PostScript

LETTERS

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Musicians playing wind instruments and risk of lung cancer: is there an association?

Lung cancer is an important public health problem. Tobacco is its main risk factor. Occupation is also an important risk factor. Some jobs have shown higher risks than others, but few investigations have asked about activities or hobbies in leisure time[1] in relation to the risk of lung cancer.

A case-control study was performed between 1999 and 2000 in the Santiago de Compostela Health District (Galicia, northwest Spain). A total of 132 cases with confirmed diagnosis of lung cancer and 187 controls were enrolled. Controls underwent trivial surgery at the same hospital as did the cases. A personal interview about lifestyle and activities (past and present) was conducted by a trained researcher.

We found that, besides tobacco and occupational exposure to carcinogens, some leisure time activities were risk factors for lung cancer.[1] Among the cases there were two musicians who played wind instruments, whereas there were no wind instrument players among the controls. The two cases had been playing the clarinet and trombone for 35 and 30 years respectively. Both were ex-smokers (moderate smokers) and played music as a hobby. They had epidermoid lung cancer and were diagnosed at 57 and 76 years of age.

Since in our population the prevalence of persons playing musical instruments and specifically wind instruments is extremely low, we think that this activity might be a risk factor in development of lung cancer. The very low number of persons playing this type of musical instrument is probably a reason for the lack of studies focused on this activity, as many occupational studies of lung cancer and occupation are based on registries of workers. One study[2] found an increased mortality rate of lung cancer for a category that included painters, potters, musicians, and actors—an inhomogeneous category that did not allow us to extrapolate results. The results were not adjusted according to smoking history.

This hobby requires inspiration and breathing of large volumes of air, making the lung alveoli expand more than in other people. This fact could facilitate the penetrance of carcinogens in the cells of the lung epithelium, and this could be more harmful in smokers. We have found no other studies that have reported this possible association. It would therefore be necessary to explore this association in greater samples of professionally exposed persons in order to ascertain whether this finding is consistent or due to chance.

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How important is personal exposure assessment in the epidemiology of air pollutants?

The paper by Harrison and colleagues[1] and the accompanying editorial by Cherrie[2] in the October 2002 issue of *Occupational and Environmental Medicine* address the important issue of personal exposure assessment (of air pollutants) in environmental epidemiology. After reading both papers we would like to make some comments with regard to the design, conduct and statistical analysis of the study by Harrison *et al* and at the same time answer the question raised by Cherrie in his editorial.

Coming from the occupational exposure assessment arena it is interesting to see that our environmental colleagues are still relying to a large extent on static (microenvironmental) sampling and even rely on shadowing to represent personal exposure. The latter brought back memories of old occupational hygiene textbooks with pictures of technicians standing with a sampling probe in the breathing zone of a worker (clearly hindered while carrying out his work task). It is interesting to note that Dr Cherrie's very relevant earlier work[3] on whether wearing sampling pumps affects exposure (it hardly did) was not mentioned in both papers.

The paper by Harrison and colleagues[1] clearly states as one of its goals to answer the question "Does modelling through the use of microenvironment measurements and activity diaries produce reliable estimates of personal exposure to air pollutants". However, in the only setting where personal exposures were actually measured (phase 1, volunteers; with regard to phase 2 we do not think that shadowing results can be seen as equivalent to personally measured exposure) it is hard to grasp from both fig 1 and table 2 which exposure was actually modelled (1 hour averages, 2–3 day averages) and how (a formula was only provided for measurements within the susceptible groups).

When comparing direct personal measurements for CO and PM_{10} with the modelled results, the authors exclude all data which are not directly comparable—that is, when the volunteer spent most of their time out of house, and all the data for smokers. It is therefore not surprising that good correlations were found between personal and static measurement results. Why were smokers excluded? Was their measured CO exposure representing a different kind of CO leading to a different health effect? We know that excluding smokers or people with unventilated gas heaters is common practice in the statistical analyses of environmental exposures, but this would only make sense if we were expecting different risks from the same exposure originating from different sources.

In fig 1 the authors present 120 comparable data points for 11 individuals; given the repeated nature of the sampling these data points cannot be seen as statistically independent. Putting a simple regression line through these points is therefore not correct and application of a mixed effects model would have been more appropriate. Besides that, when estimating environmental exposure, for instance, for a panel study, we are interested in the full range of exposures both in the temporal and spatial sense (not only for the room with the static sampler). However, Harrison *et al* conclude, "... modelled personal exposure is unable to reflect the variability of measured personal exposures occasioned by the spread of concentrations within given microenvironments".

