One solution to the problem of competing demands involves separating resources (for example, designated operating time for thoracic surgery) and enhancing the specialty of thoracic—as opposed to cardiothoracic surgery. Relying on cardiothoracic surgeons to do lung resections may also mean that some do too few to maintain competence and five year survival figures are better in those who operate more often.[4]

The need for specialist thoracic surgeons is emphasised by the fact that surgery for lung cancer represents less than half the workload of the 40 purely thoracic surgeons in the United Kingdom. Surgical management of pneumothoraces, empyema, mediastinal masses, and benign and malignant conditions of the oesophagus; lung biopsies; and lung volume reduction surgery all need expertise that justifies a specialist approach. Different skills and attributes are needed in thoracic as opposed to cardiac surgery.

Patients with respiratory disorders who need thoracic surgery, and the physicians who care for them, are grateful for the help they receive from hard pressed cardiothoracic surgeons. However, the time has now come to double the number of purely thoracic surgeons in the United Kingdom by welcoming more from overseas, retraining surgeons experienced in other surgical specialties, and enhancing the number and quality of training opportunities to entice the young into this specialty. The rest of us may be desperate for more colleagues, but the supply is limited. We should look at delivering care in different ways so that the limited number of doctors can be targeted at specialties such as thoracic surgery, where the need is desperate.

Martyn R Partridge *professor of respiratory medicine*

Imperial College of Science, Technology and Medicine, Charing Cross Campus, London W6 8RP

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Was it a heart attack?

Troponin positive acute coronary syndrome versus myocardial infarction

The World Health Organization's classic definition of acute myocardial infarction requires that at least two of the following three criteria are met: a history of typical symptoms of ischaemic chest discomfort; evolutionary electrocardiographic tracings involving the development of Q waves; and an increase in the creatinine kinase level greater than twice the upper reference limit.[1] While this definition is clear cut, many patients who have had myocardial infarction will be excluded. Recent developments in the detection of small quantities of myocardial necrosis using serum cardiac troponin levels have prompted a new definition of myocardial infarction. This may well lead to an increase in the number of patients diagnosed and improved patient outcomes, but it may also confuse clinicians, epidemiologists, and most importantly patients themselves.

Myocardial infarction without ST elevation

Acute coronary syndromes without accompanying elevation of the ST segment on the presenting standard 12 lead electrocardiogram cover a range from unstable angina to non-ST elevation or non-Q myocardial infarction. Patients in the latter category can go on to fulfil the WHO definition of myocardial infarction by developing Q waves on serial electrocardiographic tracings during their index admission. Specific patterns of ST depression are highly predictive of myocardial infarction. For example, ST depression of 4 mm or more in any lead except aVR is 97% specific for myocardial infarction, and isolated depression of 1 mm or more measured at 80 ms of the J point in six or more leads is 96.5% specific for myocardial infarction. The artery responsible for myocardial infarction is the circumflex in around 17% of patients. Less than half of these patients will show ST elevation on a standard 12 lead electrocardiogram and a third will show isolated ST depression.[2] Patients presenting with ST depression on the initial electrocardiogram who have a rise in creatinine kinase level twice the upper limit of normal are traditionally given a diagnosis of non-Q or non-ST elevation myocardial infarction.

Mortality in hospital is greater for patients who have a Q wave myocardial infarction, whereas rates of reinfarction, recurrent ischaemia, and long term mortality appear to be higher following non-Q myocardial infarction.[3 4] A large observational study in 1975-97 showed that mortality in hospital for patients with a diagnosis of Q wave myocardial infarction has declined from 24% to 14%, but mortality in hospital for non-Q myocardial infarction has remained the same at 12%.[4] Corresponding five year survival rates after Q wave and non-Q myocardial infarction were 75% and 65%, respectively. There was also an apparent increase in the incidence of non-Q myocardial infarction, possibly related to changes in management over time such as risk factor modification, reduction of prehospital delay, and improvement in access to and advances in medical care.[4] Targeted interventions improve the prognosis after non-ST elevation myocardial infarction, with most of the reported trials to date showing that a combination of IIb and IIIa receptor antagonism and early coronary revascularisation appears to be efficacious in terms of improving morbidity and mortality.[5–8]

