence of the underlying psychiatric disorder. Paradoxically, *increasing* the dosage may improve matters in some cases. Changing to a different neuroleptic may be worth a try. If that fails, all that remains is treatment with one of the many drugs that have been claimed to relieve symptoms; these include deanol, diazepam, baclofen, alphamethyltyrosine, tetrabenazine, and reserpine.

In the long term the answer must lie in the development of a new class of neuroleptic drugs that will control schizophrenia without producing tardive dyskinesia. A report by Shopsin *et al*[7] indicates that clozapine—a dibenzazepine with some important pharmacological differences from most of the standard neuroleptics—may be such a compound, but psychiatry has seen so many false dawns that it would be premature to cheer. The ghost of Voltaire is there to remind us that, even if further trials confirm the safety of clozapine in respect of tardive dyskinesia, it may turn out to produce other, as yet unknown side effects in long-term use.

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[3] *Lancet*, 1979, **2**, 447.
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Systemic sclerosis in old age

Very elderly patients are referred for specialist opinions relatively rarely. Because of this bias, many diseases have been mistakenly thought to be rare in old age. In systemic sclerosis early reports suggested that it was predominantly a disease of middle life and a comparative rarity in old age.[1–3] Before too long, however, the highest incidence was reported[4] to be in patients aged over 65. This revised view was confirmed in a personal series of 15 cases seen by one physician in geriatric medicine in 11 years[5]—representing an incidence of about one per 1000 elderly patients admitted under his care. A recent paper from a department of geriatric medicine[6] has now drawn attention to the ease with which the diagnosis of systemic sclerosis may be overlooked in the elderly because of the minor nature of the skin changes.

What, then, is the clinical picture of systemic sclerosis in old age reported in these two papers? Most strikingly, all the patients were women. Their average age was 80, closely corresponding to the overall average for women admitted to a geriatric department. All had Raynaud's phenomenon affecting the hands, and it was this symptom—otherwise rare in old age—which commonly drew attention to the disease. All the patients had skin changes in their hands, but these were most often not striking. Nevertheless, more than half had evidence of old or recent skin ulceration, whitlows, or necrosis of the pulp.

Changes in the skin were found more often in the face than in the feet: these were mainly subtle changes—minor skin tethering, recognised only after the diagnosis had been suggested by other findings—but in a few patients the facial changes were more severe and gave rise to a characteristic reduction of mouth gape with radial furrowing. Telangiectasia was another useful diagnostic pointer; this was found in over two-thirds of the patients, the small punctate telangiectases most commonly occurring around the nails and on the lips and central face.

Investigations showed that nine of the 17 patients had evidence of oesophageal lesions, five had positive serological tests for rheumatoid factor (latex or sheep cell agglutination), and three had subcutaneous calcinosis. A single patient had pulmonary fibrosis. No other instances of systemic disease were recognised.

Systemic sclerosis in old age seems, therefore, to be quite common, but most patients have benign forms of the disease, quite often corresponding to the so-called CRST syndrome. In this mild variant calcinosis, Raynaud's phenomenon, sclerodactyly, and telangiectasia are the dominant features and the oesophagus is the only site of visceral disease. Certainly, the reported patients followed a generally favourable course and had had evidence of the disease for up to 20 years. In many patients systemic sclerosis was a more-or-less incidental finding, though some did have troublesome symptoms such as frequent Raynaud's phenomenon, pain from ulcers or whitlows, or dysphagia. There were no deaths attributable to the disease. Since the prognosis is favourable attempts to treat the disease with steroids or anti-inflammatory or antifibrotic drugs are generally contraindicated.

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The improving image of A and E

Accident and emergency services have come a long way since the Platt Report was published in 1962.[1] Nevertheless, problems remain and in many, as our special correspondent points out in the last article of the series (p 1348), the calibre of the staff and especially of the consultants in charge of the departments is crucial. It is central to the problem of "deliberately striving to cultivate a true humanity," as Rutherford *et al*[2] put it in their new textbook, in conditions often conducive to "the practice of a brand of supermarket medicine"; to the supervision and teaching that should be so important a part of the work; and to the problem of preventing abuses of the department by the hospital as well as the public. The status and authority of the consultant is also vital in competition for staff and facilities.

The creation of consultant posts in accident and emergency from 1972 has improved the status of the work and helped to upgrade many departments. Health authorities and now even hospitals themselves—despite long traditions of vested interest in casualty on the part of some specialties—have begun to ask for accident and emergency consultants. The standing of these consultants in the hospital is usually good, their right to clinical responsibility is generally accepted, and many of the former disincentives to a career in the department have disappeared, especially now that a training programme[3] has become established.

Can accident and emergency medicine, however, establish itself as an attractive enough specialty to draw in the best talents? We cannot yet expect it often to be a first career choice; but its prospects surely are at least as good as those of, say, anaesthesia and radiology in their infancy. Though accident and emergency work is intrinsically episodic its image has changed from that of a mere clearing house to a specialty in immediate management—with the bonus that its practitioners never know what to expect next.

Enthusiastic teaching should fire more medical students to specialise in accident and emergency. Knowledge of this work is important for most branches of medicine, and the range of conditions offers exceptional opportunities for teaching. Indeed, in a survey of doctors' opinions about priorities in the medical curriculum "casualty" came out fourth.[4] Students appear to value the time they spend there, but many would like it to be longer and for there to be more formal teaching. A few medical schools give sadly inadequate time to the accident and emergency department but substantial teaching depends on adequate staffing as much as on enthusiasm.

Undergraduate teaching needs, then, to be strengthened—and more money is also needed for senior registrar training posts. Another essential is that departments should not be weakened by the presence of a lot of outlying or neighbouring units. Accident and emergency work cannot be either effective as a service or attractive as a career without a case load large enough to justify plentiful staff and facilities and to provide job satisfaction. Advances in the treatment of seriously injured or acutely ill patients—and, incidentally, the increased litigation hazards of medical practice—merit the concentration of services, yet despite concentration in the past 15 years some units are still not large enough. While local pressures to keep open unnecessary casualty departments should be resisted health authorities need to be sensitive to local circumstances. Nevertheless, most areas would benefit from improved local arrangements for dealing with emergencies, by general practitioners and in other ways (this has been said repeatedly but improvement is slow to come), and where necessary immediate care schemes. Above all, the public and the profession should understand more clearly what accident and emergency departments are for.

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