

He shows evidence of severe peripheral neuritis affecting both legs with marked wasting of the calves and absent sensation to pin-prick and cotton wool over the feet and legs. Plantar flexion and dorsiflexion at the left ankle are completely lost, and the interphalangeal joint of the left great toe is destroyed by neuropathic arthropathy. His feet are extremely painful and he has troublesome trophic ulcers. In addition he has gross impairment of muscle power in the proximal muscle groups of the lower limbs.<sup>1</sup>

This case would appear to be a further example of peripheral neuropathy and myopathy associated with pituitary growth hormone excess.—I am, etc.,

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<sup>1</sup> Mastalgia, F. L., Barwick, D. D., and Hall, R., *Lancet*, 1970, 2, 907.

### Amantadine-induced Aggressiveness

SIR,—The antiparkinsonian effect of amantadine was shown in 1969 by Schwab<sup>1</sup> and since then confirmed by several other authors.<sup>2</sup> The behavioural and biochemical effects of this drug in experimental animals<sup>3</sup> suggested to Vale *et al.*<sup>4</sup> the possibility of using it as an antidepressant; a positive effect was claimed by them in depressed patients.

On the basis of this finding, we have treated four aged depressed patients with amantadine for at least three weeks. The dose regimen was started with 100 mg/day and then slowly increased to 300 mg/day within 10 days.

We did not observe any positive effect on mood but, conversely, noticed a progressive increase of motor restlessness, anxiety, and sudden bursts of violent, aggressive behaviour, with hostile attacks upon the ward's staff. The hostility and aggressiveness became evident usually after 10-15 days of treatment, when amantadine plasma levels were about 800 ng/ml, and tended to disappear within 4-6 days after discontinuation of treatment. A detailed description of these results will be published later.

Since we could not find any data in the literature on amantadine-induced aggressive behaviour in depressed patients during observations have been made by others.

The appearance of aggressive and hostile behaviour in depressed patients during amantadine treatment is also of particular interest in view of a similar phenomenon recently observed by Goodwin *et al.*<sup>5</sup> in depressed patients treated with high doses of L-dopa. The same authors suggest that the aggressiveness seen in humans after L-dopa may be related to an increased catecholamine synthesis. Considering the similarity between the antiparkinsonian and pharmacological effects of L-dopa and amantadine, it is possible to hypothesize the existence of an analogous mechanism to explain the clinical results obtained with our patients.—We are, etc.,

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<sup>1</sup> Schwab, R. S., England, A. C. Jr., Poskanzer, D. C., and Young, R. R., *Journal of the American Medical Association*, 1969, 208, 1168.  
<sup>2</sup> Parkes, J. D., Zilkha, K. J., Calver, D. M., and Knill-Jones, R. P., *Lancet*, 1970, 1, 259.

<sup>3</sup> Fibiger, H. C., Fox, M., McGeer, E. G., and McGeer, P. L., *Journal of Pharmacy and Pharmacology*, 1971, 23, 724.  
<sup>4</sup> Vale, S., Espeiel, M. A., and Dominguez, J. C., *Lancet*, 1971, 2, 437.  
<sup>5</sup> Goodwin, F. K., Murphy, D. L., Brodie, H. K., and Bunney, W. E., *Biological Psychiatry*, 1970, 2, 341.

### Varieties of Smoke

SIR,—I recently re-read your leading article (18 September 1971, p. 656) which questions the value of tar assays. The views expressed were somewhat disturbing and persuade me to raise several points.

(1) Measurement of the tar delivered at the proximal end of the butt by a particular brand of cigarette is a simple, reliable procedure.<sup>1</sup> It is used in America, Canada, and Australia—at least by the tobacco industry and by private or public health organizations. Interpretation is also simple—a cigarette delivers so many milligrams of tar to the smoker's lips if smoked in standard fashion. From tests performed in the chemistry department at Monash University a smoker's "tar table" has been produced, and from it a comparison between brands is possible.

