Half as many radiologists again by 1995

In 1979 a *BMJ* leading article stated that "diagnostic imaging must improve its image, attract more candidates, and . . . ensure the best clinical value is obtained from a specialty whose capacity will never be sufficient to meet demand."¹ Seven years on the Royal College of Radiologists can be pleased to have seen the first two objectives achieved and to have made much progress with the third.

Yet manpower problems remain. Demand continues to exceed capacity, and progress in technology has accentuated the discrepancy. The past decade has seen the wide application of sonar scanning and the proliferation of scintiscanning and computed tomography. Now interventional and therapeutic radiology—when one radiologist may spend a whole session with one patient—are developing rapidly, and the future promises more of the same with magnetic resonance imaging, Doppler sonar, and mammography screening.

The college is concerned primarily with professional standards and knows that if additional work is absorbed by a static work force then standards will fall. It has produced a report detailing and quantifying the work that a radiologist may be expected to undertake.² Taking a look into the future and casting an envious glance abroad, it has estimated a need to increase the numbers of consultants by half by 1995.

Professional work is notoriously difficult to quantify. The number of attendances of patients for radiology makes no allowance for multiple examinations. Total examinations are easy to count (and administrators and authorities understand the concept) but make no allowance for the wide variation in radiologist effort needed (though the case mix is fairly steady and does allow some wide comparisons). For many years work in radiology departments has been counted in radiographic work units based on the time a patient is in the examination room, while the college has had a points system based on the radiologists' time required for each examination.

Next year sees the implementation in radiology of the Körner report.³ The college has modified its workload measurement system and allied it to the Körner examination groups, weighting each group according to the radiologists' involvement. This is not always satisfactory—for instance, obstetric sonar does not often keep the consultant radiologist busy for 10 minutes—and no account is taken of films not reported for whatever reason. Expansion of radiology is unlikely to result from this college initiative alone. When demand exceeds capacity the only practical solution, if standards are to be maintained, is to make less urgent problems wait.⁴ If the wait is unacceptable to other doctors and health authorities then they may choose to put more resources into radiology. Meanwhile, radiologists must see that limited resources are used to the best advantages for the community while continuing to encourage the introduction of the best of new technologies and techniques.

The work of the college,^{5,7} the World Health Organisation,⁸ and individual radiologists⁹ must also encourage elimination of relatively unnecessary radiology. Then we may be able to offer new radiological procedures without detriment to the many patients whose diagnosis is still made from simple radiographs¹⁰—even if the college manpower target is not met.

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Urinary tract infection in the elderly

Bacteriuria (10^5 bacteria in 1 ml of urine) is common in the elderly, but how much it matters and whether it should be treated are unclear. Its importance in the elderly may be quite different from that in the young: the association with dysuria and frequency is poor^{1,3}; fever is often absent; and there are no pus cells in a third of specimens from elderly patients with bacteriuria.⁴ Using significant bacteriuria as the main criterion for urinary tract infection thus has limitations.

"Significant bacteriuria" is a statistical concept that was introduced by Kass⁵ to distinguish contamination of urine specimens during collection from the presence of bacteria in bladder urine. It applies to those who have no urinary symptoms and from whose urine *Escherichia coli* or *Proteus* is isolated in pure growth and may not be applicable in those with symptoms or infection with other organisms.

Community studies show that about a fifth of elderly women and 10% of elderly men have significant bacteriuria, and the prevalence probably increases with age in both sexes.^{1,2,6} Lowest prevalences are found in the elderly living independently and the highest in patients in long stay geriatric wards.⁷

Bacteriuria in the elderly does seem to be related to mortality,⁸⁻¹⁰ but it may be a marker for other diseases that are the primary causes of increased mortality rather than itself being the direct cause. A recent study that controlled for age and coexisting disease seemed to confirm this epiphenomenal relation.¹⁰

It has often been assumed that bacteriuria in the elderly, particularly in women, represents bladder rather than upper urinary tract infection, and is hence benign. One study, however, showed that over half of urinary infections in elderly women in hospital may be in the upper urinary tract.¹¹ Diminished glomerular filtration rate and renal plasma flow have been reported in elderly patients with bacteriuria,¹² but whether the bacteriuria is a primary cause of renal damage or secondary to it is unknown.

