Driving, glaucoma, and the law

Regular checks on medical fitness to drive needed

EDITOR,—Potamitis and colleagues draw attention to widespread ignorance about regulations concerning visual fields for driving.¹ Because drivers are required to pass a visual acuity test as part of their driving test, however, most are aware of visual acuity as a component of visual function and of its relevance to driving. This does not necessarily imply greater compliance with the regulation concerning visual acuity than with those concerning visual fields.

We recorded visual acuity (with glasses if worn) in consecutive patients attending the accident and emergency service at Manchester Royal Eye Hospital. Over one month 1426 patients were tested, of whom 177 had a visual acuity insufficient for legal driving. Most of these patients did not drive, but 42 still drove a car. Of these 42, 21 were "borderline" failures (6/12 in the better eye) but the remaining 21 would definitely have failed a standard numberplate reading test. All these patients had evidence of chronic visual loss, which was correctable by refraction in only some cases.

Clearly, patients with acute eye problems may have acute visual deterioration. Such deterioration, however, is almost invariably unilateral. For driving a car, acceptable vision is only required in one eye. There was no evidence to suggest that these data were invalidated by acute, temporary visual loss in these patients.

Potamitis and colleagues rightly recommend that greater information should be made available to drivers, both from the Driver and Vehicle Licensing Agency and from ophthalmologists. The key role of optometrists should also be made clear. The authors comment that patients need more information and imply that this will in itself improve driving safety by resulting in voluntary withdrawal on the part of unfit drivers, but this is merely an assumption. Do we not need regular checks on medical aspects of fitness to drive, even for people under retirement age?

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1 Potamitis T, Aggarwal RK, Tsaloumas M, McLaughlin J, O'Neill E. Driving, glaucoma, and the law. *BMJ* 1994;309:1057-8. (22 October.)

Patients need more information

EDITOR,—T Potamitis and colleagues found that 83% of patients with glaucoma who were drivers had not informed the Driver and Vehicle Licensing Agency of their diagnosis.¹ We have conducted a study in which the agency's criteria for visual fitness to drive were applied to a group of patients with glaucoma and to controls.

We examined 52 consecutive patients with glaucoma and 52 controls matched for age who had no evidence of sight threatening disease. A questionnaire on driving was administered to

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all subjects, after which their visual fields were documented in the manner specified by the Driver and Vehicle Licensing Agency and their visual acuity, with spectacle correction if spectacles were worn, was assessed. The drivers were then classified as "pass" or "fail." There was no significant difference in the proportion of subjects in each group who had continued to drive: 24 patients with glaucoma and 27 controls ($\chi^2=0.15$, $P=0.7$). Three of the patients with glaucoma who had continued to drive and none of the controls failed the visual tests.

The three patients with glaucoma who failed the tests did so because of reduced visual fields (two cases) and reduced visual acuity despite spectacle correction (one). These patients were aware of their visual loss. Two of them reported that they had not been warned against driving and that they had assumed that their ophthalmologist would inform them if they were unable to drive.

We found that three (12%) of the 24 patients with glaucoma who had continued to drive were doing so with inadequate visual function according to the criteria of the Driver and Vehicle Licensing Agency. The same proportion of both patients with glaucoma and controls were driving. In Britain there are 25 million people aged 40 or more, of whom 0.4% will have glaucoma.² If the figures from our study are applied to this total there are 5520 patients with glaucoma driving with inadequate visual function, as defined by the Driver and Vehicle Licensing Agency. This estimate is intended only as an illustration; at least in our group of subjects, the present system has not ensured that patients with glaucoma who drive have the appropriate visual function. More widespread information for patients and their ophthalmologists about the obligation to inform the Driver and Vehicle Licensing Agency about a diagnosis of glaucoma is needed to improve this situation.

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2 Hollows FC, Graham PA. Intraocular pressure, glaucoma and glaucoma suspects in a defined population. *Br J Ophthalmol* 1966;50:570-86.

Diabetic patients must also satisfy regulations

EDITOR,—Potamitis and colleagues draw attention to the low level of awareness among patients with glaucoma of their legal requirement to inform the Driver and Vehicle Licensing Agency of their condition.¹ The agency's regulations also require diabetic patients to have their binocular field of vision assessed if they have declared that they have undergone laser treatment. Treatment for proliferative retinopathy often requires the application of laser beams to all retinal areas outside the macula; this can have a severe effect on the patient's visual field. In one study 15 of 30 patients treated with panretinal photocoagulation failed the agency's regulations regarding visual fields.² Other investigators, however, have found that if a laser beam spot of smaller diameter is used less loss of visual field occurs.³

In her commentary on Potamitis and colleagues' report Margaret Hawkins states that patients with glaucoma at any stage must notify the Driver and Vehicle Licensing Agency.¹ She also declares that "failure to advise their patients correctly may render doctors liable to litigation." The same rules thus apply to diabetic patients who have undergone laser treatment. The only way accurately to advise patients with diabetes or glaucoma of their legal driving status (and thereby avoid possible litigation) is to plot their binocular visual fields. This test is not routinely done in the management of patients with either diabetes or glaucoma, and thus to implement it would pressurise an already overtaxed facility. It would also lead to an increased interval between routine tests of visual fields, which are required for the management of glaucoma. This might compromise the care of patients with glaucoma. Alternatively, new staff would have to be recruited to take on the increased workload.

