research for the first comprehensive national study of police surgeons.

Our researches confirm that intoxication is implicated in many call outs. However, it was often not the prime reason for the call out, typically occurring in combination with other factors, usually some form of physical injury. In cases focusing on fitness to be detained it was the injury rather than the intoxication that was the prime reason for the call out. In cases involving the assessment of fitness to be interviewed, mental state assumed a greater importance. Overall, injury and assault accounted directly for 36% of consultations and alcohol for 19%. This finding concurs with the personal study of Payne-James.[5]

With regard to the assessment of fitness for interview, a key issue is the need for any standardised approach to incorporate a record of the reasons for particular decisions. We believe that the present system within the Metropolitan Police District carries a potential for discrepancy between custody records, surgeons' notes, and records of a surgeon's attendance. Only a minority of police surgeons' decisions subsequently receive in depth scrutiny and our work found no reason to doubt the quality of the service, but this shortcoming should be addressed.

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The cervical spine in rheumatoid arthritis

EDITOR,—I agree with the subtitle of A K Agarwal and colleagues' editorial on the cervical spine in rheumatoid arthritis that it "needs careful assessment."[1] I have two comments to make from my own observations on this problem.

Naturally, larger amounts of anteroposterior subluxation carry a higher risk of neurological deficit. However, the rate of development of the subluxation may be even more important than its amount. Thus a relatively slight flexion injury to a radiologically normal cervical spine in a patient with rheumatoid arthritis can lead to a tetraplegia even if the subluxation is not very great. Thus all patients with rheumatoid arthritis having a general anaesthetic should have their cervical spines supported in a collar. In addition, a spurious "improvement" in anteroposterior subluxation may be reported by the unwary or inexperienced rheumatologist or radiologist,[2] although it really represents the dangerous addition of vertical descent to anteroposterior subluxation. The foramen magnum is in effect being threaded over a conical cervical odontoid process.

The interesting phenomenon of a mid or upper cervical cord lesion causing a suspended neurological deficit is also shown in the rheumatoid cervical spine.[3] For example, severe subaxial subluxation at the C3 or C4 level can lead to compression of the anterior spinal artery by the upper posterior edge of the body of C4. The cord can be compressed and the subsequent ischaemic changes in the cord can lead to changes as low as T1. This can lead, for example, to wasting of the small muscles of the hand. Generally this is accompanied by sensory symptoms and extensor plantar responses and can be confirmed by electromyography. It is an example of the value of clinical electromyography in rheumatological practice—in helping patient management and elucidating a previously unexplained clinical phenomenon.

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Low blood pressure and wellbeing

EDITOR,—The finding of Annika Rosengren and colleagues, that low blood pressure is associated with decreased psychological wellbeing,[1] is consistent with several studies recently published in the *BMJ* including our own.[2-4] These authors are right to point out that in cross sectional analyses it is difficult to make assumptions of causality.

The concept of a "hypotensive syndrome" implies that low blood pressure causes the associated physical and psychological symptoms, such as impaired psychological wellbeing or mild depression. This is clearly the assumption in those countries where the condition is widely "treated" with a variety of drugs.[5] An alternative hypothesis, which has considerable intuitive plausibility, needs to be refuted before this assumption can be justified. According to this hypothesis, depression or decreased psychological wellbeing causes low blood pressure by a variety of possible mechanisms, including diminished physical activity, altered diet, and decreased autonomic arousal. We tested this hypothesis by examining the case notes of 1046 consecutively admitted psychiatric patients, comparing the blood pressure on admission of depressed and non-depressed patients. If minor degrees of depression or diminished wellbeing lead to significantly lower blood pressure, then it should follow that depression that is sufficiently severe to lead to hospital admission will be associated with substantially reduced blood pressure.

The potentially confounding variables of age, alcohol consumption, and medication were controlled for in a logistic regression model, with the binary variable depression versus all other diagnoses as the dependent variable, and systolic and diastolic blood pressure as independent variables. Analyses were conducted separately for men and women. No significant differences were found between the two groups of patients for either systolic or diastolic blood pressure.

The finding that even severe depression does not lead to a lowering of blood pressure suggests that the mild depression and decreased wellbeing associated with low blood pressure in the studies referred to above is not due to a causal effect of the former on the latter, which strengthens the case for the existence of a hypotensive syndrome.

Systolic and diastolic blood pressure by psychiatric diagnosis, controlling for confounding variables

Primary diagnosis	Blood pressure (mm Hg)	
	Systolic	Diastolic
Men		
Depression (n=173)	137·5	86·1
All others (n=353)	136·9	87·1
Women		
Depression (n=264)	133·2	84·7
All others (n=256)	132·7	82·8

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Gangliosides in neurological diseases

EDITOR,—Albert Figueras and colleagues suggest that gangliosides can be withdrawn from the market because their efficacy is not clearly delineated.[1] The accompanying editorial, without scientific justification, calls for suspension of all human studies of possible efficacy of gangliosides because the substances are considered to be potentially immunogenic.[2] We do not agree with either statement.

The editorial was apparently written on the basis of inadequate information and some of the data, as outlined in this letter, may not have been available to Peter O Behan and B A G Haniffah.

Why continue studying the efficacy of gangliosides in humans? They could be continued on the basis of efficacy studies in experimental animals and existing data in humans. In experimental animals, gangliosides have been shown to be protective in a number of conditions including anoxic ischaemia, models of Parkinson's disease, central nervous system trauma, autoimmune neurological disease, and peripheral nerve injury. In humans, gangliosides have been shown to be potentially effective in various central and peripheral nervous system disorders. In addition, data presented at the second world congress of stroke in Washington, DC, last September showed efficacy of ganglioside treatment in acute stroke in two separate large multicentre studies.[3]

With regard to immunogenicity, the only paper to describe rabbits immunised with brain gangliosides which later developed a "ganglioside syndrome" was published by Nagai *et al* in 1976.[4] The purity of the ganglioside preparation used in this study has, however, never been established, and this work has not been reproduced in other laboratories.

An abstract recently presented by R K Yu *et al* at the 22nd annual meeting of the Society for Neuroscience in Anaheim, California, indicated no clear association of treatment with monosialoganglioside (G_{m1}) and the development of antibodies to it in 418 samples from human subjects receiving parenteral gangliosides. In addition, serum antibodies to gangliosides, particularly monosialoganglioside, have been reported in patients with a variety of neurological conditions and those without neurological disease. These antibodies are found in patients who have never received parenteral gangliosides. The association of these antibodies with any human diseases is currently unclear. A strong case can be made for the antibodies being consequential to neurological disease,[5] and there is no correlation between the presence of these