Both Cherrie and Harrison *et al* claim that microenvironmental sampling would be a good alternative for direct personal exposure measurements that supposedly are "costly and time consuming". However, the costs for sampling microenvironments in a general population study will be far greater if we want to measure all the microenvironments people end up in (for instance, in table 1 seven environments are indicated, and most of them will most likely be different for each study participant). In addition, it will be practically impossible to measure some of these environments as the authors point out. In their study, it was not possible to collect data for all appropriate microenvironments, even for a comparatively small number of subjects.

Recently, a very insightful paper was presented at the X2001 conference in Gothenburg. Seixas and colleagues[4] showed that in a study to assess occupational noise exposure, a task based methodology (analogous to microenvironmental sampling in environmental exposure assessment) could only account for 30% of variability in daily exposures. They even considered this estimate somewhat optimistic since their estimated noise exposures were derived from the same data on which the daily average exposures were estimated. In addition they clearly pointed out that using simple task based averages that artificially compress exposure variability resulted in a very substantial negative bias in the estimated daily exposure.

In our opinion, we should aim to collect personal exposure measurements when estimating exposure for epidemiological studies.

We agree that smaller and lighter sampling instruments will need to be developed, as was suggested by Cherrie in his editorial. Recent studies in both the occupational and environmental arenas have shown that study subjects are capable of carrying out personal measurements themselves (and by doing so, cutting out the costs of the technician).[5–9] In all these studies except one,[7] far more than 100 personal measurements were generated, which shows that studies of this size are not exceptional as was suggested in the editorial by Cherrie.

The question raised by Cherrie, "How important is personal exposure assessment in the epidemiology of air pollution?", can only be answered with a firm "very important", if we want to capture the full range of personal exposures experienced in the general environment. In addition, given the relatively low concentrations in the general environment, we will need to measure these accurately. Microenvironmental monitoring and consequent modelling based on diaries will not provide sufficient resolution and accuracy.

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Will sewage workers with endotoxin related symptoms have the benefit of reduced lung cancer?

Thorn and colleagues[1] reported that sewage workers suffer from various symptoms which can be related to bacterial endotoxin (lipopolysaccharide) exposure. Other studies[2–5] have shown that some members of this occupational group are commonly exposed to endotoxin. However, there appears to be a large discrepancy in endotoxin exposure among those categorised within this group.[2] Endotoxin exposure to some of these workers appears to be sufficient to induce a respiratory response characteristically associated with endotoxin.[2] Workers that have the highest exposure in sewage treatment are suggested to be associated with the waste treatment process.[3] Professor Rylander pointed out that endotoxin exposure to this occupational group is low overall (personal communication with Professor Rylander). Rapiti and colleagues[6] suggested that the lack of an increased lung cancer rate in one study[7] and reduced risk of lung cancer in another[8] for sewage workers may be related to endotoxins in their occupational environment as was originally reported for cotton textile workers.[9] Other studies[10 11] that reported on lung cancer rates for sewage workers support these findings as suggested by Rapiti and colleagues.[6] Rylander[12] and Lange[13] previously reviewed the epidemiological literature on reduced cancer rates in various occupations that are exposed to endotoxin.

A number of epidemiological,[12–16] experimental,[17 18] and clinical[19 20] studies have suggested that endotoxin is effective against cancer. A recent study in humans by Palmberg and colleagues[21] reported that there is a rapid blood response of total leucocytes, monocytes, and granulocytes within seven hours followed by a dramatic decline within 24 hours. These findings are supported by an investigation by O'Grady and colleagues[22] in humans, in which endotoxin was instilled into a lung segment; increased tumour necrosis factor (TNF) and interleukin 1 were found in the bronchoalveolar lavage fluid 2–6 hours afterwards. Cytokine levels returned to normal concentrations within 24–48 hours after treatment. An increase of TNF in lung fluids as a result of exposure to endotoxin and dust containing endotoxin has been reported by others conducting human investigations as well,[23 24] including the suggestion of a dose-response relation.[25] Thus, periodic exposure as would likely be experienced by those in sewage and dusty occupations may afford a continual or pulse stimulation of the immune system. Such stimulation may enhance production of anticancer mediator factors and cells[26] that are suggested to be responsible for observed reduced lung cancer rates.[13]

Experimental studies[27] have suggested that benefit of endotoxin exposure is most effective during initiation of lung cancer with a finding of less benefit for established tumours. This, together with results from Palmberg and colleagues,[21] supports the hypothesis[14 27] that endotoxin in an occupational setting is effective against the early formation of lung cancer. This further suggests that endotoxin reduces the incidence of lung cancer by stimulating the immune system to guard against early lung cancer events.

