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Preference is given to the letters commenting on contributions published recently in the *JRSM*. They should not exceed 300 words and should be typed double spaced.

Autopsy and medical education

I have read with interest the paper by Charlton (April 1994 *JRSM*, pp 232–6). However, I have to take issue with the statement that an autopsy can be 'an unpleasant and unrewarding examination'.

As one who has performed autopsies in many odd parts of the world where the conditions of both environment and material presented are far from ideal, I cannot say that this was apparent. One is faced with a problem to be solved and this can be an intellectual challenge.

I think the statement begs a fundamental lack of appreciation of the role of a morbid anatomist. If one does not like to perform autopsies then it is not the specialty to be chosen, as it is a rather fundamental part of the job description. With regard to the unrewarding aspect this is again an attitude. No two autopsies are the same, and even in the 'normal' it must be a collection of data to observe the changing patterns of disease, or the early stages of disease formation.

Possibly when I was fired with enthusiasm for autopsies by Professor Austin Gresham he had an attitude that you conducted the autopsy in an objective manner, and at the end reviewed the findings to arrive at a reasoned conclusion.

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Epidemiology of oesophageal cancer

With reference to the paper by Drs Macfarlane and Boyle (June 1994 *JRSM*, pp 334–7). They reviewed the evidence that oesophageal cancer is linked to alcohol misuse, smoking and dietary deficiencies, while the consumption of fresh fruit and vegetables appears to reduce the risk. I write to propose an explanation for these observations.

Alcohol and dietary deficiencies appear to be injurious to the oesophageal mucosa. Alcoholics have a high incidence of oesophagitis[1] while in animal models, deficiencies of zinc and riboflavin result in mucosal hyperplasia[2,3]. In man, alcoholism and poor nutrition are commonly associated and it is proposed that the resultant oesophagitis constitutes a pre-cancerous lesion. Inflammation of the oesophageal mucosa is associated with the excessive production of pro-inflammatory prostaglandin E_2 (PGE_2) and PGE_2 seems to be a carcinostimulant in the oesophagus[4]. Further, studies from areas with a high incidence of oesophageal cancer have consistently found a high prevalance of chronic oesophagitis[5]. Cancer onset in longstanding oesophagitis may thus occur during continued alcohol and tobacco use since these agents contain pre-formed and pre-cursor nitrosamines. Nitrosamines are potent oesophagotropic carcinogens, hence repeated oesophageal exposure may provoke malignant degeneration.

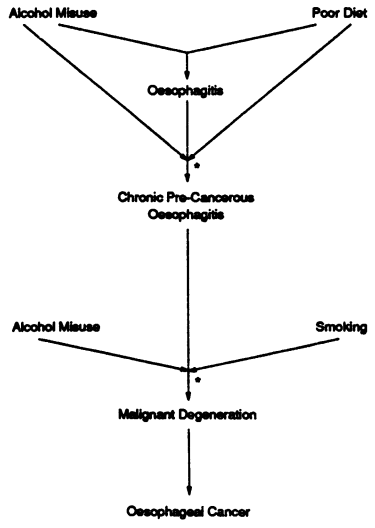

Figure 1 **A flow diagram showing the possibility of developing oesophageal cancer. Primary prevention of oesophageal cancer through sensible drinking and dietary factors (vitamins C and E, riboflavin and zinc)**

Fruit and vegetables may be protective for several reasons. First, their consumption would help to prevent the deficiency hyperplasia. Secondly, vitamin C can inhibit nitrosamine formation from ingested pre-cursors[6]. Thirdly, antioxidant vitamin E inhibits the formation of free radicals and retards tumour growth in animals[7].

Oesophageal cancer may therefore develop after many years of alcohol misuse, smoking and poor diet through the intermediate of chronic oesophagitis. This possibility is illustrated in Figure 1.

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