

It was aimed to use intracardiac electrocardiography and programmed cardiac stimulation techniques to assess autonomic influences on normal function of the human sinoatrial node (SAN), atrioventricular node (AVN) and intraventricular conduction system (IVCS).

Right heart catheterisation was undertaken in 22 patients (aged 39-82 years) with normal SAN, AVN and IVCS function being studied primarily for arrhythmias. Intracardiac conduction intervals, sinus node recovery time (SNRT), the Wenckebach threshold (WT) to right atrial pacing, cardiac refractory periods and the QT interval were determined before (ND) and after autonomic block induced by atropine (A), 0.03 mg/kg IVI, and propranolol (P), 0.15 mg/kg IVI.

Sympathetic and larger depressant vagal effects on SAN function (cycle length and SNRT) were demonstrated. SNRT (pacing at 100 bpm) was 1165 ± 22 ms (ND), and 981 ± 24 ms (A + P) ($p < 0.001$). Autonomic effects on AVN function (AH interval, WT and AVN refractory periods) were small, of similar magnitude and opposing. His bundle conduction and HV interval (measuring IVCS conduction) were unchanged by either drug. Effects on IVCS refractory periods could not be assessed because of AVN refractoriness.

As a measure of ventricular repolarisation, the QT interval with pacing at 100 bpm (338 ± 4 ms) decreased significantly following atropine (320 ± 5 ms) ($p < 0.001$) but was unchanged following propranolol.

It was concluded that - 1. vagal effects are predominant on SAN function, 2. vagal and sympathetic effects on AVN function are small and counterbalancing, and 3. there are no autonomic effects on the IVCS.

Factors which influence the degree of damage caused by myocardial ischaemia include cardiac work and myocardial perfusion, both of which are determined, in part, by blood pressure.

The effects of a wide range of blood pressures (mean BP 34 to 166 mm Hg) on indices of myocardial ischaemia were studied in eight open-chest anaesthetised dogs. The left anterior descending coronary artery was totally constricted for eight minutes to induce ischaemia without infarction during a control period, during angiotensin II-induced hypertension and during sodium nitroprusside-induced hypotension. Arterial and coronary sinus concentrations of creatine kinase (CK), lactate and potassium (K^+) were measured. The fractional extraction (FE) of lactate was greater during hypertension than during hypotension ($30.2 \pm 6.4\%$ and $2.3 \pm 9.8\%$ respectively, $p < 0.05$, one-way analysis of variance, 23 d.f.). FE lactate and mean BP showed a significant positive correlation ($r = 0.48$, $n = 24$, $p < 0.05$). FFA metabolism did not account for this relationship. Significant arterial-coronary sinus differences in concentration (CA-CS) of CK were observed during control, but not during hypertension or hypotension. Coronary sinus K^+ concentrations were increased in each period but CA-CS K^+ achieved statistical significance only during hypertension. In this model, higher BPs are associated with less myocardial anoxia, as judged by FE lactate. CK may be released during ischaemia without infarction. The dissociation between CK release, lactate extraction and K^+ release suggests that these indices may reflect different aspects of cellular ischaemia.