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: 10 714 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: 20 455 appendices or gallbladders
- or: 34 615 vats 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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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 the individual study, 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 atherosclerotic vessels, they cannot show whether infection precedes 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 in some populations 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.
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>Male/female prevalence</td>
<td>35-44 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>Course</td>
<td>Male:female prevalence 5.5:1 (35-44 years). Ratio reduces with increasing age</td>
<td>Equal</td>
<td>1-2%; 3% in women over 65 years</td>
<td>Variable. Mortality not increased</td>
<td>Variable</td>
</tr>
<tr>
<td>Geography/race</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>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>Progressive</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>“A modern” disease? Little archaeological evidence before 15th century</td>
<td>No change</td>
<td></td>
</tr>
</tbody>
</table>

**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, this technique shows promise in improving the diagnostic rate for M tuberculosis in solitary pulmonary nodules of < 4 cm diameter. 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. 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, would be greatly enhanced by a more enthusiastic use of the PCR technique.

**Authors’ response**

Wong and Ward concur with the major points we made in highlighting the efforts that are required to unravel 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, 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 spondyloarthropathy subcertainly needs to be substantiated, for an association between C pneumoniae and the HLA-B27 positive spondyloarthropathy subcertainly needs to be substantiated. There is some evidence, which several diseases, such as the presence of an antichlamydial immune response. Arthritis Rheum 1998;41: 845–54.


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References

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Revised January 1999
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D F Griffiths

J Clin Pathol 1999 52: 398
doi: 10.1136/jcp.52.5.398a

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