Additional studies are warranted on the relation of endotoxin and reduced lung cancer rates. This relation has been suggested for textile and agricultural workers.[12–16] There is no reason to believe that it will not exist for other occupational groups exposed to endotoxin. Many have explained that the relation is not one of benefit, but rather methodology and bias, including differences in smoking rates.[6–9] However, this explanation is not supported by experimental and clinical investigations involving endotoxin. The major influence on lung cancer is tobacco use (smoking). Although smoking is identified as one of the reasons for lower than expected rates in some populations, some studies[6–9] have shown that smoking is not always an explainable factor or bias for reduced lung cancer. For example, Rapiti and colleagues[6] reported that the consumption of cigarettes and prevalence of smoking in a population of municipal waste workers was higher than the general population, but the incidence of cancer deaths (standardised mortality ratio) for lung cancer in this group was 0.55. Epidemiological studies need to include and report not only detrimental outcomes but also potentially beneficial associations.

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Neurobehavioural testing in workers occupationally exposed to lead

The article of Dr Goodman and coworkers on "Neurobehavioural testing in workers occupationally exposed to lead"[1] covers an interesting approach with a surprising main message: "None of the individual studies is conclusive or adequate in providing information on the subclinical neurobehavioural effects ...". Such a sentence astonishes a reader since the studies used were selected from established journals.

A long section of the discussion deals with an article of Meyer-Baron and Seeber,[2] the beforehand published meta-analysis on the topic. We agree that prospective studies are the best basis to receive a stable knowledge about exposure effects, also in neurobehavioural studies. However, the repeated information on cross sectional studies should also be accepted as source for conclusions on (neurobehavioural) effects due to exposures. Meta-analyses are one approach to search such summarising information.

Taking into account that the extended study selection in the article of Goodman *et al* may lead to different results we do not agree with several arguments. For example, they refer to the bias problem, the exposure range, the interpretation in terms of age related changes, and the results for the digit symbol test. On these problems an exchange of opinions has been published in *Archives of Toxicology*.[3 4] Without making reference to this discussion, several arguments and conclusions were presented again. They are identical with the main conclusions in an anonymous "expert opinion" for the German Battery Association.[5]

From our point of view it makes no sense to repeat the same details of argumentation for a second time. However, we believe that the readers of your journal should be informed that the conclusions of the article of Goodman *et al* have been discussed in other places. In the meantime an additional article on the subject has been published.[6] In this article the data of the original "expert opinion"—the basis of the article in *Occupational and Environmental Medicine*—and the data of our first meta-analysis were comparatively evaluated. We hope that the critical readers of your journal pick up the full information on the matter. Thereupon they may draw their own conclusions regarding meta-analyses of neurobehavioural effects due to occupational exposure to inorganic lead.

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Authors' reply

We thank Drs Seeber and Meyer-Baron for their comments on our paper,[1] and also Drs Schwartz, Stewart, and Hu for comments published in the September 2002 issue of *OEM*.[2]

The following is our response to the specific criticisms made by Schwartz and colleagues:

(1) "*No evaluation of the quality of the evidence from available studies based on study design and analytical method.*" Study quality assessment was the first task we completed. As discussed in our methods section, our quality criteria included evaluating pre-exposure status, use of blinding procedures, and adjustments for age, other occupational exposures, alcohol use, and socioeconomic factors (income level, education, etc).

(2) "*Data were combined from poorly done studies with data from well done studies.*" Table 1 shows that no study satisfied all of the above quality criteria. Schwartz *et al* did not provide criteria to distinguish a "poorly done" from a "well done" study. However, we conducted an additional analysis of the five relatively well designed studies that adjusted for age, education, and alcohol use (Baker and colleagues,[3] Campara and colleagues,[4] Chia and colleagues,[5] Maizlish and colleagues,[6] and Williamson and Teo[7]). These five allowed us to conduct a meta-analysis for only three tests. For the Santa Ana preferred hand test, the effect size changed from non-significant negative to non-significant positive. For the Santa Ana non-preferred hand the result changed slightly towards the null and remained statistically non-significant. For the digit symbol test, the result changed away from the null and remained statistically significant in the fixed effects model, but changed slightly towards the null and was no longer statistically significant in the two random effects models.

