Recording HIV status on police computers

SIR,—J K Mason's editorial on recording people's HIV status on police computers cries out for correction.¹ I have been a police surgeon in London for nearly 17 years and have seen over 9000 people at the request of the police; most have been prisoners in custody. From time to time the custody officer says, "Doc, be careful, he's got HIV." I always ask how the officer knows, and the reply is invariably, "It's on the computer"; I have never heard of any validation. Equally often, the prisoner angrily denies being HIV positive or having provided the information.

I am the first to admit that people in custody do not always tell the truth and often change their stories. My experience, however, is so much at variance with the comfortable official line that Mason quotes with approval that I am not comforted by the editorial's conclusions. In fact, in the short report from which Mason quotes three of the seven people (57%) thought by the police to be HIV positive were not.²

I can see no reason for the police to record this information at all. In practice, the infection will be spread by only two activities, neither of which should occur in police stations. In my experience contamination of wounds with infected blood is exceedingly rare; all blood should be treated as infected anyway, whatever is on the computer. The label "HIV positive," whether true or not, serves only to increase tension in an already tense situation.

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1 Mason JK. Recording HIV status on police computers. *BMJ* 1992;304:995-6. (18 April.)

2 Sadler DW, Pounder DJ, Urquhart GED, Porter-Boveri M. Prevalence of HIV antibody in forensic cases. *BMJ* 1992;304:1027-8. (18 April.)

SIR,—As an occupational physician and a forensic medical examiner I share J K Mason's views on recording people's HIV status on police computers.¹ There is, however, room for improvement.

On several occasions I have seen the fact that a prisoner is positive for HIV recorded on the board commonly found in the charge rooms of the Metropolitan Police to facilitate the requirements of the Police and Criminal Evidence Act. This is to remind custody officers of the hazard, but the board may be read by anybody in the room; this may include other detainees as well as police staff. I regard this as too public; an indication outside the cell door would be more appropriate and would restrict the information to officers and others on a need to know basis.

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SIR,—As a doctor who has researched into HIV infection and the risk of exposure for health care staff, I was dismayed by J K Mason's editorial on recording people's HIV status on police computers.¹

Firstly, under international law people with HIV infection and AIDS have the right of liberty and security of person and the right to privacy.² Both of these rights are violated when police store information concerning a person's HIV status that may then be accessed. More outrageous is the fact that when such information is obtained from a third party the person concerned is not subsequently asked for his or her consent for it to be stored on a computer. This raises many ethical questions about how this has been allowed to occur without public debate.

In contrast, medical professional bodies insist on maintaining confidentiality and obtaining proper informed consent and counselling subjects before HIV testing. Only recently an ethics review committee failed to approve a protocol for a clinical trial that included HIV testing of volunteers, on the grounds that such information could expose the volunteers to potentially punitive actions from several sources.

I agree that the police are at risk, but how many documented cases exist of transmission of HIV to police compared with figures obtained for health care workers? Reports state that in a medical setting the maximum risk of acquiring HIV infection after sustaining a puncture wound or splash injury is 0.5% or 1 in 500 exposures, so in real terms the risk to a member of the police force must be marginal.³ To minimise their risk of exposure to HIV health care staff have adopted good laboratory safety practice. Could the police not minimise their risk by treating all people as potential sources of infection?

Mason states that knowing a person's HIV status allows police who may have been exposed to the virus to be counselled and given prophylaxis with zidovudine. Surely, it is adequate for a HIV test to be performed in cases in which exposure may have occurred, as is current practice among health care staff.

In conclusion, why are doctors going to such lengths to protect their patients' rights when police have access to sensitive personal data via a computer screen, and in some cases such access may be without the subject's consent or knowledge? Should such inconsistencies be tolerated in our society?

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1 Mason JK. Recording HIV status on police computers. *BMJ* 1992;304:995-6. (18 April.)

