Geriatric Problems

SIR,—Dr. F. Allen Binks (3 February, p. 269) is to be congratulated for seeing the wood from the trees. In geriatrics this is not always easy. His clearly reasoned paper demonstrates the need for one of two things in our society of increasingly frail and disabled old people. Either the geriatric service must have a very much larger share of hospital resources—doctors, nurses, therapists, social workers, beds, and equipment—or else other clinicians will have to learn something of geriatric techniques and deal with more of their "geriatric problems."

If our colleagues prefer the former, geriatricians can cope, given proper facilities. If the latter, clinicians may be encouraged by Dr. Binks's admirable demonstration that "geriatric problems" are human and absorbing aspects of medicine, when approached without prejudice.—I am, etc.,

JOHN C. BROCKLEHURST.
The Lennard Hospital,
Bromley, Kent.

Haemodynamics in Cardiac Transplantation

SIR,—Mr. W. J. Dempster and others (20 January, p. 177) mention the use of a mechanical heart instead of cardiac transplantation as being possibly more practical. Experience with just such a device connected to an analogue model of the circulation, reported elsewhere,[1 2] led me to put it into abeyance until further advance in haemodynamics had been achieved. The ultimate haemodynamic problem appears to be analogous to that of matching "impedance," a concept derived from the study of electrical circuits and applied to problems of the circulation of the blood by McDonald[3 4] and Womersley[5 6] and others.

The first essential in matching for impedance is to match for frequency. If this is applicable to the vascular system a new heart should be selected that beats naturally at the same rate as the old. Secondly, assuming that the second heart sound is the echo of the first heart sound, triggered by the aortic standing wave first described by Hamilton,[7] the natural interval between first and second heart sounds of the new heart should be identical with that of the old. Thirdly, the stroke output should be of sufficient volume to ensure adequate filling of the arterial system, the body being at rest, without need for increased frequency of heart beat.

I trust that haemodynamic considerations such as these, in addition to those connected with tissue-typing and perfusion experiments, are being taken into account in the surgery of cardiac transplantation. I would welcome assurance to this effect.—I am, etc.,

J. E. MALCOLM.
Royal Air Force Hospital,
Ely, Cambs.

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1 Malcolm, J. E., *J. Physiol. (Lond.)*, 1961, **157**, 2P.
2 —— *Brit. med. J.*, 1961, **2**, 450.
3 McDonald, D. A., *J. Physiol. (Lond.)*, 1952, **118**, 328.
4 —— ibid., 1955, **127**, 533.
5 Wormersley, J. R., ibid., 1955, **127**, 553.
6 —— *Phil. Mag.*, 1955, **46**, 199.
7 Hamilton, W. F., *Amer. J. Physiol.*, 1944, **141**, 235.

Royal Malady

SIR,—I have just read the article by Dr. Ida Macalpine and others (6 January, p. 7) claiming that porphyria variegata can be traced in the Royal Houses as far back as Mary Queen of Scots. I would like to congratulate them on a very fine study, but regret I am completely unconvinced by the evidence, and in fact consider it highly unlikely for the following reasons:

(1) One immigrant to South Africa, who married at the Cape in 1688, now has over 8,000 living descendants who have inherited porphyria variegata.[1] This inherited disorder of metabolism, as far as we know from family studies, caused no significant symptoms, except for a light-sensitive skin, until the introduction of modern drugs, particularly barbiturates and sulphonamides. Those who had inherited porphyria variegata had just as large families on average as the rest of early white settlers in South Africa. They lived on average as long as anyone else. There is nothing in old medical records or in the family records—and I have studied many old South African records and many families with the disorder—to suggest that they suffered from symptoms of acute porphyria. Today the South Africans with porphyria variegata who avoid barbiturates and sulphonamides and a few other, seldom-used drugs keep as well as those who have not inherited the gene, and are accepted for life insurance.