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1 Potamitis T, Aggarwal RK, Tsaloumas M, McLaughlin J, O'Neill E. Driving, glaucoma, and the law. [With commentary by M Hawkins.] *BMJ* 1994;309:1057-8. (22 October.)
2 Buckley SA, Jenkins L, Benjamin L. Fields, DVLC and panretinal photocoagulation. *Eye* 1992;6:623-5.
3 Hulbert MFL, Vernon SA. Passing the DVLC field regulations following bilateral pan-retinal photocoagulation in diabetics. *Eye* 1992;6:456-60.

Visual field required for safe driving wrongly defined

EDITOR,—T Potamitis and colleagues highlight the fact that doctors and patients are often unaware of the driving standards set by the Driver and Vehicle Licensing Agency on the recommendation of the Royal College of Ophthalmologists.¹ The definition of the visual field required for safe driving given by Margaret Hawkins in her commentary on their paper, however, is inaccurate.¹ Her statement that the field should be at least 120° along the horizontal is correct, but the vertical component should measure 20° above and below the horizontal, not 20° above or below as stated.

The standard defined by the Royal College of

Ophthalmologists is more helpful and avoids confusion. This states:

The minimal visual field for safe driving is a field of vision of at least 120 degrees on the horizontal measured by the Goldmann perimeter using the III4e settings (or equivalent perimetry). In addition there should be no significant field defect in the binocular field which encroaches within 20 degrees of fixation either above or below the horizontal meridian. By these means homonymous or bitemporal defects which come close to fixation, whether hemianopic or quadrantanopic, are not accepted as safe for driving. Isolated scotomata represented in the binocular field near to the central fixation area may also be inconsistent with safe driving.²

This definition is incorporated in the forthcoming revised edition of the booklet *Medical Aspects of Fitness to Drive*³ and is thought to be the best interpretation of the standard as it is applied to individual patients.

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1 Potamitis T, Aggarwal RK, Tsaloumas M, Rene C, McLaughlin J, O'Neill E. Driving, glaucoma, and the law. *BMJ* 1994;309:1057-8. (22 October.)

2 Royal College of Ophthalmologists. *Definitions of the minimum field of vision for safe driving for group 1 ordinary drivers*. London: RCP, 1994.

3 Medical Commission on Accident Prevention. *Medical aspects of fitness to drive*. London: MCAP (in press).

Childhood antecedents of schizophrenia

Difference in antecedents between sexes may not be genuine

EDITOR,—In their study of the 1958 birth cohort of the national child development study D John Done and colleagues suggest that there may be significant differences between the sexes in the childhood antecedents of schizophrenia.¹ They base their conclusion on the finding that, despite a similar behavioural profile for both sexes at age 7, by age 11 the girls showed an appreciable tendency towards underreaction (social withdrawal and isolation), which was not seen in the boys.

There seem to be at least three possible ways to interpret this finding. Firstly, as the authors suggest, there may be a genuine difference in antecedent behavioural risk factors between the sexes, with social underreaction being confined mainly to preschizophrenic girls.

Secondly, the difference between the sexes at age 11 may be a transient phenomenon related to the earlier onset of behavioural underreaction in girls compared with boys. For example, Bettles and Walker have shown that negative psychotic symptoms such as social isolation and inappropriate affect are more prevalent in girls than boys at age 11-12.² By age 15-16, however, rate of negative symptoms in boys are equal to those in girls, and by late adolescence the rates have become higher in boys.

Finally, the symptoms of behavioural underreaction at age 11 may represent the first signs of an emerging schizophrenic illness. In this case one would predict that there would be more girls than boys among the subjects with the very earliest onset of schizophrenia in the study. Clearly, this would challenge the general assumption of an earlier onset of schizophrenia in male subjects.

An important message from Done and colleagues' study is that the behavioural antecedents of schizophrenia are not static but change with age. The question of whether the difference in antecedents between the sexes is genuine or transient needs to be resolved by study of the subjects at a third time point—namely, the 1974 follow up at age 16. Knowledge of the temporal relation between the onset of behavioural underreaction and the onset of schizophrenia would also

help to resolve the question of whether behavioural underreaction should be considered to be an antecedent risk factor or a prodrome of schizophrenia.

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1 Done JD, Crow TJ, Johnson EC, Sacker A. Childhood antecedents of schizophrenia and affective illness: social adjustment at ages 7 and 11. *BMJ* 1994;309:699-703. (17 September.)

2 Bettles BA, Walker E. Positive and negative symptoms in psychotic and other psychiatrically disturbed children. *J Child Psychol Psychiatry* 1987;28:555-68.