2 UK declaration of the rights of people with HIV and AIDS. *BMJ* 1991;303:541.

3 Seef LB, Wright EC, Zimmerman HJ, Alter HJ, Dietz AA, Selscher BF, et al. Type B hepatitis after needle-stick exposure. *Ann Intern Med* 1978;88:285.

SIR,—In the editorial on recording people's HIV status on police computers J K Mason assures us that "offenders' rights to privacy are protected . . . on current evidence. . . ." The author fails, however, to present any evidence that convincingly supports this assertion.

Firstly, Mason states that "the holding of sensitive personal data in police computers therefore seems to be tightly regulated." Can this be true when millions of inquiries are made each year? Random checks are made, we are told, but data on how often these are performed and how often access is found to have been gained without good reason are lacking. Are any sanctions taken against

officers found to be abusing the system? This crucial information is lacking.

The fact that most information is volunteered is beside the point; some is not and may be placed on record without the person's knowledge or consent. Furthermore, as David W Sadler and colleagues point out, this information may be in error.¹ To dismiss this worrying observation as immaterial because it was not based on the computer records is fallacious; the data in a computer are only as good as the data entered into it, which presumably are the same as those held by the police (albeit in an unspecified way) in Sadler and colleagues' article.

The issue of risk to police officers needs to be justified by some data. How common are events that might pose a risk of infection? Are these documented? Given that most such events must surely involve people who are not listed on the police computer or whose records cannot be accessed before the event occurs, how many such events does flagging records really prevent? Police officers need to treat all people as potentially infectious if they are to protect themselves. Mason suggests that informing a pathologist before a necropsy that a body may be HIV positive is comparable in terms of HIV testing. This is clearly not true: the subject of a necropsy has little to lose, whereas the suspect (not "offender" as stated) does.

Surely the real issue is whether the fact that the police may hold, and have readily available, medical information such as HIV status may prejudice the just and fair treatment of those with HIV infection. I fear that it might and find no evidence in Mason's article to reassure me.

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1 Mason JK. Recording HIV status on police computers. *BMJ* 1992;304:995-6. (18 April.)

2 Sadler DW, Pounder DJ, Urquhart GED, Porter-Boveri M. HIV antibody prevalence in forensic fatalities. *BMJ* 1992;304:1027-8. (18 April.)

Cholera: getting the message across

SIR,—Bernard Dixon appeals to medical microbiologists to redirect efforts towards simple measures to control cholera.¹ Similar thinking lay behind the innovative campaign against cholera led by the Centro de Investigación de Enfermedades Tropicales, an international centre for the epidemiology of tropical diseases in the faculty of medicine of the Universidad Autónoma de Guerrero in Acapulco, Mexico. Although the centre is one of the leading Latin American centres in immunoepidemiology and uses modern research techniques like the polymerase chain reaction, its approach to cholera included mobilising university students in a community based education programme and establishing a network of inexpensive diagnostic facilities throughout the state of Guerrero.

At the request of the university's rector, and without any additional funding, the centre mobilised and trained 23 000 university students, who travelled to hundreds of remote communities to give advice on preventing cholera and on low cost or no cost techniques to treat all diarrhoeal

diseases, including cholera. A recently infected victim who did not have any knowledge about cholera might take antacids or refrain from taking liquids—the worst possible management. At the beginning of the campaign few rural dwellers had any idea of what cholera was or how it was spread. Within a month some 600 000 people were contacted at their homes and more read the messages that the university placed in local newspapers, taxis, and buses. Over 50 000 primary school children were taught the basics of preventing cholera and of early treatment in a “child to community” scheme.

