Letters

Workload measurement in histopathology

I am surprised that both Suvarna and Kay's paper on workload measurement in histopathology¹ and Furness' subsequent commentary² 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.³ 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 pathology⁴ and are published in the Federal Register.⁵ The services and procedures are defined in the American Medical Association's *Current procedural terminology*.⁶

As an illustration, using the limited published data on individual workload⁷ 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 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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- 1 Suvarna SK, Kay MS. KU activity: a method for measuring histopathologists workload. *J Clin Pathol* 1998;51:530-4.
- 2 Furness P. How much work do you do in a day? *J Clin Pathol* 1998;51:642.
- 3 Hsiao WC, Braun P, Dunn D, et al. Resource-based relative values, an overview. *JAMA* 1988;260:2347-53.
- 4 Gorstein F. Pathologists input to Harvard resource based relative value scale survey: a major determinant in the accuracy of a Medicare physician fee schedule. *Hum Pathol* 1990;21:243-4.
- 5 Federal Register 1995;60(December 8):63123.
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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).^{1,2} In broad terms it parallels the Korner unit (KU) in attributing work to case/diagnosis and has been tried in some centres.³

We chose to limit ourselves to common units 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 snomed codes together, lack of necropsy work assessment, and lack of 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 are similar for KUs and RVUs (table).

We do not believe that anyone could seriously imagine a workload of pure appendices/colons/cytology/vasa as above. Do just over 2 FNA samples equate to 1 colectomy, or do 3 vasectomy samples equate to 1 hemicolectomy, using the RVU or KU respectively? We imagine not, but if nothing else it serves to illustrate the need for workloads to be seriously addressed if funded service provision is to go forwards. There is an increasing trend for standardised dissection and reporting across the UK. The Royal College of Pathologists' workload numbers of

Work	Case load	Relative ratio	KU value	Cases/year KU
FNA	4286	8.0	1	9020
Cx cytology	10 714	3.2	2	4510
Colon	1974	17.5	3	3007
Gastric biopsy	6000	5.8	2	4510
Appendix	20 455	1.7	1	9020
Vas	34 615	1	1	9020

4000 and 2500 specimens, for district general and teaching hospitals, are clearly arbitrary and of little relevance. These figures may reflect the RCPATH committee members' lack of experience of work outside teaching environments. That Welcans and Korner have limitations is also evident. To expect that KUs would be accepted across the country "as the best thing since sliced bread" would be naive, but whether we proceed with KUs, RVUs, or another system, the problem is going to have to be addressed soon. Ideally this will involve all histopathologists across the UK.

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- 1 Hsiao WC, Braun P, Dunn D, et al. Resource-based relative values. An overview. *JAMA* 1988;260:2347-53.
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- 3 Black-Schaffer WS, Young RH, Harris NL. Subspecialisation of surgical pathology at the Massachusetts General Hospital. *Am J Clin Pathol* 1996;106(suppl 1):S33-42.
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Chlamydia pneumoniae and atherosclerosis

In their leader article,¹ 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 atherosclerotic vessels, they can never 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.² 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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- 1 Taylor-Robinson D, Thomas BJ. *Chlamydia pneumoniae* in arteries: the facts, their interpretation, and future studies. *J Clin Pathol* 1998;51:793-7.
- 2 Grayston JT. Antibiotic treatment of *Chlamydia pneumoniae* for secondary prevention of cardiovascular events. *Circulation* 1998;97:1669-70.

Some of the diseases associated with *C pneumoniae*

	Coronary artery disease	Asthma	Rheumatoid arthritis	Sarcoidosis	Alzheimer dementia
Main age(s) of onset	Rises steeply from 35 years onwards	Early (childhood) and late onset types recognised	30–40 years	20–30 years	Above 50 years
UK prevalence	3–4% (40–49 years) 6–7% (60–64 years)	5%; 15% in second decade	1–2%; 3% in women over 65 years	19/100 000	Exponential increase with age
Male:female prevalence	5.5:1 (35–44 years). Ratio reduces with increasing age	Equal	1:3	Men<women	Men<women
Course	Variable but increased risk of mortality	Childhood asthma can improve in teens but frequently returns. Adult asthma can improve with age	Variable. Mortality not increased	Variable	Progressive
Geography/race	Common in countries where fat consumption is high	Uncommon in Far East and developing countries	Global although it can be uncommon in black Africans	All racial groups but 10 times more common in Afro-Caribbeans than whites	
Change in prevalence in recent times	Reducing in most developed countries	Increasing in industrialised countries	A “modern” disease? Little archaeological evidence before 15 th century	No change	

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 is, of course, the overriding question that needs to be resolved. Many would think an association of *C pneumoniae* with asthma¹ and to a lesser extent with sarcoidosis¹ 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,² 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 subgroup of juvenile chronic arthritis,³ but we are aware of only a single report of *C pneumoniae* in one adult with rheumatoid arthritis.⁴ Furthermore, while there is a report of this microorganism being found in brain tissue of patients with Alzheimer disease,⁵ 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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- 1 Saikku P. Chlamydia pneumoniae—clinical spectrum. In: *Chlamydial infections*. Proceedings of the Ninth International Symposium on Human Chlamydial Infection. San Francisco; University of California 1998:145–54.
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- 3 Taylor-Robinson D, Thomas B, Rooney M. Association of Chlamydia pneumoniae with chronic juvenile arthritis. *Eur J Clin Microbiol Infect Dis* 1998;17:211–12.
- 4 Wilkinson NZ, Kingsley GH, Sieper J, et al. Lack of correlation between the detection of Chlamydia trachomatis DNA in synovial fluid from patients with a range of rheumatic diseases and the presence of an antichlamydial immune response. *Arthritis Rheum* 1998;41:845–54.
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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 significant response to a therapeutic trial of chemotherapy) tested positive by PCR, and one of three not validated by either technique also tested positive by PCR.² 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 tuberculous) lesions,³ would be greatly enhanced by a more enthusiastic use of the PCR technique.

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- 1 Kearns AM, Freeman R, Steward M, et al. A rapid polymerase chain reaction technique for detecting *M tuberculosis* in a variety of clinical specimens. *J Clin Pathol* 1998;51:922–4.
- 2 Shim JJ, Cheong HJ, Kang E-Y, et al. Nested polymerase chain reaction for detection of mycobacterium tuberculosis in solitary pulmonary nodules. *Chest* 1998;113:20–34.
- 3 Jolobe OMP. Lessons from symposium on moving points in respiratory medicine [letter]. *Proc R Coll Physicians Edinb* 1998;28:617.

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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 “Acridine 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 McMullin 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 and 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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References

- References must be numbered in the order they appear in the text and include all information (Vancouver style; references with more than three authors should give only the first three followed by *et al*):
 - 1 Fletcher CDM, McKee H. Sarcomas - a clinicopathological guide with particular reference to cutaneous manifestations. I. Dermatofibrosarcoma protuberans, malignant fibrous histiocytoma and the epithelial sarcoma of Enzinger. *Clin Exp Dermatol* 1984;9:451-65.
 - 2 Washington JA. Conventional approaches to blood culture. In: Washington JA, ed. *The detection of septicemia*. West Palm Beach, Florida: CRP Press, 1978:41-87.
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- 1 International Committee of Medical Journal Editors. Uniform requirements for manuscripts submitted to biomedical journals. *BMJ* 1991;302:338-41.
- 2 International Committee of Medical Journal Editors. Protection of patients' rights to privacy. *BMJ* 1995;311:1272.

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Revised January 1999