(3) "*Inclusion of studies that did not control for age and education.*" Schwartz *et al* do not provide evidence that age and education are "*the two most important predictors*". One could argue that alcohol use or the presence of pre-existing neuropsychiatric conditions could also act as powerful confounders. The studies in our meta-analysis had non-overlapping strengths and limitations and further inclusion or exclusion based on quality would be a matter of judgement. However, an additional analysis based on the 13 studies that adjusted for age and education revealed that, as opposed to our original findings based on all 22 studies, none of the tests showed a statistically significant difference in all three models. (See *OEM* website for results table.)

(4) "*No adjustment for age, education, or lead dose differences across studies.*" This criticism appears to be somewhat theoretical, as the data did not allow such adjustment.

(5) "*Reliance on exposed versus control comparisons*" rather than "*only including studies that reported beta coefficients for the blood lead versus test score relation, or adjusting for mean blood lead levels in exposed and non-exposed groups.*" We used the same definition of exposure as the previously published meta-analysis by Meyer-Baron and Seeber[8] (less than 70 µg/dl) to find out if the results of our two studies were reproducible. The direct comparison of the two analyses in the discussion section was important in explaining our position with regards to meta-analysis as a research technique. We agree that other approaches could also be informative. The statement "*The authors conclude that blood lead levels, that are described as 'moderate' in one location in the manuscript and 'low' in another, are not associated with neurobehavioral test scores*" misrepresents our conclusions listed on page 222 of our paper.

(6) "*Reliance on a small number of unspecified studies for effect estimates. Table 2 of the study reports the number of studies that were combined to derive effect estimates, but does not specify which studies were combined.*" The original version of the paper included information on each individual study; however, based on the reviewers' and editor's comments, we had to shorten the manuscript substantially. We will make this information available on request. With respect to the purported omission from our meta-analysis of the May 2001 article by Schwartz and colleagues,[9] this article was unavailable when our manuscript was submitted for publication in December 2000. The other two studies they cite did not meet our inclusion criteria. While we have not had an opportunity to evaluate the association between cumulative exposure to lead and neurobehavioural testing results, we did note that the

2001 article by Schwartz and colleagues[9] found no association between tibia lead levels and test scores.

With regard to Seeber and Meyer-Baron's statements that "*the repeated information on cross-sectional studies should also be accepted as source for conclusions on (neurobehavioural) effects due to exposures*" and that "*meta-analyses are one approach to search such summarising information*", after having reviewed the results of five meta-analyses on the subject (two presented in the recent article by Seeber and colleagues,[10] our paper,[1] and the two additional re-analyses discussed here), we found five different sets of results with no evidence of consistency to qualify these results as "repeated". Therefore, we have to adhere to our original conclusions.

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Ambient neighbourhood noise and children's mental health

Readers may be interested to know that there are other recent studies that have provided equivocal evidence concerning the effects of environmental noise on children's mental health that have not been cited in the article by Lercher *et al*, published in the June 2002 issue of *Occupational and Environmental Medicine*.[1] These new results need to be considered in the light of fact there has not been clear research evidence to support or dispute whether noise exposure in linked to mental health problems in children.

We have found inconsistent mental health results in our three recent studies examining the impact of aircraft noise on child health around Heathrow airport.[2–4] In the West London Schools Study,[4] aircraft noise was weakly associated with hyperactivity and psychological morbidity as measured by the Strengths and Difficulties Questionnaire (SDQ[5]) completed by parents.

The SDQ is one of the most widely used psychometrically valid instruments to detect psychological morbidity in children in both the UK and internationally. However, in our other two studies using both the parent completed SDQ, the teacher completed Student Behaviour Checklist, and child self reported Depression (Child Depression Inventory, CDI) and Anxiety (Revised Child Manifest Anxiety Scale) we did not find any association between mental ill health and aircraft noise exposure.[2 3]

The Austrian results should be placed within the context of existing studies with respect to two points: (1) the construct being measured in the Austrian study; and (2) the small effect size and inconsistency with previous research.

In the Heathrow studies we used internationally recognised child mental health screening tools, that have equivalent psychometric properties to the KINDL (only used in German speaking countries). It is worth noting that the KINDL is normally defined as a "valid and reliable index of quality of life",[6] rather than a sensitive screening tool to detect specific mental health problems. It is possible that the mental health results reported by Lercher and colleagues are tapping into impaired quality of life and wellbeing, rather than a precise mental health outcome such as "depression". The definition of "mental health" used by the authors needs to be clarified. The fact that the Austrian results do not replicate our Heathrow results raises the question: Does the KINDL measure wellbeing and quality of life rather than mental health? Furthermore, teacher reports of classroom adjustment would not normally be classified as a "mental health". Perhaps it might be more accurate to conclude from the Austrian research that: "ambient levels of noise in the community are associated with decreased quality of life and poorer classroom behaviour (rather than 'mental health') in elementary school children".

