experienced coupling with short bursts of ventricular tachycardia.

In view of the poor response to quinidine, procainamide, and lignocaine other antiarrhythmic medications were considered. Propranolol was rejected because of the congestive failure. 6 months earlier the patient had participated in a clinical-pharmacology study of nadolol ('Corgard', Squibb). P.V.C. frequency had been significantly reduced, and although he was in congestive heart-failure at the time, his clinical state did not deteriorate. Because of this previous response nadolol was tried again and other antiarrhythmic medications were discontinued. The dose was 10 mg every 6 h. Within 15 h, after a cumulative dose of 30 mg nadolol, the patient's mean P.V.C. frequency was reduced from 325±12 to 47±12 ectopic beats/h (mean±S.E.). Trigeminy and ventricular tachycardia disappeared. A plot of P.V.C. frequency, beginning shortly before nadolol therapy began and continuing throughout the first day of treatment, is shown in the accompanying figure. Beta blockade was confirmed by a drop in heart-rate from 65/min (range 52–85) to 58 (range 55–65).

The patient was discharged 10 days after admission on his usual drugs without any antiarrhythmic medication other than nadolol (85 mg) once daily. He has been on this therapy for over 12 months with a very satisfactory response.

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PORCINE CORONAVIRUS ANTIBODIES IN ENDEMIC (BALKAN) NEPHROPATHY

SIR,—It has been suggested that endemic (Balkan) nephropathy (E.N.) might be caused by a slow porcine coronavirus infection.[1] We confirmed the presence of viral particles in cells from E.N. patients but could find no consistent evidence of serum antibodies to several non-porcine viruses.[2] We now report the results of serological studies with a virus of porcine origin. Antibodies were measured by hæmagglutination inhibition and by single radial hæmolysis in gel.[3,4] Sera from 34 E.N. patients, relatives sharing their housing and living conditions, and 67 selected controls were examined. Sera from 21 pigs kept by patients were also examined. All sera were heat-inactivated before testing. Antigen was prepared in our laboratory using the 2063/68 virus strain, originally isolated in England from piglets with vomiting-and-wasting disease. This strain is closely related to, if not identical with, the hæmagglutinating encephalomyelitis virus of swine[5,6] and also related antigenically to the transmissible gastroenteritis virus of pigs.[7]

None of the pigs from which serum was taken had vomiting-and-wasting disease, transmissible gastroenteritis of swine, infantile pyloric stenosis of pigs, or encephalomyelitis of suckling pigs. Low titre antibodies to the 2063/68 virus strain were found in serum from only 1 animal. The sera from E.N. patients and controls were all negative.

The simplest interpretation of these data is that E.N. patients have not had contact with porcine coronavirus and that the virus-like particles found in renal epithelial cells of E.N. patients,[1,8] are of an antigenically unrelated strain, or a different type. It is possible, however, that anticoronavirus antibodies are transient and thus not detectable in patients with established disease.

The coronavirus strains used came from Dr D. J. Alexander, Central Veterinary Laboratory, Weybridge; Dr S. R. Hopkins, Southeast Poultry Research Laboratory, Athens; Dr H. S. Kaye, Center for Disease Control, Atlanta; Dr K. J. Sørensen, State Veterinary Institute for Virus Research, Lindholm; Dr D. A. J. Tyrrell, Clinical Research Centre, Harrow.

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WARFARIN ENANTIOMERS, ANTICOAGULATION, AND EXPERIMENTAL TUMOUR METASTASIS

SIR,—Coumarin derivatives reduce metastasis in experimental tumours.[1,2] The role of the induced anticoagulation in this effect has been questioned by Hilgard,[3] who, with others,[4-6] has suggested a direct cytotoxic activity on cancer cells and inhibition of cell motility and of mitotic activity. Coumarin derivatives had an antimetastatic effect in some experimental systems in which heparin was ineffective.[7]

TABLE I—EFFECT OF TREATMENT WITH RACEMIC, R(+), OR S(−) WARFARIN ON 3LL LUNG COLONIES (AT DAY 16)

Treatment	Lung colonies	
	No.	Weight (mg)
Water	23·9±2·8	440±55
RS warfarin	4·2±1·0*	18±8*
R(+) warfarin	30·3±5·4	458±120
S(−) warfarin	2·0±0·5*	8±1*

Data represent mean ±S.E. of results obtained from 20 animals per group.
* $P<0.01$ (Duncan new multiple range test).

TABLE II—EFFECT OF TREATMENT WITH RACEMIC, R(+), OR S(−) WARFARIN ON 3LL PRIMARY TUMOUR AND SPONTANEOUS METASTASES (AT DAY 21)

Treatment	Tumour weight (g)	Metastases weight (mg)
Water	9·4±0·1	96±12
RS warfarin	7·8±0·4*	50±21†
R(+) warfarin	10·1±0·3	103±14
S(−) warfarin	6·8±0·5*	41±14†

* $P<0.01$. † $P<0.05$.

Warfarin used for anticoagulant therapy is a racemic mixture of equal parts of R(+) and S(−) enantiomers; these compounds differ in pharmacokinetic properties and in anticoagulant activity.[8,9]

We have used the two warfarin enantiomers in mice bearing

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