practice should include the measurement of serum levels and the avoidance of polypharmacy; and drug firms should cease the manufacture of combined tablets of phenobarbitone and of anticonvulsants whose major action is to increase the serum levels of other more active preparations.

The debate has been narrowed to epilepsy and the private motorist. Why, one may ask, is a person with a history of epilepsy, perhaps in the distant past, provided with a special licence and thus discriminated against, whereas psychiatric illness, visual defects, or cardiovascular diseases before 70 years of age do not affect a person's fitness to hold an ordinary licence? Should the same regulations govern motor boats, light aircraft, or gliders? What obligation has a doctor to give correct and early advice to a person who has had a fit after 3 years of age before he unwittingly seeks a career driving public service or heavy goods vehicles or undertakes work dependent on long hours at the wheel?

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¹ Raffle, A, ed, *Medical Aspects of Fitness to Drive*, 3rd edn. London, Medical Commission on Accident Prevention, 1976.

* * * The first sentence of our article implies that doctors are expected to assess and advise patients on the effects of any disability on driving in the light of the new regulations. Like all the laws of the land, the driving licence regulations have to be applied automatically, but when assessment of individual cases is a matter for expert medical judgment specialist advice will be obtained. In our article the current regulations were set out in detail and the interpretation and application of the regulations were outlined in a way that (it was hoped) would satisfy those closely concerned. It was not, of course, suggested that advice to alter treatment for a recurrent fit would be casual, and we agree with Dr Critchley that measurement of serum drug levels and avoidance of combined tablets and of polypharmacy are important in the modern management of epilepsy. These current views about methods of improving seizure control are well worth emphasising, though one could hardly expect to encompass all possible aspects within the constraints of a leading article focusing on fitness to drive.

Dr Critchley is not correct in saying that psychiatric illness, visual defects, or cardiovascular diseases before 70 years of age do not affect a person's fitness to hold an ordinary licence. In fact, licence holders are required by law to inform the Licensing Centre as soon as they become aware that they are suffering from any "relevant" disability (that is, one likely to cause the driving of a vehicle to be a source of danger to the public), or a "prospective" disability which, by virtue of its intermittent or progressive nature or otherwise, may in time become a relevant disability. In such cases, where appropriate, the licence is revoked or replaced by a more limited licence.—Ed, *BMJ*.

Meditation or methyl dopa?

SIR,—A key statement in your leading article (12 June, p 1421) on this subject requires considerable qualification if it is not to be grossly misleading.

You state that "a well-conducted and controlled clinical trial of antihypertensive treatment in the United States in men with diastolic blood pressures between 90 and 115 mm Hg showed that the death rate from hypertension was reduced significantly and the overall incidence of complications correspondingly diminished." It is essential to note three points about the important trial¹ to which you refer: (a) definite evidence of benefit was found only in those with a diastolic pressure of 105 mm Hg or over; (b) the diastolic pressure was recorded at the fifth phase, not at the fourth phase as is customary in Britain—the fourth phase is on average at least 5 mm Hg higher; and (c) the blood pressures on which the study was based were not the patients' "casual" pressures, which would probably have been at least 5 mm Hg higher.

Thus it is probably true to say that there is as yet no conclusive evidence of benefit from antihypertensive treatment in the case of middle-aged men with "casual" fourth-phase diastolic pressures below 115 mm Hg.²

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¹ Veterans Administration Cooperative Study Group, *Journal of the American Medical Association*, 1970, **213**, 1143.

² Short, D, *British Heart Journal*, 1975, **37**, 893.

Nodular vasculitis of tuberculous origin in an elderly woman

SIR,—Classical descriptions of the Bazin form of erythema induratum emphasise its preponderance in young women. However, at least five cases of Bazin's disease have been reported in women aged 51-57.^{1,2}

Nodular vasculitis, on the contrary, affects chiefly women aged 30-60.³ The syndrome is a hypersensitivity reaction to many causes, of which tuberculosis is one, and in a number of cases the exact cause of this clinical entity remains obscure. In some such cases the response to prolonged antituberculosis therapy provides the solution.² Since we have diagnosed nodular vasculitis of tuberculous origin in a woman aged 84 we wish to draw attention to the fact that at any age tuberculosis has still to be considered as a cause of chronic nodular lesions of the legs.

