

## Cardiac surgery audit raises concern over equity of access

Richard Woodman *London*

Concern over equity of access to cardiac surgery has been raised following the first comprehensive audit ever carried out in the United Kingdom by the Society of Cardiothoracic Surgeons. Data provided by nearly three quarters of units undertaking adult cardiac surgery show that the "urgent" operation rate varied from 10% to 60% between units.

Twenty per cent of patients undergoing surgery in some units were over 70 years old compared with only 10% in others. In one unit 30% of the patients had a good ejection fraction, in another, 80%. Similarly, the proportion of patients presenting for surgery who had severe angina was 40% in one unit, compared with less than 10% elsewhere.

The report says that the reasons for these variations are unclear but may in part represent differences in treatment strategies, subjective interpretations, or, of more concern, equity of access to surgery.

The society's secretary, Mr Bruce Keogh, consultant cardiothoracic surgeon at Queen Elizabeth Hospital, Birmingham, said that future reports would explore to what extent the variations were linked to differences in units' resources and treatment strategies.

A steady upward trend in the volume and complexity of car-

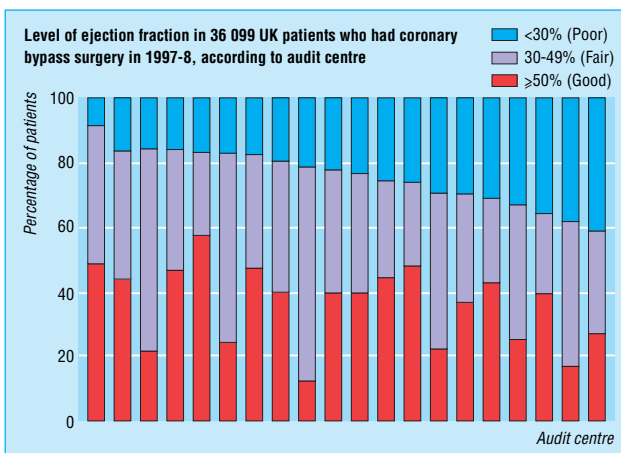
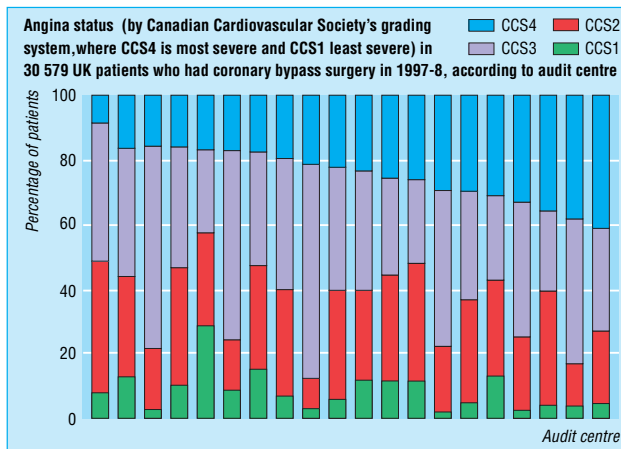
diac surgical workload is identified by the report. The number of coronary artery bypass operations rose more than ninefold, from 3040 in 1977 to 28 499 in 1997, whereas operative mortality remained relatively constant at around 2-3% despite increasing risk.

The data highlight the influence of age, sex, operative priority, and repeat surgery on operative mortality. They also show that it is possible to predict operative mortality and to stratify for case mix, and that this stratification can be extended from simply predicting mortality to also predicting resource consumption within the NHS.

Mr Keogh said that it was particularly reassuring that the data showed that UK cardiac surgery compared favourably with the United States and seemed to have equivalent outcomes.

The society's president, Julian Dussek, said: "Doctors are frequently criticised for being secretive about their results, and cardiac surgeons in particular are very much in the public spotlight at present... This report represents a huge step forward, not only in collecting risk stratified data from 70% of units in the United Kingdom but in being prepared to publish the information for all to see."

Mr Barry Jackson, president



of the Royal College of Surgeons of England, said: "The college welcomes this important report as a major step in the continuing process of raising surgical standards and improving patient care." □

*National Audit of Cardiac Surgical*

*Database Report 1998* is available at a charge of £25, including postage (payable to the Society of Cardiothoracic Surgeons of Great Britain and Ireland), from Bruce Keogh, Department of Cardiothoracic Surgery, Queen Elizabeth Hospital, Birmingham B15 2TH (tel: 0121 627 2533 fax: 0121 627 2545).

## Wimps can blame their genes

Deborah Josefson *San Francisco*

Anecdotal evidence has shown that people vary greatly in their sensitivity to pain and in their response to analgesics. Now, new research indicates that there is a genetic basis for differences in pain perception. The findings may lead to a more tailored approach towards determining doses of analgesic and to a better understanding of opiate addiction.

Working with mouse models,

scientists from Johns Hopkins University in Baltimore and the National Institute of Drug Abuse in Baltimore identified a candidate gene involved in the regulation of nociception (pain perception) (*Proceedings of the National Academy of Sciences USA* 1999;96:7752-5). The gene encodes the mu opiate receptor, the primary target of morphine and other opiates.

The researchers, led by Dr George Uhl of Johns Hopkins University, found that pain perception in mice, as measured by length of time for a response to temperature and pressure stimuli, varied inversely in relation to the density that mu opiate receptors displayed—the more opiate receptors available, the smaller

the response to the stimulus.

Mice that lacked mu opiate receptors had a lower threshold for reacting to pain: they responded to stimuli that were 66% of the strength of the stimulus required to elicit a response in mice with normal densities of mu opiate receptors. For mice with half the normal number of receptors, pain reactions occurred when the strength of stimulus was about 80%.

Using eight different strains of mice, through genomic analysis the researchers then traced the differences in density of mu opiate receptors to polymorphisms in the regulatory regions of the mu opiate receptor genes.

Several candidate promoter DNA sequences were identified.

The promoter is a region of DNA lying upstream of the actual gene to which proteins bind that "promote" the transcription and ultimately the expression of the gene—in this case the opiate receptor. Sequence comparison of the gene encoding the mu opiate receptor showed that it was highly conserved between mice and men, suggesting that similar regulatory differences may account for differences in pain perception in humans.

Commenting on the work, Dr Uhl said: "People have long been sceptical that pain has a genetic basis... Many people assume the way people respond is voluntary. But now people can think of pain as a genetically regulated problem." □