Correspondence

Long term exposure to industrial solvents

SIR,—A man aged 69 with a history of long term (30 years) exposure to dry cleaning solvents, was seen at the Andrus Older Adult Center (AOAC) at the University of Southern California for evaluation of memory problems. Alzheimer’s disease had been diagnosed on the basis of confusion, disorientation, and clinically significant memory impairment. The WAIS-R subtest digit span had been administered on three occasions. Careful examination of the medical records showed a fact inconsistent with the diagnosis of Alzheimer’s disease, the patient’s digit span scores had steadily improved since an episode of acute confusion; the scores of patients with Alzheimer’s disease decline over time. Neuropsychological testing at the AOAC showed no brain dysfunction. Testing was repeated after four months, again, no dysfunction was found. On both testing occasions at the AOAC, he performed normally or in the superior range. This fact, in addition to his medical history, led us to hypothesise that he had been the victim of a delirious episode rather than a progressive dementia. His work history included, in addition to the 30 year exposure to dry cleaning solvents, several other indications of toxic exposure: (a) his cleaning plant had been cited by the state for toxic fumes; (b) he had experienced, at about the time of the delirious episode, complete hair and nail loss, an occurrence for which his physicians could find no plausible cause; (c) the onset of a seizure disorder concurrent with the delirious episode; and (d) his serum concentration of perchlorethelyne was 746 ppm (the mean for the general population is about 27 ppm). These facts led us to hypothesise that the delirious episode resulted from long term exposure in the workplace. Possible health consequences of long term exposure to industrial toxins have not been sufficiently considered, and cases such as this lend support to the notion that epidemiological research on this topic is necessary. To our knowledge, no data base which has the necessary long term epidemiological data to investigate this problem exists. It is the intent of this letter to inform clinicians as to the possibility of this syndrome and to bring it to the attention of scientists who are able to test this hypothesis.

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Occupational bladder cancer and the hospital clinician

SIR,—Your recent editorial (1989;46:73) causes us a certain amount of concern. While fully supporting the general message and welcoming the proposal to set up an independent unit to advise urological surgeons about occupational bladder cancer, we think two points should be challenged.

Firstly, Baxter dismisses organised urinary cytology as a screening procedure of doubtful benefit. In fact in the article to which he refers Jacobs never said this. Indeed, she stated in her conclusions that “Urinary cytology is a valid and acceptable screening technique . . . . ” Objective assessment of the benefit relating to duration of survival has been studied by Cartwright et al,2 who concluded that survival was prolonged as a result of urinary malignant cell cytology screening. This increase in survival was due partly to a greater proportion of early stage disease being diagnosed and partly because of other differences in the screened group. Possibly future studies may confirm these conclusions.

Subjectively it would also be apparent that the technique makes it possible to monitor the incidence of occupational bladder cancer and therefore the effectiveness of control measures at an early stage. It ensures that affected employees receive prompt treatment and it allows individuals to benefit from compensation where relevant. Finally, those individuals who have been affected by the disease are usually grateful that the technique was available for early diagnosis and treatment and those who continue to be screened feel reassured that a beneficial health surveillance programme is in existence and see it as evidence that the employer cares.

Secondly, Baxter seems to assume that occupational carcinogens will continue to emerge. We suggest that this is an unduly pessimistic view. Although it may well be that theoretical carcinogens will continue to be identified in laboratory studies, modern methods of hazard and risk assessment coupled with improvements in the control of hazardous substances as required by the COSHH regulations will practically eliminate new causes of occupational cancer in man.

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References


Dr Baxter replies:

Brooke et al should have finished their quotation from Jacobs: “Urinary cytology is a valid and acceptable screening technique, but as yet there is no clear evidence of increased length of survival for screened groups, nor have claims of improved quality of survival in screened groups been adequately investigated” (my italics).

Until future studies do resolve this issue, urinary cytology will remain of doubtful benefit, a view shared by the Health and Safety Executive who recently decided not to recommend the technique for workers exposed to MbOCA. Brooke et al make other points which do not relate to its validity as a screening tool but are reasons for undertaking medical surveillance.

I wrote that occupational carcinogens will continue to be slowly identified through a combination of laboratory, epidemiological, and clinical means. In a previous editorial (1988;45:721-6) Magos described how there was no toxicity information available for most of the thousands of chemicals in commercial use. Too little is known about the health effects of industrial exposures to most chemicals to declare that the control of occupational carcinogens is now complete.

Relation between mercury and selenium in pituitary glands of dental staff

Sir,—Analyses of mercury concentration have shown high concentrations of mercury in the pituitary glands of dental staff.1

By contrast with mercury (Hg), selenium (Se) is an essential trace element. After simultaneous administration to rats, Hg and Se have been reported to be associated with a single protein fraction in the plasma. The atomic ratio of Hg to Se in this fraction was close to 1:1 for different doses of Hg2+ and SeO3.4 Coadministration of Hg and Se usually leads to increased whole body retention of both elements, especially of mercury. Se decreases the acute toxicity of inorganic mercury in short term animal studies and conversely Hg decreases the toxicity of Se.5 Lindh and Johansson reported mutually decreased toxicity when Hg and Se were given at the same time to rats.4 In histological sections Hg and Se were shown to be retained in a constant relation in the reticuloendothelial and proximal tubuli cells of the liver and kidneys, respectively. A protective mechanism through specific binding or chemical association was suggested.

Hg and Se concentrations have been analysed in four pituitary glands from dental personnel (table and figure). The extremely high correlation between Hg and Se at such high concentrations suggests some form of chemical association between the elements. In 1975 Kosta et al reported an approximate 1:1 atomic ratio in several organs, including the pituitary gland, from former mercury miners.6 This relation has not been further elucidated in man. The present data show an atomic ratio somewhat different from 1:1. As this result, however, is based on a small sample it does not warrant any conclusions as to the exact quantitative relation. The two dentists with the highest Hg and Se concentrations were old and had not been occupationally exposed for several years due to retirement and incapacitating neurological disorders. As hypothesised by Kosta et al, these data suggest coaccumulation of Hg and Se with long biological half times.

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Mercury and selenium concentrations in pituitary glands. Analyses performed with radiochemical neutron activation analysis (RNAA).

<table>
<thead>
<tr>
<th>Case</th>
<th>Hg (ng/g wet weight)</th>
<th>Se (ng/g wet weight)</th>
</tr>
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<tbody>
<tr>
<td>Dentists: 1 (M, 80)</td>
<td>4040</td>
<td>1820</td>
</tr>
<tr>
<td>2 (M, 50)</td>
<td>343</td>
<td>710</td>
</tr>
<tr>
<td>3 (M, 80)</td>
<td>3650</td>
<td>1700</td>
</tr>
<tr>
<td>Dental assistant: 4 (F, 67)</td>
<td>1280</td>
<td>970</td>
</tr>
</tbody>
</table>

Mercury and selenium concentrations in pituitary glands. Analyses performed with radiochemical neutron activation analysis (RNAA).