The campaign—“Not one more death from diarrhoea!”—also introduced an inexpensive and rapid diagnostic technique that identifies *Vibrio cholerae*. At the bedside of a person with suspected cholera a drop of faeces is placed in the well of a McArthur-style inverted slide and brought into focus at a magnification of $\times 400$ with a handheld plastic Eritrean public health programme microscope, based on the McArthur design.² While the characteristic darting movement of the vibrio is observed a drop of diluted antiserum—the same as is used to identify cholera serotypes in agglutination tests—is added to the well. The immediate and dramatic inhibition of movement is pathognomonic of *V. cholerae* serotype 01. With this technique cases were diagnosed in each of the seven regions of the state and later confirmed by conventional culture and biochemical examination.

Diagnosing and managing cholera do not require high tech or expensive solutions. They require the reorientation of thinking towards communities at risk.

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1 Dixon B. Basic science overlooked in fighting cholera. *BMJ* 1992;304:784. (21 March.)
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Work related disorders of the upper limb

SIR,—J M Porter and colleagues¹ seem to have completely overlooked the important message in N J Barton and colleagues' paper on occupational causes of disorders in the upper limb.²

So called “work related” disorders of the upper limb have been with us for many years, and with over 25 years' experience in occupational medicine I have seen my share of such cases, especially when I assessed “prescribed diseases” for the then Department of Health and Social Security. The apparent epidemic of work related musculoskeletal disorders may have resulted from irresponsible reporting of cases. There is no doubt that if the media report an influenza epidemic then every patient's sniff or cough is labelled influenza—similarly, nearly every ache or pain in muscles is considered to be work related.

Barton and colleagues rightly condemned vague diagnoses such as repetitive strain injury. If I apply creosote to my greenhouse I develop aching muscles because I have undertaken an unusual task, but the condition resolves quickly and completely. An acute frictional tenosynovitis is well recognised and easily diagnosable and can be directly related to forceful, twisting and frequently repeated movements. I have yet to see a real case of acute frictional tenosynovitis resulting from keyboard work.

Acute frictional tenosynovitis commonly arose in agricultural workers working with sugar beet

before sugar beet harvesters became commonplace. The combination of cold and wet conditions, the forceful repetitive movements, and the sudden seasonal introduction of the process contributed to this. The condition was indeed caused by the work process. Such is not the case with many other conditions well described by Barton and colleagues. A middle aged woman with osteoarthritic changes in the wrist joints may be predisposed to the carpal tunnel syndrome; work procedures may exacerbate this but do not cause it. An amateur footballer who returns to work after fracturing a tibia may experience pain in the leg because of prolonged standing at work, but the condition can hardly be described as work related.

I concur wholly with Barton and colleagues. The message is clear: it is vitally important to make a definite diagnosis in all of these cases at the earliest opportunity, when physical signs are present. We may have to identify and label a painful hand more accurately, based on actual pathological change. Only with such accuracy in diagnosis can we start correct treatment and consider the work conditions that might have to be modified. In the industries for which I work several tasks ought to result in musculoskeletal disorders but do not. This merely convinces me that the work is not the only factor that produces such disorders. The importance of ergonomic considerations is clear, but to embark on extensive and costly surveys of workplaces solely on the basis of possible work associated disease cannot be justified.

If we, as doctors, are unable to establish firm and specific diagnoses we would be prudent to abandon all terminology mentioning “work related” until further valid studies have indicated irrefutable aetiology.

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1 Porter JM, Buckle P, Robertson JC. Occupational causes of disorders in the upper limbs. *BMJ* 1992;304:842-3. (28 March.)
2 Barton NJ, Hooper G, Noble J, Steel WM. Occupational causes of disorders in the upper limb. *BMJ* 1992;304:309-11. (1 February.)

SIR,—In their review of occupational causes of disorders in the upper limb¹ N J Barton and colleagues refer to a report by this school of manufacturing and mechanical engineering.² I consider that the authors, by not correctly reporting the methods we used and by excluding the results of our analyses, seriously undervalue the importance of our work. I take issue with the statement that “the unsubstantiated assumption was made that because a symptom was present it was the work which had caused it.”