An 84-year-old woman had been well until two months previously, when she noticed the eruption of tender nodules on both her legs. There were no other symptoms. On examination multiple tender, subcutaneous, red and purplish nodules without ulceration were present on the anterior aspects of the legs below the knees, the inner aspect of the right ankle, and the dorsa of the feet. The nodules ranged in size from 1 to 2 cm, some of them forming plaques. No other abnormalities were found.

The urine was normal. The haematocrit value was 40%. The white cell count was $8.2 \times 10^9/l$ ($8200/mm^3$) with a normal differential count and the platelet count was $143 \times 10^9/l$ ($143,000/mm^3$). The ESR was 55 mm in 1 hour (Westergren). C-reactive protein was present (— + +). The antistreptolysin titre was 125 Todd units. Rheumatoid factor, antinuclear antibodies, and LE cells were not found. The total plasma protein was 87 g/l with albumin 48 g/l and globulin 39 g/l, with an increase in the gamma component (34%). A skin tuberculin test (OT 1:10 000) was positive (— + + +). No mycobacteria or other pathogens were found in the sputum or urine. A throat culture was negative for β -haemolytic streptococci. X-ray and

tomography of the chest showed no abnormalities. Biopsy of a skin nodule revealed typical tubercle formation with caseation. Treatment with streptomycin, isoniazid, and ethambutol was initiated. During the next 20 days some of the lesions became smaller and a few healed, leaving atrophy. The patient was then discharged to continue the same treatment. Two months later no skin lesions were present and after eight months the patient was well and the plasma γ -globulin level and the ESR were normal.

Nodular vasculitis among the middle-aged or elderly is not often attributed to tuberculosis. The tuberculous origin of nodular vasculitis is too seldom considered a possibility in the differential diagnosis of leg nodules in the elderly. Personal and family history and radiological, histological, and bacteriological investigations may at times fail to disclose any evidence of tuberculosis.² In unexplained cases of nodular vasculitis a strongly positive tuberculin reaction¹ combined with an increase of γ -globulin concentration³ should suggest the possibility of tuberculosis and the response to adequate and prolonged antituberculosis therapy^{2,4,5} may confirm this diagnosis.³

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¹ Montgomery, H, O'Leary, P A, and Barker, N W, *Collected Papers of the Mayo Clinic*, 1944, **36**, 461.

² Feiwel, M, and Munro, D D, *British Medical Journal*, 1965, **2**, 1109.

³ Rook, A, Wilkinson, D S, and Ebling, F J G, *Textbook of Dermatology*, vol 1, p 675. Oxford and Edinburgh, Blackwell Scientific, 1969.

⁴ Stegmaier, O, *Archives of Dermatology*, 1959, **80**, 350.

⁵ *British Medical Journal*, 1963, **2**, 981.

⁶ Benians, R G, *British Medical Journal*, 1963, **2**, 1267.

Natural history of chronic bronchitis

SIR,—In your leading article on this subject (29 May, p 1297) you start with the hypothesis that mucopurulent bronchitis is a stage on the road to obstructive bronchitis, with a reference¹ in which I can find no evidence for this claim. You follow by stating that infection is one of the factors leading to the "development and progression" of bronchitis, with a reference to the late Professor J R May's book on the chemotherapy of chronic bronchitis² in which I can find no evidence to support this statement. Indeed, the preface only claims that "it may be that long continued effective suppression of the relevant pathogenic bacteria will ultimately be shown to delay the progressive deterioration of pulmonary function to which most bronchitics are subject." Finally you state, without any references, that the bronchitic's "respiratory function invariably worsens" with mucopurulent episodes.

In a leading article in 1973³ you made the same claim about the effects of respiratory infections on lung function in bronchitis and I wrote⁴ to ask you to justify it, quoting publications which showed that they very rarely have any permanent effect. A further paper confirming this evidence has recently been published.⁵ And surely the finding by Johnston and his colleagues,⁶ which you quote, that antibiotic treatment had no effect on the decline in forced expiratory volume makes it improbable that infection itself has much effect on it.