helical structure of DNA during freezing and thawing suggests that some different types of interaction with water are involved in these biological macromolecules.

I thank Prof. K. Aoki for his advice.

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PHYSIOLOGY

Heart Valve Lesions following Papillary Muscle Damage

THE genesis of rheumatic valvular disease in man has been difficult to establish owing to the scarcity of human material in the early stages and the absence of any comparable condition in experimental animals. On the evidence at present available it has been stated that the calcific lesions found in human rheumatic valvular disease are unlikely to be the direct result of the rheumatic process in the valve cusps¹. In recent experiments we have been studying the effects of papillary muscle damage and inactivation on the function of the mitral valve and the left ventricle of the dog². In these experiments either the anterior or the posterior papillary muscle was injected with 0.5–1.0 ml. ethanol and the animal allowed to recover. A surprising finding was that when the animals were killed three months later, four out of twelve dogs in addition to papillary muscle damage also had fibrotic, calcific or chondritic lesions of the mitral valve cusps (Fig. 1). In all four animals the valvular lesion was in the segment of the aortic cusp of the mitral valve which was attached to the damaged papillary muscle and there was no continuity of the valvular lesions with either the papillary muscle lesion through the chordae tendinae or the atriotomy scar in the three animals in which this had been carried out. In ten dogs used as controls in which the ethanol injection was made into the lateral wall of the left ventricle between the papillary muscles, no valvular lesions were found. Histological examination of the lesions showed a fibrous plaque on the atrial surface of the valve cusp with replacement by calcification and chondrification (Fig. 2). There was no evidence of an infective lesion.

Chondrification of endothelial vascular tissue in response to trauma has been reported previously in both dogs and

Fig. 2. Longitudinal section of aortic cusp of mitral valve in dog, 15 weeks after papillary muscle injection. The plaque on the atrial aspect of the cusp is composed of fibro-cartilage with patchy calcification

chickens^{3–5}. Cartilagenous tissue was found in the chicken beyond an artificial coarctation⁴ and it was suggested that it was the consequence of abnormal compression forces; but invagination of the left atrial appendage over a silastic ball, although producing papilliform excrescences on the heart valves, did not produce chondrification within the heart⁶.

Endoscopic photography in these animals has shown excessive 'flutter' in the valve cusps and mitral incompetence⁷. Our present results suggest that the absence of normal papillary muscle action may give rise to abnormal stresses within the valve cusps which may produce calcific and chondritic lesions. It is therefore possible that in human valvular disease, also, calcification is the consequence of traumatic stress.

This research was carried out under a grant from the Scottish Hospitals Endowment Research Trust.

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Fig. 1. Open left ventricle of dog, 10 weeks after injection of papillary muscle. Area of papillary muscle damage and lesion on aortic cusp of mitral valve outlined

Some Behavioural Correlates of the Transcephalic d.c. Potential in Cats

NEURAL direct current (d.c.) potentials have recently received much attention¹. A steady d.c. potential across amphibian cortex in a fronto-occipital plane has been observed². These measurable exterior surface potentials are correlated with anaesthesia and sleep^{3,4}. High correlations have been reported between transecephalic d.c. potential (measured between mid-forehead to low union), level of anaesthesia and electro-encephalograph (EEG)⁵. In the awake state the surface-recorded steady potentials are frontally negative, while in anaesthesia the frontal