As you say, smoking habits vary, but a packet of Australian cigarettes near the top of this table delivers 100 mg of tar and 6 mg of nicotine; a packet of cigarettes from the bottom of the table delivers 520 mg of tar and 32 mg of nicotine. These are gross differences. The fact that more frequent puffing may occur could hardly make up a five-fold difference in tar and nicotine delivery.

(2) The smoking dogs of Hammond and Auerbach<sup>2</sup> demonstrated that lung damage is related quite directly to regular tar intake. Mortality rates in filter smokers, as recorded by Bross,<sup>3</sup> suggest the same.

If low-tar cigarettes were available on the British market (you will only find them by testing), some smokers who used them would notice diminution of cough and return of taste (personal observation).

(3) The people who are going to die soon from smoking are the addicts of 20, 30, or 40 years. If they cannot give up, they can smoke pipes, cigars or low-tar cigarettes and thereby lower their risk. We should not abandon the present generation of breadwinners. None of this suggests low-tar cigarettes are "safe," they are merely less risky.

(4) It is not difficult to differentiate between relative risks in a public campaign. It has been done in the U.S.A. and Canada, and our own experience in the State of Victoria is also of some interest.

We can reasonably claim to have generated a demand for low-tar cigarettes among addicted smokers over the past three years. Since per capita cigarette consumption has not changed much, this is presumed to be at the expense of high-tar brands. We have seen no signs that this campaign has encouraged or unduly reassured smokers, although some have worked down the ladder and eventually given up. Two low-tar brands are now among the more heavily promoted brands in this State. (The tobacco industry is not notably responsive to pressure of public health opinion, but is acutely sensitive to the demands of the market). I think this campaign is a useful gesture to the smoker with a high and immediate risk. It need not detract from the overall anti-smoking message.

If your editorial advice is taken, you will never see low-tar cigarettes on the market, never discover the benefit of this to the coughing, tasteless smoker, and never gain material for the necessary epidemiological study which is needed to determine just *how much* a lower daily tar intake lowers smoking risk.—I am, etc.,

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<sup>1</sup> Moore, G. E., Bross, I., Shamberger, R., and Bock, F. G., *Cancer*, 1967, 20, 323.  
<sup>2</sup> Hammond, E. C., *et al.*, *Archives of Environmental Health*, 1970, 21, 740.  
<sup>3</sup> Bross, I. D. J., and Gibson, R., *American Journal of Public Health*, 1968, 58, 1396.

### Presenile Dementia

SIR,—Unfortunately, the facts presented in our paper (29 April, p. 249) do not help to answer many of the questions posed by your correspondents.

The mean age of the 106 patients investigated for presumed dementia was 61 years; the youngest was aged 34, and the oldest was aged 78. The outcome of investigation in relation to the age at which it was carried out is shown below.

Age	<50	50-59	60-69	70+
No. of patients	8	40	43	15
Not demented (uncertain in parentheses)	3 (2)	7 (3)	3 (2)	2
Diagnosis of Dementia				
Space occupying mass		2	5	1
Arteriosclerotic		1	5	2
Alcoholic		4	2	
Normal pressure hydrocephalus		1	3	1
Creutzfeldt - Jakob disease		1	1	1
Huntington's chorea	2	1		
Post - traumatic cerebral atrophy		1		
Postsubarachnoid haemorrhage	1			
Limbic encephalitis			1	
Cerebral atrophy		19	21	8

Our data do not help define "an age criterion" for selection of demented patients for full investigation asked for by Dr. P. K. Bridges (20 May, p. 465). This decision remains a matter of judgement in which chronological age is one of many factors taken into consideration. For this reason the term "presenile" (which has no age definition) was used for it conveys the same concept of biological age as does the indefinable term adult life.

To answer Dr. H. M. Hodgkinson (13 May, p. 404) on "the value of neurological investigation at any given age in middle life" would require a much larger number of patients, and to complete the picture one would wish to know the cause of dementia in those elderly patients who are not fully investigated in life.

Dr. Hodgkinson also asks for further information on those patients who were judged to be amenable to (that is, capable of) treatment. We felt it would be useful to give some conservative estimate, and the 15% figure quoted included the three patients with benign intracranial tumours,