In summary, we feel that new research is necessary to provide further evidence about the effects of noise on child mental health. Even though Lercher and colleagues have taken the field of research forward with their two stage study design strategy, there is still more work to be done to clarify the terminology and measurement of mental health in the field of non-auditory health effects of noise. Specifically, a clear definitional and operational distinction needs to be made between stress/wellbeing/quality of life and mental health.

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No change in sex ratio in Ramsar (north of Iran) with high background of radiation

A few areas of the world show high levels of natural radiation, and one of these areas is located in Iran. Ramsar is a northern coastal town situated in the Caspian littoral (in Mazandaran province, Iran) on the slopes of the Alborz mountain range, and surrounded by forests. It is situated at 49° 40′ eastern longitude and 36° 53′ northern latitude. The area is rich with mineral springs. Investigations into the amount of radium-226 in water started more than 30 years ago.[1] It has been reported that inhabitants of Ramsar receive an annual radiation absorbed dose from background radiation that is up to 260 mSv, substantially higher than the 20 mSv that is permitted for radiation workers.[2]

Annual births subdivided by gender, were obtained from Statistical Center of Mazandaran province. Because of the relatively small number of annual births in the urban area of Ramsar (currently about 670 per annum), analysis was carried out on the 11 year total for male and female live births, for the period 20 March 1989 to 19 March 2001, equal to Iranian calendar 1368 to 1379 Hejirae Shamsi (HS). The data was not available for the 1378 HS (equal to 20 March 1999 to 19 March 2000).

To test the null hypothesis that the probability of a male live birth in Ramsar is equal to that in the control populations, a χ^2 test was conducted. A value of $p < 0.05$ was considered significant. The sex ratio is expressed as the proportion of total live births that were males.

The sex ratios at birth in the urban area of Tonekabon, the nearest city to Ramsar (about 20 km distance) and the urban areas of Mazandaran province (excluding Ramsar) were used as controls. The overall sex ratios in Ramsar, Tonekabon, and the urban areas of Mazandaran province were 0.511 (total live births = 7591), 0.517 (total live births = 14 266), and 0.509 (total live births = 253 918), respectively. There was no significant difference between Ramsar and either Tonekabon ($\chi^2 = 0.95$, df = 1, $p = 0.33$) or urban areas of Mazandaran province ($\chi^2 = 0.13$, df = 1, $p = 0.71$).

It has been reported that the sex ratio in the offspring of male radiologists is significantly lower than that in control populations.[3] However, this is not consistent with the present result. This discrepancy could be attributed to the exposure of both parents to ionising radiation. Alternatively, because the inhabitants of Ramsar have lived for many generations in an area of high background radiation,

some kind of adaptation might have occurred. This study was supported by Shiraz University

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William Harvey and air pollution

Thomas Parr died, on 14 November 1635, at what was recorded as the advanced age of 152 years and 9 months. A postmortem examination was performed and a record made by William Harvey. A translation by Alan Muirhead of Harvey's account is included in the Everyman edition of De Motu Cordis.[1] Parr seemed remarkably well preserved, and when considering the cause of death, Harvey identified air pollution as a possible contributory factor. His words are worth reading:

> "It was consistent to attribute the cause of death to the sudden adoption of a mode of living unnatural to him. [Parr had been brought to London not long before he died by Lord Arundel.] Especially did he suffer harm from the change of air, for all his life he had enjoyed absolutely clean, rarefied, coolish, and circulating air, and therefore his diaphragm and lungs could be inflated and deflated and refreshed more freely. But life in London in particular lacks this advantage—the more so because it is full of the filth of men, animals, sewers, and other forms of squalor, in addition to which there is the not inconsiderable grime from the smoke of sulphurous coal constantly used as fuel for fires. The air in London therefore is always heavy, and in autumn particularly so, especially to a man coming from the sunny and healthy districts of Shropshire, and it could not but be particularly harmful to one who was now an enfeebled old man."

Harvey went on to point to the possible adverse effects of changing from a simple diet to a rich one. Harvey's observation on the possible effects of air pollution are interesting in that they antedate Evelyn's much better known analysis by 26 years. In retrospect we can see that Harvey identified the effects of short term exposure to high levels of air pollution on a vulnerable person.