Environmental influences should not be ignored

EDITOR,—D John Done and colleagues claim that there is no strong evidence from twin, family, and adoption studies of environmental factors having a role in schizophrenia.¹ I am surprised that they are not aware of the many publications of Tienari and colleagues in Finland.^{2,3} Their nationwide studies evaluated the mental health of the families into which children whose biological parents had schizophrenia were adopted. Healthily functioning families had a significant protective effect on the prevalence of serious psychopathology and psychosis in the adopted children. Done and colleagues' dismissal of environmental contributions to schizophrenia may have led to their not considering evaluating the psychological health of the families in which they detected childhood antecedents of schizophrenia and affective illness.

The tendency to ignore environmental factors in conditions with a genetic component reminds me of the situation in Huntington's disease, known since 1872 to be a genetic condition. For years considerable resources have been directed at trying to identify the mechanism by which the (now located) gene causes its damage. This effort was not, however, balanced by any attempt to pursue the possibility of environmental and family factors having a role in the perpetuation of the disease. One hundred and thirty years after Huntington's discovery only a few professionals have begun to provide simple systematic information and counselling services for those at risk of Huntington's disease.⁴ Some of the results are striking: those counselled about their genetic vulnerability reduced up to fivefold the number of babies born with the gene.

I do not suggest that focusing on the influence of the family on those with a genetic predisposition to schizophrenia should have a similar aim or outcome. I wish, however, to make the point that when scientific evidence about environmental influences on schizophrenia is selectively ignored a similarly tragic situation is perpetuated whereby preventive psychotherapeutic interventions for these environmental factors are not even considered in the present, while we excessively emphasise the hope in the future from genetic and biological studies.

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1 Done DJ, Crow TJ, Johnstone EC, Sacker A. Childhood antecedents of schizophrenia and affective illness: social adjustment at ages 7 and 11. *BMJ* 1994;309:699-703. (17 September.)

2 Tienari P. Interaction between genetic vulnerability and family environment: the Finnish adoption family study of schizophrenia. *Acta Psychiatr Scand* 1991;84:460-5.

3 Tienari P. Interaction between genetic vulnerability and rearing environment. In: Werbart A, Cullberg J, eds. *Psychotherapy of schizophrenia: facilitating and obstructive factors*. Oslo: Scandinavian University Press, 1992:154-79.

4 Martindale BV, Yale R. Neglected opportunities for preventive medicine. *Lancet* 1993;341:634-6.

5 Harper PS, Tyler A, Smith S, Jones P, Newcombe RG, McBroom V. Decline in the predicted incidence of Huntington's chorea associated with systematic genetic counselling and family support. *Lancet* 1981;ii:411-3.

Conclusions not supported by data

EDITOR,—D John Done and colleagues' conclusions—that abnormalities of social adjustment are detectable in childhood in some people who later develop psychotic illness—seem to go well beyond the data presented.¹ It is difficult to see how comparing mean scores of groups at only two ages can reasonably lead to the conclusion that the rate of development of different components of the capacity for social interaction is an important determinant of the risk of psychosis and other psychiatric disorders in adulthood. The authors do not seem to have measured rates of change in individual children or to have attempted to correlate them with the risk of psychiatric disorders.

The authors' generalisation that "even at the age of 7 subjects who will later develop schizophrenic illness differ from schoolmates in the eyes of their teachers" is also questionable, especially as it implies that they are a recognisable group. The number of subjects who were later admitted to hospital with schizophrenia and for whom social adjustment scores were available at age 7 was fairly small (33). Also, although this group's mean scores were higher than those of the control group, little detail of the individual scores of children is provided.

The authors state that half the boys in this group were considered to be as deviant as the most deviant tenth of the school population. Even if all those who later developed schizophrenia had been among the most deviant tenth of 7 year olds, however, the risk of a child in the most deviant tenth developing schizophrenia by the age of 28 would have been only about 1 in 40 (using the authors' figure that 40 out of over 15 000 subjects in the national child development study had been admitted to a psychiatric hospital with schizophrenia by this age).

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Authors' reply

EDITOR,—John Temple suggests that our data do not permit us to discuss rates of development. We agree that extrapolating from two datapoints to derive a statement about rate is unwise. We do, however, have additional information—namely, that psychotic behaviour was documented in adult life. The children did not manifest psychotic symptoms, but these symptoms developed some 15 or more years later, rather than emerging spontaneously. In the development of cognition (for example, problem solving) qualitative changes occur at different ages, and thus the rate of development cannot be explained by a numerical statement. We suggest that the early abnormalities of social interaction that we reported represent a state in the abnormal development of the capacity for social interaction which culminates in psychotic behaviour. To regard the results simply as potential predictors or markers of a later pathological event seems blinkered. Delusions, hallucinations, and the other phenomena of psychosis have remained isolated from any acceptable theory of psychological functioning. As a result they remain unexplained phenomena. We suggest that an alternative approach, based on analysis of the pattern of development of these symptoms, may prove to be more illuminating.

Our other point concerns the practical relevance of our findings. The lifetime risk of schizophrenia is about 8 per 1000, and any behavioural measure