carriers. In doubtful cases the personal history of relatives or other close contacts may give the clue.

Towards the end of the first world war some 11,000 ex-malarial patients arrived in the United Kingdom from Salonika, north Italy, and other endemic areas. During 1917-18 117 cases and 1918-19 63 cases of malaria (figures from memory only, as I have no present means of checking) were reported in people who had never been out of this country. These had obviously been infected from chronic carriers who had arrived from endemic areas. I had two severe bouts of malaria while serving in northern Italy during the first world war, and on my return to home service I was living with my family in Fleet, Hants. There was a marsh beyond our garden boundary, the Basingstoke canal 400 yards (360 m.) beyond,

and Fleet pond less than a mile away. Mosquitoes were prevalent in July and August, and I had seen anopheles, *A. maculipennis* among them. The children's nanny and our cook, sisters from Gloucestershire, both developed febrile conditions in September which to me looked like malaria. With laboratory facilities readily available I found *P. vivax* in nanny's blood but failed with cook's. A fellow officer with tropical experience confirmed my findings. Antimalarial treatment rapidly cleared up both cases.

In this present age of rapid transport and mass tourism from potentially malarious areas the possibility of indigenous infection is greatly increased and should be borne in mind if we get a summer suitable for mosquito life.—I am, etc.,

Chumleigh,
N. Devon.

TREFFRY O. THOMPSON.

unstable sinus rhythm with many ectopic beats. These were abolished with the introduction of prednisolone.

The reason for the success of steroid therapy is not clear. It was given partly in the hope that it might suppress an irritable focus in the myocardium and partly empirically. At the present time this man has been free of tachycardia for six months. He remains on digoxin, frusemide, and prednisolone 12.5 mg. daily.

We would like to thank Dr. K. S. Maclean for permission to report this case.

—We are, etc.,

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Adams-Stokes Attacks and Corticosteroids

SIR,—The use of corticosteroids in the treatment of Adams-Stokes attacks due to heart block is well known. Other arrhythmias may occasionally prove to be very resistant to orthodox treatment and may too be life-threatening, especially when many attacks occur over a short period of time. There have been a few accounts of the use of short courses of prednisolone,¹ hydrocortisone,² and triamcinolone³ in the management of paroxysmal supraventricular and ventricular arrhythmias. We would like to record our experience with a 64-year-old man in whom corticosteroids appeared to halt a life-threatening sequence of attacks.

In 1964 the patient, then aged 62, suffered a posterior myocardial infarction. Since that time he has had a number of admissions to hospital with episodes of tachycardia. From

the electrocardiographs it has been difficult to determine whether this was a nodal or ventricular tachycardia (see Fig.). In 1965 he had two admissions, reverting to sinus rhythm on the first occasion with quinidine, and on the second occasion with propranolol after quinidine had failed. In 1966 he was again admitted twice. In March he responded to an increased dose of propranolol. In July he had a complicating melaena (attributed to a bleed caused by anticoagulants), and after an initial response to increasing the dose of propranolol he required four D.C. shocks in the course of eight days. In 1967 he had a series of admissions, with a tendency for his arrhythmia to reappear frequently. He was developing signs of left ventricular failure after 12 to 18 hours in the abnormal rhythm. The Table summarizes the sequence of events leading up to the administration of steroids.

It will be seen that most of the traditional drugs were used to try to achieve and maintain a stable sinus rhythm. It could be argued that the introduction of prednisolone had merely coincided with a spontaneous improvement—a common problem in evaluating the benefits of drugs for arrhythmias. These points seem to us to be against this interpretation:

(1) At the time when prednisolone was introduced this man's course was downhill. Periods of remission were lasting a shorter time, and he had seven episodes of tachycardia in one month, all of them needing D.C. version. He was developing signs of left ventricular failure after progressively shorter periods of tachycardia.

(2) His electrocardiograph after D.C. version before steroids had always shown an

Taken during Tachycardia

Taken during Sinus Rhythm

Date	Regimen at Onset of Attack	Drugs used in the Acute Attack	Changes in Regimen	D.C. Shock Needed (i.e., Drugs Failed to Revert)
February	Phenindione, propranolol		Digoxin for propranolol	Yes
April	Phenindione, digoxin		Propranolol added	No
May	Phenindione, digoxin, procainamide		Neostigmine added	Yes
16 June	Phenindione, digoxin, procainamide, neostigmine	I.V. propranolol	Digoxin dose raised	Yes
22 June	Phenindione, digoxin	I.V. lignocaine	Quinidine	Yes
23 June	Phenindione, quinidine	I.V. atropine	Amytal	Yes
2 July	Phenindione, quinidine, amylorbarbitone			Yes
9 July	" " "			Yes
13 July	" " "		Digoxin, frusemide, heroin	Yes
21 July	Phenindione, digoxin, frusemide, heroin		Amylobarbitone, prednisolone* + digoxin, + frusemide	Yes

* Starting with 10 mg. t.d.s. and reducing over two and a half weeks to 12.5 mg. daily.

Royal Malady

SIR,—While full of admiration for the painstaking historical researches of Dr. Ida Macalpine and others (6 January, p. 7), along with Professor C. E. Dent (3 February, p. 311), we have misgivings regarding their interpretations and conclusions.

They assert that the dominant gene for variegate porphyria has been transmitted in the lineage of the Royal Houses of Stuart, Hanover, and Prussia. In essence this assertion rests on two pieces of evidence: the diagnosis of variegate porphyria in two contemporary descendants of George II; and their interpretation of the surviving medical and lay accounts of illnesses whose symptoms and manifestations they feel are "characteristic of porphyria." In our opinion the evidence they present is insufficient to support a diagnosis of variegate porphyria and in many instances contradicts such a diagnosis.

Consider first the diagnosis of variegate porphyria in the two contemporary descendants. No clinical or biochemical data on patient A are available for assessment, and our experience of the frequency with which porphyria is erroneously diagnosed leaves us hesitant to accept this diagnosis without such data. While the symptoms recorded for patient B would do reasonably well for variegate porphyria, the faecal protoporphyrin and coproporphyrin concentrations given make this diagnosis unlikely. In our laboratory over 90% of patients have values exceeding 500 µg./g. dry weight of faeces, and a total faecal porphyrin concentration of less than 200 µg./g. dry weight in a patient of the age of patient B would virtually exclude the diagnosis.¹ The diagnostic emphasis placed by Professor Rimington on the concentration of "X-porphyrin" in the faeces of patient B is, in our present state of knowledge, unwarranted. In 1963 Sweeney² reported the presence in the urine of a jaundiced patient with variegate porphyria of a hydrophilic porphyrin that was not uroporphyrin. Rimington³ has subsequently reported on some of the characteristics of a similar porphyrin in the faeces and indicated that its quantitation is difficult.⁴ This has also been our experience, and we have