Troponin positive *v* troponin negative acute coronary syndrome

The uncertainty around the electrocardiographic diagnosis of myocardial infarction has been further complicated following the development of sensitive and specific serological markers that enable the detection of very small amounts of myocardial necrosis. The prognostic significance of cardiac troponin levels is

BMJ 2002;324:377–8

firmly established and incremental.[9–11] Troponin I values of less than 0.4 ng/ml are associated with a 42 day mortality of 1% and this risk increases progressively to a mortality of 7.5% at values of 9.0 ng/ml or more. Troponin levels greater than the 99th percentile for a given reference control are defined as high. The assay is accurate, relatively straightforward, and can be done at the bedside. Patients presenting with cardiac chest pain and electrocardiographic changes who do not have a significant rise in creatinine kinase can therefore be further classified as having troponin positive or troponin negative acute coronary syndromes, with consequent prognostic and therapeutic implications. Troponin negative coronary syndromes can also be labelled as unstable angina.

Proposed redefinition of myocardial infarction

A recent consensus document from the European Society of Cardiology and the American College of Cardiology has offered a redefinition for acute, evolving, or recent myocardial infarction. This requires a typical rise and fall of biochemical markers of myocardial necrosis such as troponin or creatinine kinase (MB fraction) with at least one of the following: ischaemic symptoms; development of pathological Q waves; electrocardiographic changes indicative of ischaemia (ST segment elevation or depression); and coronary intervention.[12] As discussed by the authors of the consensus statement, this new definition has major implications for individual patients (psychological, life insurance, career, driving), research (inclusion criteria for clinical trials and trial endpoints), healthcare systems (epidemiology, admissions, procedures, rehabilitation), and society. It will hopefully stop the arbitrary use of terms such as "infarctlet" and "CK leak" and identify more patients who need aggressive secondary prevention. The latter, along with safe early discharge of patients who do not meet the new criteria, may reduce costs of healthcare.

If this new definition is widely adopted, as is likely, there will be a dramatic (but spurious) increase in the incidence of myocardial infarction across countries where troponin estimation is the new standard biochemical marker for myocardial necrosis. Physicians and epidemiologists may be able to accommodate such changes through recognising the value of more precise risk stratification, more targeted patient management, and better care. For the patient, however, the only accurate answer to the ubiquitous question—was it a heart attack doctor?—will be yes. Qualification of this response, in terms of newer more sensitive methods of diagnosis and little residual cardiac damage, may well be ignored.

Charles J McKenna *international fellow*
J Colin Forfar *consultant cardiologist*

Department of Cardiology, John Radcliffe Hospital, Oxford OX3 9DU

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Regulating the regulators

The "overarching council" does not have an overarching vision

The British government has plans for a new council to oversee the eight statutory regulators of health professionals (see box). The plans went out for consultation in the summer[1] and are included in a bill currently before parliament.[2] Proposals for the new overarching council were mooted in the NHS plan and backed by the recent Kennedy report into children's heart surgery at the Bristol Royal Infirmary.[3 4]

Under the proposals the new Council for the Regulation of Health Care Professionals will "build and manage" a coordinated and consistent framework for regulation across health professions. It will put patients first and ensure open, transparent, and consistent procedures within each regulatory body. The council will comprise representatives of the public, the professional regulatory bodies, and government appointees (to be in a majority of one) including members of the public, and health service managers. It will be led by a chairperson appointed, for the first term, by the secretary of state. The council will be able to require regulators to change their procedures; refer their decisions on individual cases to the high court when it judges such an appeal to be in the public interest; and investigate claims of maladministration.

BMJ 2002;324:378–9