It is thus difficult to draw practical conclusions from available research on how best to treat old people with bacteriuria. Examination for bacteriuria is usually undertaken in an elderly patient because she has one of Isaac's "giants of geriatrics"—immobility, instability, incontinence, and intellectual deterioration.¹³ The detection of bacteriuria then leads almost inevitably to treating the "infection" because it may be causing the atypically presenting disease. In geriatric wards about one third of all courses of anti-

microbial chemotherapy are given for urinary tract infections, and bacterial resistance to broad spectrum antimicrobial agents is common in those wards.¹⁴ Clarification of the role of bacteriuria in the atypical presentation of disease in the elderly might thus allow fewer patients to be treated with antibiotics and so reduce bacterial resistance in wards for the elderly.

The effect of treating all asymptomatic elderly individuals with bacteriuria has not been examined; a very large multicentre study would be required to eliminate confounding variables. The elderly with bacteriuria are a heterogeneous population, and better understanding of underlying causes of urinary infection could result in the identification of subgroups of infected individuals who would benefit from prophylactic treatment.

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Campylobacter pyloridis: what role in gastritis and peptic ulcer?

Much excitement has recently been generated by the possibility that *Campylobacter pyloridis* might be the cause of gastritis and peptic ulcer.^{1,2} This spiral organism colonises the gastric antrum in 90-100% of patients with duodenal ulcer, over 70% of patients with gastric ulcer, and half of patients with non-ulcer dyspepsia.^{3,4} When present, it is invariably associated with histological evidence of gastritis.⁵

Spiral organisms were seen in the stomach of animals before the turn of the century,⁶ were reported in man in 1906,⁷ and raised further interest in 1940,⁸ but their possible clinical importance was not recognised until recently.⁹⁻¹¹ *C. pyloridis*,¹² a microaerophilic spiral bacterium, is like many other campylobacters and shows some antigenic cross reactivity,² but it differs from others metabolically, morphologically, and in its chemical structure.¹

The organisms are found on the surface of gastric epithelial cells even when acidity is normal—they are protected from acid by the overlying mucus layer and the alkaline secretion

of the epithelial cells.¹³ They are mobile in highly viscous material¹⁴ and appear to be specially adapted to the mucus layer above these particular cells as they are not found in areas of intestinal metaplasia within the stomach, and when identified in the duodenum are found only in areas of gastric metaplasia.¹⁰ Ultrastructural studies show adherence pedicles at points of membranous adhesion^{1,15}; microvilli are absent or depleted, and intracellular oedema is prominent. These appearances are reminiscent of intestinal epithelial cells infected by enteropathogenic *Escherichia coli*.

Is *C. pyloridis* pathogenic? Data from many different centres have confirmed the association with active gastritis,^{1,2} but the organisms are less often found in gastritis secondary to duodenogastric reflux¹⁶ and pernicious anaemia.¹⁷ These data, together with the ultrastructural appearances, strongly support a primary aetiological role. Further support comes from an experiment carried out by a normal volunteer who developed an acute self-limiting dyspeptic syndrome with acute gastritis after swallowing the bacteria.¹⁸ Other evidence links *C. pyloridis* infection with an epidemic of hypochlorhydric gastritis in 17 of 37 volunteers who underwent acid secretion studies with a pH electrode that had not been sterilised between experiments.^{1,19} Eradication of *C. pyloridis* with antibiotics or bismuth leads to histological improvement⁴ but relapse is common. Serum IgA and IgG titres to *C. pyloridis* are appreciably raised in patients with campylobacter,^{20,21} and local gastric juice antibodies are also present in some of these.²²

The clinical relevance of the strong association between gastritis and *C. pyloridis* is, however, controversial as gastritis occurs in many of the normal population²³ and campylobacter associated gastritis has been reported in a quarter of normal volunteers. More interesting is its relation with peptic ulcer.^{4,10} Numerous studies have confirmed that campylobacter associated gastritis is present in virtually all non-iatrogenic cases of duodenal ulcer. *C. pyloridis* may infect the islands of metaplastic gastric epithelium found in the duodenum, thereby weakening the mechanisms which normally protect these cells from acid attack, which leads to a breakdown in mucosal integrity and ulceration.²⁴ A similar sequence in the stomach leads to gastric ulceration. The evidence for this hypothesis remains circumstantial, but metronidazole, to which *C. pyloridis* is sensitive, has had a beneficial effect in both gastric and duodenal ulceration.²⁵ Further, the lower recurrence rate when duodenal ulcers are healed with colloidal bismuth subcitrate rather than acid reducing drugs may be related to the effect of bismuth on *C. pyloridis*.²⁶

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