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Alternative methods of administering amyl nitrite to victims of cyanide poisoning

The traditional method of administering amyl nitrite to a victim of cyanide poisoning, is to break an ampoule in a handkerchief and then intermittently hold this under the victim's nose.[1 2]

I would like to suggest two alternative methods for administering amyl nitrite. The first method is to use a nebuliser. The second method is to use an inhaler similar to the Penthrox device, normally used to administer methoxyflurane for emergency analgesia.

With appropriate training, either method could be used by first aid staff. This could be of particular value to remote mine sites where the absence of medical staff may preclude intravenous administration of cyanide antidotes such as dicobalt edetate, sodium thiosulphate, sodium nitrite, or hydroxocobalamin.

Both methods offer the following advantages over the traditional method:

- Oxygen can be administered during treatment
- Rapid delivery of the drug
- Accurate dose delivery
- Less risk of inhalation by first aid or medical staff
- Less risk of injury due to glass fragments.

The inhaler device would also be particularly well suited to the treatment of large numbers of victims following industrial disaster or terrorist attack—the risk of which has been recently alluded to.[3]

One concern about introducing these methods is the potential for amyl nitrite toxicity. Experimental research is recommended to determine safe dosages and frequencies for each method.

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BOOK REVIEWS

Basic Statistics and Epidemiology, A Practical Guide

Antony Stewart (pp 151; £19.95) 2002. Oxford: Radcliffe Medical Press. ISBN 1 85775 589 8

This book is "aimed at people who want to understand the main points, with minimum fuss"—no small task when the subject at hand is statistics! However, this book manages it by using short, concise, easy to read chapters that contain simple examples and a minimum of mathematics. The style is suitable both as a text to read from start to finish and as a reference book. It introduces students to the basic terms and concepts in statistics and epidemiology and provides a very basic "walk through" of some simple formulae.

The book is loosely divided into two parts. It begins with a brief description of what are *statistics*, their role in the study of *populations*, and ways in which *samples* can be drawn from populations in order to make statements about individuals in the population. Concepts such as probability, significance testing, and standard errors are introduced and explained before a very brief mention of some simple statistical tests. In these later chapters insufficient information is provided to allow the reader to understand the mechanisms of these tests, or the conditions required for their application. However, useful references are given where the reader may find further detail.

In the second "half" of the book the author covers basic epidemiological concepts, describing the difference between prevalence and incidence, and how to measure disease frequency, and discussing bias and confounding. Later chapters in this section introduce basic study designs such as cohort, case-control, and randomised clinical trial (or RCT), and describe the planning and use of questionnaires.

The book provides a useful glossary of terms, including mathematical symbols and a number of statistical tables. A set of exercises is given and answers are provided. These are an invaluable addition to the book.

For the non-mathematical health student faced with the daunting prospect of having to begin studying statistics, this 150 page book is an excellent primer. It introduces basic terms and concepts and gets the student started. However, statistical concepts can be difficult to understand, and in some chapters in this book the brief introduction given falls short of helping the student understand the concepts properly. Therefore the interested student may see this book as a first introductory text, shortly to be followed or indeed accompanied by a more full statistical or epidemiological textbook. For this purpose an excellent, current bibliography is provided.

R Atkinson

Occupational Disorders of the Lung: Recognition, Management and Prevention

David J Hendrick, P Sherwood Burge, William S Beckett, Andrew Churg (pp 638; £99.99) 2002. London: WB Saunders. ISBN 0 7020 2507 0

The authors of this book aim to draw attention to "the changing nature of the contribution the occupational environment makes to lung disease, and to the particular difficulties this poses for those who find themselves responsible for patient care or the management of relevant industries". The result is a book which is easy to read, helped greatly by use of a standard format for each chapter. The format includes management of both the individual and the workforce, and prevention. The authors have also used difficult or "grey" cases, similar to one other textbook in the field. The difference here is

that the cases were circulated to all the contributors to this volume and the overall response summarised in the text. The lack of complete agreement in many instances is comforting at one level—"textbook" cases are the exceptions in practice—and this approach gives a far better feel for the real life situation.

Another attractive feature of this book is the chapters dedicated to descriptions of certain industries and the problems that arise from those workplaces, including mining, farming, the automotive industry, and health carers among the seven chapters. This does lead to repetition of some information between chapters but, as the authors rightly point out, readers will tend to dip into one particular part of the book, and repetition under these circumstances is helpful rather than an irritation. The chapters on specific disciplines used in the investigation and management of occupational lung disease (for example, imaging and occupational hygiene) are good and sufficient for most needs in this context. The chapters on legislation divided geographically into North America, Western Europe, and the Pacific, Far East, and Australasia is an excellent attempt to widen the relevance of the book.

