Letters

Workload measurement in histopathology

I am surprised that both Suvarna and Kay's paper on workload measurement in histopathology and Furness' subsequent commentary do not refer to North American reports on this subject. A formal method of assessing physician workload and value has been developed in the USA from 1988 onwards and is now in widespread use for billing.

The basis of the system has been published in detail. The system uses a relative value unit (RVU) for comparing the "physician value" of different activities. The value includes: time taken; mental effort and judgement; technical skill and physical effort; and psychological stress. Relative values are assigned to a service by asking a sample of practitioners to rate the value of the service in relation to a reference service. It has been shown to be reproducible and valid. Relative value scales have been developed for pathology and are published in the Federal Register. The services and procedures are defined in the American Medical Association's Current procedural terminology.

As an illustration, using the limited published data on individual workload and making reasonable assumptions, an efficient whole time equivalent pathologist working 44 weeks a year, seven laboratory sessions a week (three sessions for meetings, audit, management and research) might "earn" about 4500 RVUs per annum. This equates to:

Either: 4286 FNAs
or: 10714 pathologist interpretations of cervical cytology
or: 1974 large resections, for example bowel, prostate, or lung for neoplasm
or: 6000 biopsy specimens of colon, stomach, or prostate
or: 20455 appendices or gallbladders
or: 34615 vas or fallopian tubes from sterilisations.

[Note: the unit of service is the specimen and extra credit is given for special stains.]

Accurate measurement of workload is important to determine departmental resources, for internal departmental allocation of work, and to assess the likely impact of service developments. It may become even more important in the near future when the manpower planning failures of the last few years lead to short staffed departments and gross discrepancies between individual workloads. This situation will doubtless lead to demands for redistribution of work and/or remuneration; these potential disputes can only be solved fairly if there is an accepted way of measuring workload.

The RVU system seems fair and should be easy to apply and to audit. It is consistent, has been validated, and is under regular review. There is every good reason to introduce it to the United Kingdom as a way of measuring workload in histology and cytopathology.

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5 Federal Register 1995;60 (December 8):63123.

Authors' response

We are grateful for the response from Dr Griffiths and his illustration of an alternate methodology of calculating histopathologists' workload. The resource based relative value is derived from an assessment of time and intensity of work with practice cost and opportunity cost factors (training, and so on). In broad terms it parallels the Kornor unit (KU) in attributing work to case/diagnosis and has been tried in some centres.

We chose to limit ourselves to commonly used UK units of measurement in our paper since these are used to justify funding and service provision plans. We consider we have illustrated the limitations of the Welcan, KU, and case load number assessment (as suggested by the Royal College of Pathologists). We feel there is merit in a simple system providing a more interactive assessment of histopathologists' activity and consider that the KU figures can be altered progressively as reporting standards change, to reflect the alteration in workload. Nevertheless, the RBRV methodology deserves comparison.

The limitations of the RBRV model include the arbitrary grouping of named codes together, lack of necropsy work assessment, and a specific time allocation to whole session activities (for example, research). However, if we use Dr Griffiths' examples of an "average" histopathologist we can see the ratios of work assessment, and lack of specific time and antibiotic intervention trials are therefore required to prove that C pneumoniae is clinically important. However, the concern is that some populations in the prevalence of chronic active C pneumoniae infection may be too low to enable unselective trials to show an effect where in fact one may exist. Furthermore, for reasons of appropriate prescribing alone, antibiotics should only be given to subjects in whom there is good reason to believe there is current C pneumoniae infection, especially if, as has been suggested, treatment may need to be for prolonged periods of up to a year. Future research should therefore include efforts to determine how infected individuals can be rapidly identified. We believe serology is inadequate. Specific tests of current infection, such as probing for C pneumoniae DNA in blood or monocytes need urgently to be validated.

Chlamydia pneumoniae and atherosclerosis

In their leader article, Taylor-Robinson and Thomas cogently discussed the association of C pneumoniae and atherosclerosis and examined whether C pneumoniae infection is specific for arteries and atheroma. We agree that current evidence suggests that it is not. In fact, C pneumoniae has been associated with several diseases, among which the only common factor seems to be the presence of diseased tissue (table). The authors also discussed how future studies should be designed to investigate its role in atherosclerosis. We agree that although pathological studies have demonstrated the presence of C pneumoniae in atheroecrotic vessels, they cannot show whether infectious organisms or follows its development. Animal studies and antibiotic intervention trials are therefore required to prove that C pneumoniae is clinically important. However, the concern is that some populations in the prevalence of chronic active C pneumoniae infection may be too low to enable unselective trials to show an effect where in fact one may exist. Furthermore, for reasons of appropriate prescribing alone, antibiotics should only be given to subjects in whom there is good reason to believe there is current C pneumoniae infection, especially if, as has been suggested, treatment may need to be for prolonged periods of up to a year. Future research should therefore include efforts to determine how infected individuals can be rapidly identified. We believe serology is inadequate. Specific tests of current infection, such as probing for C pneumoniae DNA in blood or monocytes need urgently to be validated.