(2) If royal personages as far back as Mary Queen of Scots had porphyria variegata there should be thousands of descendants alive today, many of whom one would expect to have inherited porphyria variegata. Some of them in recent years should have had typical acute porphyria attacks, say after a thiopentone anaesthetic. Only two very doubtful living members are quoted by the authors. Patient A, in whom a distinguished physician is quoted as having made the diagnosis of acute porphyria. I have known many distinguished physicians to make a mistake in the diagnosis of porphyria. Did Professor C. Rimington receive a specimen for examination? If this patient is indeed a porphyriac the gene might have been introduced to her family recently. Patient B—the increase in porphyrin quoted (copro-12, proto-74 μg./g. dry weight) is not at all diagnostic of porphyria, and is often seen in those who do not have porphyria variegata. It is just a little raised and would suggest that further specimens should be examined and specimens from other members of the family. In porphyria variegata the stool porphyrin is often 10 times higher than this. So far little is known about the secretion of X-porphyrin in those who do not have porphyria variegata, but the level quoted (15.6 μg./g. dry weight) is not high.

(3) James I. Acute porphyria causes constipation, not diarrhoea. A red urine was most likely due to "gravel," to which our ancestors were notoriously liable in the seventeenth century. He had pain over the left kidney. Red spots on his face and bruising are not the skin lesions of porphyria; the characteristic skin lesions are water-blisters or a tendency for the exposed skin to abrade. Why should the "two concretions" found at necropsy be "obviously congenital, non-functioning malformations" and not renal stones? Mayerne's diagnosis of kidney disease and haematuria and some form of arthritis, perhaps gout, is much more probable than recurrent attacks of acute porpyhia. (What drug precipitated the acute porphyria variegata in 1613?)

James I's eldest son, Henry Frederick, Prince of Wales, also died from an acute illness with diarrhoea. I have never seen acute porphyria with diarrhoea. Constipation is the rule.

Mary Queen of Scots in her acute illness with pain in her side, made worse by breathing, quite likely had pleurisy. If she "became delirious, lost her sight and speech, had a series of fits, remained unconscious for some hours, and was thought to be dead," and these symptoms were caused by acute porphyria, she would almost certainly have died. If she had not died she would not be "up and about again" within 10 days. Colic due to gastroenteritis must have been very common at the time of Mary Queen of Scots, and she may have had a tendency to epileptic fits, from which she would indeed recover completely in 10 days.

If all the royal personages quoted had the symptoms mentioned as a result of attacks of acute porphyria why is it that our thousands of white South African porphyriacs alive today remain well as long as they avoid modern drugs, particularly barbiturates and sulphonamides? I can think of no drug that was used in the time of George III and earlier which caused acute porphyria.

In conclusion, while Dr. Macalpine and her co-workers are to be congratulated for making a very fine study of the diseases of the Royal Houses, the evidence they present that so many royal personages from the time of Mary Queen of Scots suffered from attacks of acute porphyria variegata is, in my opinion, "not proven."—I am, etc.,

GEOFFREY DEAN.
Port Elizabeth,
S. Africa

REFERENCE
1 Dean, G., *The Porphyrias: A Story of Inheritance and Environment*, 1963. Pitman's Medical Publishers, London.

SIR,—While agreeing with most of the points made by Professor C. E. Dent (3 February, p. 311), I feel he is not altogether fair.

As regards urine colour, I doubt whether there is much difference between the colour of Alicante wine and that of "port wine."[1]

I am much less experienced than Professor Dent, but I am sure there are some attacks in the variegate type whose aetiology is obscure and that some attacks can be precipitated by infections which may have been more frequent in previous centuries, and I think it probable that when attacks are thought to have been precipitated by barbiturates they may in fact have only aggravated an already existing attack. In general there seems considerable scope for fairly frequent attacks in previous centuries.

I am more sympathetic than Professor Dent to the high refusal rate among the "royals." In all my surveys during the last 20 years (with the exception of one for carcinoma of the cervix), we found that the refusal rate was positively related to social class. It was high enough among doctors, clergymen, and senior executives; goodness knows what it would have been among a "royal" population.