My criticisms are few and minor. While there are good generic sections on how to take an occupational exposure history and on surveillance, it might have been a useful addition to include a chapter on epidemiological aspects unrelated to surveillance and more to the research field. This would allow greater expansion on the healthy worker effect and perhaps also the opportunity to compare the now burgeoning literature on the health effects of the broader environment and how these findings might apply to the occupational scene. Boxes have been used for specific sections within chapters. Sometimes this works, but sometimes it does not. There are one or two boxes which run to four or five pages and I feel that these would quite happily sit as sections within the chapter rather than boxes. Boxes need to be short and punchy.

This book is an excellent addition to the literature in this area, complimenting nicely the classical standard textbooks, and at a penny under £100 is good value for money. It is targeted at all physicians, hygienists, health and safety officers, and administrators, and successfully hits that target for all these groups. For exam purposes (for example, AFOM in the UK) this should be regarded as the standard text.

J G Ayres

Bone's Atlas of Pulmonary and Critical Care Medicine, 2nd edition

Edited by G Douglas Campbell Jr and D Keith Payne (pp 315 plus index and colour plates; $95) 2001. Haggerstown, MD: Lippincott Williams & Wilkins. ISBN 0 7817 3436 3

This book aims to cover an enormous subject, and the editors have done very well to contain it to a little over 300 pages. Its 26 chapters are grouped into six sections, the lion's share being occupied by respiratory topics, with critical care being limited to the relatively short final section. The atlas format is certainly stylish and on the whole achieves the editors' aim—that is, of helping busy clinicians and students of chest disease absorb a large amount of information in a relatively short amount of time.

Despite the numerous contributors, the book's layout is uniform and very accessible; text is limited and punchy and extensive use has been made of diagrams, flow charts, and tables to supplement the generally good quality photographic images. The grouping of the colour plates to the final pages of the book, to contain printing costs, is a little distracting but a justifiable compromise.

All of the material is up to date and well referenced, though tends to some extent to be dominated by North American sources and opinion. I found the chapters dealing with lung cancer, bronchiolitis obliterans and other bronchiolar airway disorders, and sarcoidosis to be particularly useful and excellent sources of a large and diverse amount of information. In contrast the chapter dealing with interstitial lung disease was to me a little disappointing. The chapters covering sleep disorders, HIV and fungal infections, lower respiratory tract infections, and nutrition are new to this edition and are welcome additions. The use of graded evidence based recommendations for diagnostic and therapeutic interventions is variable between chapters and its more consistent application would add further to this book's already considerable value.

I am sure this atlas will have broad appeal to both undergraduate and postgraduate students of chest medicine as well as busy practitioners. It would be a valuable aid to those preparing for postgraduate exams as well as to specialist registrars in respiratory medicine, who I'm sure would find it a very useful source throughout their trainee years. Intensivists and trainees in critical care will, I expect, find the balance towards respiratory medicine less appealing. It has few competitors in terms of its breadth and clarity and it represents good value for money; in short it deserves a place in all good medical libraries.

W S Tunnicliffe

The Health Effects of Chrysotile Asbestos

R P Nolan, A M Langer, M Ross, F J Wicks, R F Martin (pp 304, $38) 2001. Ottaza, Ontario: The Canadian Mineralogist. ISBN 0 921 294 41 7

The famous mortality study led by Corbett McDonald has followed 11 000 Canadian chrysotile miners and millers until 80% were dead; only 33 mesotheliomas were reported and excess lung cancers occurred only at very high exposure levels. Yet that same chrysotile used in textile manufacture in South Carolina was associated with a 50 times greater lung cancer mortality.

This volume, published in 2001 by The Canadian Mineralogist, reports the papers presented and the ensuing discussion and commentary at a symposium in 1997 called by the Canadian Government to discuss the health issues surrounding the continued production and use of chrysotile asbestos. Can the mineral be used safely? To most uninformed observers, the answer must be a clear no. The true answer is of course not so clear cut. Much of the evidence suggests that chrysotile itself is much less hazardous than the amphiboles and that the serious risks associated with chrysotile are a consequence of its contamination by tremolite, an amphibole that is found in geological intrusions into the chrysotile ore body. These are the issues discussed by the distinguished geologists, mineralogists, epidemiologists, risk analysts, and pathologists who contributed to the symposium. Among them are the last published contributions of two who made great contributions over many decades to investigating the hazards of asbestos and to protecting workers, the late Chris Wagner and Bob Murray.

