The author replies below

Dr James has every reason to be confused - the words 'centripetal' (ie towards the centre) and 'centrifugal' (away from the centre) have been transposed! I am grateful to him for drawing my attention to this error. With reference to his second point, I entirely accept that the descriptions by Etienne and Sarpi of the venous valves preceded Fabricius' own account. However, since neither author made use of mechanical analogy in his description they did not fall within the scope of this particular article.

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Chronic snorers and sleep apnoea

The article by D E Golding-Wood and his team (June 1990 *JRSM*, p 363) on the assessment of chronic snorers and the incidence of sleep apnoea makes one wonder if there is not a relationship between apnoea and the so-called degenerative diseases of the CNS, eg senile dementia and Alzheimer's disease. In the course of clinical practice I came across several patients whose dementia seemed to be closely related to the experience of a dental anaesthetic. In sleep apnoea, when the PaO_2 becomes sufficiently low, the patient awakes, sometimes in ventricular arrhythmia. But perhaps by then oxygen lack may have already 'persuaded' a senescent cell complex in the cerebral cortex to become nonfunctional. If sleep apnoea episodes become part of a patient's nocturnal existence cortical damage could become profound. Perhaps the prevention of sleep apnoea should become a matter of some urgency in a population in which nearly 40% are chronic snorers.

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The death of Mozart

I was intrigued by the letter to you from Dr Banerjee (June 1990 *JRSM*, p 414) with regard to Mozart's skull in the Mozarteum.

I had always understood that Mozart had died at an early age and was buried in an unmarked pauper's grave on a grey and rainy day, with not a friendly soul present in attendance. This scene was poignantly recreated in the film *Amadeus*.

Mozart presumably took his adult skull to the grave with him. I wonder which of Mozart's skulls is in the Mozarteum, the one he had in childhood, that as a youngster, or that as an adolescent?

Is it perhaps as substantial as King Charles' head in Dickens' *David Copperfield*?

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The death of Schubert

In a book on *Music & Medicine*, reviewed by V C Medvei (July 1990 *JRSM*, p 480), Dr J O'Shea drew attention to the diagnostic uncertainties of many diseases, including syphilis, in previous centuries, and to indiscriminate treatments with laxatives (herbal and mercurial), repeated bleedings, etc. which often added iatrogenic disorders to the patients' ills.

The tragic death of Franz Schubert (1797-1828) appears to be of particular topical interest. In a recent biography of Schubert, not mentioned in O'Shea's book, Marek¹ describes his life in Vienna, in a circle of mainly aristocratic *bon vivants*, some of which had homosexual leanings. This might have been the case of Franz von Schober (1796-1882) a dilettante, who befriended Schubert; and extended to him hospitality in his home (rather the home of his mother; von Schober remained unmarried till he was about 58 years of age). When Schubert stayed with von Schober from 1821 to 1823, he became seriously ill; syphilis was suspected, and he was treated, *inter alia*, with large doses of mercurial preparations. The health of Schubert sufficiently improved to allow him to perform in the provinces in the company of another friend, the singer, Johann Michael Vogt.

However, a subsequent spell of von Schober's hospitality resulted in deterioration of Schubert's health. He went to live with his brother, in September 1828, developed abdominal pain, intractable diarrhoea, etc. in October, and died on the 19 November 1828, when only 31 years old. His friend, von Schober, lived to the advanced age of 86 years. Schubert's death has been variously attributed to typhus, typhoid fever, fish poisoning, and others. The rapid course of his stomach disorder, whatever its causes, suggests low immune status.

The recent outbreak of AIDS among the modern gay communities, focused attention on the importance of the immune system, and on its vulnerability to a variety of toxic factors, whether exogenous, or formed endogenously, under specific conditions^{2,3}. It is usually the passive partners (the catamites), who are the main sufferers of AIDS. The poor, defenceless Schubert, preoccupied with his musical creation, might possibly have died of AIDS. How to prove, or disprove this hypothesis?

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Diet and large bowel cancer

I have read Dr Bingham's editorial (July 1990 *JRSM*, p 420) with great interest. In 1981 J L Thornton put forward the hypothesis in the *Lancet* that a high colonic pH promoted the degradation by colonic bacteria of bile acids and cholesterol into (CO) carcinogens¹. He also mentioned that a high fibre diet lowered the colonic pH as a result of bacterial digestion of fibre into short chain fatty acids.