In our study 256 subjects at 18 industrial sites were randomly selected from volunteers who reported themselves as injured or free of injury (designated as cases or controls, respectively). Each was physically examined, and a biomechanical analysis was made of work activities. Data on work organisation and personal data were also recorded. The physical examinations and work observations were made on site by two examiners, who were blind both to whether the subjects were cases or controls and to each other's findings. Tasks were then analysed to determine whether there was an anatomical link between the task and the subject's site of injury. If there was the task's biomechanical variables were analysed. This was also done for non-injured subjects to see whether the presence or absence of these factors could correlate with the injured or non-injured status.

The biomechanical variables together with organisational factors were subjected to a discriminant analysis, with injury as the dependent variable. Results showed that there was no significant association between injury and cycle time or overtime. Hand deviation and limb extension,

force, and lack of job rotation were all significantly associated with injury ($p<0.0001$).

I consider that these findings do not warrant the use of the term “unsubstantiated assumption,” and I propose that identifying these work factors associated with injury can be vital to the successful design of work.

In 1746 Ramazini wrote a treatise on: “The diseases of tradesmen, to which they are subject by their particular callings. With the method of avoiding and treating them.” It is a sad reflection that so little progress has been made in the past 250 years on methods for preventing avoidable work related injuries.

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1 Barton NJ, Hooper G, Noble J, Steel WM. Occupational causes of diseases in the upper limb. *BMJ* 1992;304:309-11. (1 February.)
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Blood glucose and diabetic retinopathy

SIR,—Olaf Brinchmann-Hansen and colleagues' study suggests that long term lowering of blood glucose concentration retards the progression of diabetic retinopathy.¹ Multiple regression analysis of glycated haemoglobin (HbA_{1c}) concentration and the progression of diabetic retinopathy showed the predictive value of a single measurement of HbA_{1c}.²

We measured by radioimmunoassay the thromboxane B₂ and 6-ketoprostaglandin F_{1α} concentrations in platelet poor plasma from 55 diabetic patients (aged 11.6 (SD 1.6) years) and 33 controls (aged 10.5 (1.3) years). We calculated the ratio of thromboxane B₂ to 6-ketoprostaglandin F_{1α} (normal value <0.5). We divided the patients into four groups according to the duration of their diabetic disease. Early vascular lesions of diabetic retinopathy were sought by fluorescein angiography in 52 patients. Thirty one children did not have such lesions and 21 did.

The table shows our findings. By linear correlation we found positive correlations between HbA_{1c} and thromboxane B₂ concentrations ($r=0.58$, $p<0.05$) and the ratio of thromboxane B₂ to 6-ketoprostaglandin F_{1α} ($r=0.59$, $p<0.001$). There was a negative correlation between HbA_{1c} and 6-ketoprostaglandin F_{1α} concentrations ($r=-0.79$, $p<0.01$).

Our results suggest that the abnormality in the ratio of prostaglandins is a consequence not of the diabetic vascular lesion but of the poor diabetic

Ratio of thromboxane B₂ to 6-ketoprostaglandin F_{1α} concentrations and HbA_{1c} concentrations in children with diabetes and healthy children. Figures are means (SD)

	No of patients	Thromboxane B ₂ : 6-ketoprostaglandin F _{1α}	HbA _{1c} (%)
All patients	55	0.95 (0.22)	13.25 (2.77)**
Duration of diabetes (years):			
<1	12	0.93 (0.25)*	12.88 (4.06)**
1-5	19	0.91 (0.22)*	13.05 (2.36)**
6-10	15	1.19 (0.35)**	14.34 (2.83)**
>10	9	0.79 (0.14)	12.84 (2.26)**
Fluorescein angiography:			
Positive	21	0.87 (0.36)*	13.24 (2.51)**
Negative	31	0.83 (0.26)**	13.24 (2.59)**
Controls	33	0.42 (0.20)	6.20 (0.97)

* $p<0.01$, ** $p<0.001$ Compared with healthy controls.