The resolution of this conundrum may seem unimportant to those who live in countries where past exposures have been to mixtures of amphiboles and chrysotile and where use of asbestos has effectively ceased. However, industry continues to need durable fibres and the poor world sees substantial advantages in using cheap asbestos cement for water pipes and roofing material. And the issue is of course important to the Canadian and Russian chrysotile industries and their employees. Anyone who has been involved in the asbestos debate, who gives advice to industry or lawyers on asbestos issues, or who is interested in the complexities of the interface between science and regulation will find much of fascination in this volume.

A Seaton

NOTICES

First World Congress on Work-Related and Environmental Allergy (1st WOREAL), and Fourth International Symposium on Irritant Contact Dermatitis (ICD), Helsinki, Finland, 9–12 July 2003

Congress on Work-Related and Environmental Allergy

- Work related and environmental aspects of respiratory and skin allergy
- Specific issues related to pathophysiology and skin allergy
- Management and prevention of allergy

Irritant Contact Dermatitis Symposium

- Occupational irritant dermatitis
- Prevention of irritant dermatitis
- Alternative methods for the assessment of irritants
- Irritant dermatitis from cosmetics

Satellite events

- Satellite Symposia, 9 July 2003
- Allergy School, 9–10 July 2003
- 7th International NIVA Course on Work-Related Respiratory Hypersensitivity, 11–15 July 2003

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NIVA Training Programme 2003: Advanced Courses in Occupational Health and Safety

NIVA Training Programme 2003 offers 12 advanced courses on current themes of work life. Further information is available from the NIVA Office:

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Fax: +358 9 4747 2497, +358 9 2414 634
Email: niva@ttl.fi
Website: www.niva.org

Assessment of Psychological Factors at Work
3–6 March 2003, Geilo Hotel, Geilo, Norway

Evaluation and Good Occupational Health Practice
23–27 March 2003, The Fell Hotel, Saariselkä (Lapland), Finland

Principles of Etiologic/Etiodiagnostic Research
11–16 May 2003, Hanasaari Cultural Center, Espoo (Helsinki), Finland

Toxicokinetic and Toxicodynamic Modeling in Occupational Health
15–19 June 2003, Red Cross Educational Training Center, Gripsholm, Sweden

Work-related Respiratory Hypersensitivity
10–15 July 2003, Marina Congress Center, Helsinki South Harbour, and The Sunborn Yacht Hotel, Naantali, Finland

Bullying and Harassment at Work
11–15 August 2003, Hotel Eckerö, Åland, Finland

Good Management Practice—Interaction of Environment, Safety and Quality
31 August–4 September 2003, Hotel Levitunturi, Sirkka (Lapland), Finland

Workplace Health Promotion—Practice and Evaluation
The first part 15–17 September 2003, Hotel Eckerö, Åland, Finland and the second part 19–21 January 2004, The Nordic School of Public Health, Gothenburg, Sweden

Indoor Air Quality Problems—Link between Indoor Pollution, Psychological Factors and Complaints
22–26 September 2003, Vilvorde Course Center, Vilvorde (Copenhagen), Denmark

Occupational Health Risk Assessment and Management
6–10 October 2003, Medical Academy of Latvia, Riga, Latvia

Introduction to Occupational Epidemiology
23–29 October 2003, Hotel Gentofte (Copenhagen), Denmark

Work-related Musculoskeletal Disorders: Current Research Trends
1–7 November 2003, The Sunborn Yacht Hotel, Naantali, Finland

CORRECTIONS

The authors of "Association between job strain and prevalence of hypertension: a cross sectional analysis in a Japanese working population with a wide range of occupations: the Jichi Medical School Cohort Study" (Tsutsumi A, Kayaba K, Tsutsumi K, Igarashi M, *Occup Environ Med* 2001;**58**:367–7) have asked for the following errors to be pointed out.

- There are errors in the abstract (line 16) and text (page 368, left hand column, line 5). A part of the baseline data was collected in 1995 in two of the 12 study sites so that the correct period was 1992–95 (not 1992–94).
- On page 368, left hand column, line 24, the number of older participants (over 69) should be 696 and not 699.

These facts do not, however, affect the study findings.

We apologise for the following error in the book review, "Late lessons from early warnings: the Precautionary Principle 1896–2000" by R L Maynard. A copy of this book is available to download free of charge from EEA Online. The URL, however, was published incorrectly. The correct link is: http://reports.eea.eu.int/environmental_issue_report_2001_22/en.