In 1982 I reported a lower faecal pH in a population of railway workers in North India than in a similar group from South India². The North Indian diet of unrefined wheat, pulses and other legumes has a high fibre content and is accompanied by a higher intake of short chain fatty acids in the form of yoghurt, white cheese and ghee.

The South Indian diet of rice, tapioca, ragi and tamarind powder is low in fibre and also low in milk and fermented milk products.

Table 1. Mean faecal pH values ($\pm$ SD)

Colorectal cancer	
60 patients	8.6 $\pm$ 1.68*
Controls	
60 railway employees	7.0 $\pm$ 1.57
60 from general population	6.5 $\pm$ 1.56

* $P < 0.001$ compared with each group of controls

Table 2. Distribution of faecal pH values

	pH									
	5.0-6.0	6.1-6.5	6.6-7.0	7.1-7.5	7.6-8.0	8.1-8.5	8.6-9.0	9.1-9.6	9.6-10.0	
Colorectal cancer										
60 patients	0	0	4	6	6	7	24	10	3	
Controls										
60 railway employees	10	8	18	7	3	5	7	2	0	
60 from general population	18	12	7	8	6	5	2	2	0	

χ^2 test: cancer patients v controls (grouped)
(test for independence applied by group intervals to obtain theoretical frequencies of not less than five)
 $\chi^2 = 29.88$, $P < 0.001$

The incidence of colorectal cancer is higher in South Indians than in North Indians^{3,4} which would support the hypothesis propounded by Thornton in relation to colonic pH. I have done a further study of faecal pH comparing 60 patients with colorectal cancer from the Cancer Registry of the South-eastern Railway Calcutta with 60 matched controls from railway employees and 60 matched controls from the general population. Twenty-four hour faecal samples were collected and pH measurements were made after thorough mixing.

The mean pH measurements are shown in Table 1. To avoid the problems associated with mean values of logarithmic measurements the distribution of the different pH ranges is shown in Table 2.

It will be seen that the pH value of the faeces was significantly higher in the colorectal cancer patients than in both groups of controls.

An alkaline milieu renders mucus more fluid, and in that state it escapes more easily from the cell⁵. Loss of mucus from cells is reported to result in hyperplasia, metaplasia, cell atypia and a 40-fold increase in mitotic activity^{6,7}.

There is supporting evidence that a high pH may be a prelude to cancer in other situations. Exposure to calcium hydroxide results in carcinoma in situ changes in the cheek pouches and oesophagus of hamsters^{8,9} and carcinoma of the mouth is associated with the chewing of pan-containing lime in India. The increased risk of carcinoma of the colon after cholecystectomy^{10,11} may be due to the increased alkalinity of the bile resulting from failure of the absorption of bicarbonate which normally takes place in the gall bladder.

In conclusion, the finding of higher pH values in patients with colorectal cancer and the fact that these can be affected by diet adds increasing weight to the importance of diet in the aetiology of this condition.

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Spontaneous rupture of renal artery aneurysm

We read with interest the recent case report by Cheng *et al.* (July 1990 *JRSM*, p 464) of spontaneous rupture of a renal artery aneurysm but would like to raise some additional points about this increasingly diagnosed condition.

Firstly, we would disagree with their statement that renal artery aneurysm alone is seldom associated with hypertension. The association of proven renovascular hypertension and renal artery aneurysm without renal artery stenosis is well recorded in the literature and such cases stand a good chance of becoming normotensive after surgical correction of the aneurysm¹. The cause of this associated hypertension is not clear but kinking of the artery, distal embolization or haemodynamic effects due to interference with pulsatility have been suggested as possible reasons². We have recently treated a case of solitary renal artery aneurysm, without associated stenosis, in which the changes produced on Captopril-renography were identical to those of classical renal artery stenosis³ and many authors have shown abnormal renal vein renin levels in these patients. The key is the correct diagnosis of the renovascular nature of the hypertension preoperatively as associated hypertension from other causes can not be expected to improve. In the case described, the authors did not report the presence of any stenotic lesion, but also unfortunately failed to tell us whether the patient was hypertensive or normotensive before nephrectomy was performed.

The indications for surgery in renal artery aneurysm remain controversial. Follow-up and post mortem studies have failed to document a significant risk of rupture in most renal artery aneurysms⁴. However, there are many reported cases of rupture in pregnant women and the literature would suggest that