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Some of the diseases associated with C pneumoniae

<table>
<thead>
<tr>
<th>Main age(s) of onset</th>
<th>Coronary artery disease</th>
<th>Asthma</th>
<th>Rheumatoid arthritis</th>
<th>Sarcoidosis</th>
<th>Alzheimer dementia</th>
</tr>
</thead>
<tbody>
<tr>
<td>UK prevalence</td>
<td>Rises steeply from 35 years onwards 3-4% (40-49 years) 6-7% (50-64 years)</td>
<td>Early (childhood) and late onset types recognised 5% 15% in second decade</td>
<td>30-40 years</td>
<td>20-30 years</td>
<td>Above 50 years</td>
</tr>
<tr>
<td>Male/female prevalence</td>
<td>5.5:1 (35-44 years)</td>
<td>Ratio reduces with increasing age</td>
<td>Equal</td>
<td>1-2% 3% in women over 65 years 1:3</td>
<td>Men: women</td>
</tr>
<tr>
<td>Course</td>
<td>Variable but increased risk of mortality</td>
<td>Childhood asthma can improve in teens but frequently returns. Adult asthma can improve with age</td>
<td>Variable Mortality not increased</td>
<td>Variable</td>
<td>Progressive</td>
</tr>
<tr>
<td>Geography/race</td>
<td>Common in countries where fat consumption is high</td>
<td>Uncommon in Far East and developing countries</td>
<td>Global although it can be uncommon in black Africans</td>
<td>All racial groups but 10 times more common in Afro-Caribbeans than whites</td>
<td>No change</td>
</tr>
<tr>
<td>Change in prevalence in recent times</td>
<td>Reducing in most developed countries</td>
<td>Increasing in industrialised countries</td>
<td>&quot;A modern&quot; disease? Little archaeological evidence before 15th century</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Authors' response

Wong and Ward concur with the major points we made in highlighting the efforts that are required to unroll the role of Chlamydia pneumoniae in atherosclerosis. Their comments, however, go beyond this and focus on a possible association of C pneumoniae with some other diseases. Despite the occasional voice of dissent, there is now overwhelming evidence of an association between C pneumoniae and atherosclerosis. What this means is quite unclear and is, of course, the overriding question that needs to be resolved. Many would think an association of C pneumoniae with asthma and to a lesser extent with sarcoidosis is a reasonable proposition. However, as the microorganism is to be found in peripheral blood mononuclear cells of a substantial proportion of individuals with and without cardiovascular disease, it is possible for it to lodge just about anywhere in the body and all sorts of relations with disease might be imagined, many perhaps turning out to be spurious. Indeed, is it reasonable to list rheumatoid arthritis and Alzheimer dementia in the same context as atherosclerosis? We think not. There is some evidence, which certainly needs to be substantiated, for an association between C pneumoniae and the HLA-B27 positive spondylarthropathy subgroup of juvenile chronic arthritis, but we are aware of only a single report of C pneumoniae in one adult with rheumatoid arthritis. Furthermore, while there is a report of this microorganism being found in brain tissue of patients with Alzheimer disease, is a single, unconfirmed report sufficient to talk in terms of an association that means anything? While it is feasible, it would, nevertheless, be startling if future research showed that this single microorganism was responsible for so many diverse diseases.

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PcR to detect M tuberculosis

Although aspirates from solitary pulmonary nodules were not included among specimens used to assess the rapid polymerase chain reaction (PCR) technique,1 this technique shows promise in improving the diagnostic rate for M tuberculosis in solitary pulmonary nodules of < 4 cm diameter.2 Seven of eight aspirates from patients with other evidence of tuberculosis (including five with bacteriological proof, and three with other evidence of tuberculosis) tested positive by PCR, and one of three not validated by either technique also tested positive by PCR.3 The sensitivity of PCR for aspirates from solitary pulmonary nodules is therefore impressively high, with virtually no false positives. The clinical decision process for malignant solitary pulmonary nodules which at present generates a positive predictive value of the order of a mere 50%, thereby yielding an unacceptably high resection rate for benign (including tuberculosis) lesions,4 would be greatly enhanced by a more enthusiastic use of the PCR technique.


Notice

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Further details from: Dr Jana Hercogova, Charles University 2nd Medical School, Department of Dermatology, V úvalu 84, 150 18 Prague 5, Czech Republic; tel +4202 2443 8700; fax +4202 2443 8720; email: jana.hercogova@lfmotol.cuni.cz

Corrections

In the leader by Dobbins, Kite and Wilcox in the March issue (vol 52, page 169) there is an uncorrected error in the section headed “Acidine orange staining of catheter blood.” Line 4 of this section should read: “The technique requires as little as 50 µl of blood...”

In the leader by McMullen in the April issue (vol 52, page 247), a single sentence has been separated unintentionally into two. In the left hand column, three lines from the bottom of the page, the sentence as corrected should read: “As the gene is on the X chromosome but the other X chromosome is “lyonised” in females, leading to inactivation of the lyonised gene, damage to a single gene results in abnormal GPI anchor expression.”
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- Are the abbreviations spelt out?
- Are the measurements in SI units?
- Are the references in Vancouver style?
Chlamydia pneumoniae and atherosclerosis.

Y Wong and M E Ward

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