1·32 ± 0·06. In general, higher values were observed with increasing age, as previously noted by Biörck[3].

In the group of hypertrophied hearts with congenital lesions, the higher concentration of cytochrome *c* was always found in the more severely affected ventricle. The six specimens with right ventricular hypertrophy, one of which was obtained in the neonatal period, had an LV/RV ratio of 0·69 ± 0·07. In two cases of left ventricular hypertrophy these ratios were 2·56 and 1·74.

Badeer[7] has reviewed the current theories concerning the nature of the stimulus to cardiac hypertrophy. He proposed the hypothesis that any chronic condition leading to an increase in metabolic rate of a heart chamber might lead to its hypertrophy. Increased oxygen uptake and hypertrophy both appeared to develop most readily in situations that raise the pressure in a heart chamber during systole, such as valvular stenosis or increased peripheral vascular resistance. The capacity of a tissue for oxidative metabolism is believed to be reflected by its content of cytochrome *c* (ref. 8). In the present investigation, higher concentrations of cytochrome *c* were generally present in the ventricle the work load of which is known to be elevated[9,10]. These findings suggest a response in oxidative capacity per unit mass associated with the normal functional maturation of each ventricle, as well as with altered function in congenital heart lesions.

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Nuclear Membrane Hydrolysis of Adenosine Triphosphate

ADENOSINE triphosphate (ATP) hydrolysis was studied cytochemically in embryonic myocardia of the chick, using a modification of Wachstein and Meisel's method[1]. Fresh embryonic hearts were pre-fixed in ice cold 2·5 per cent glutaraldehyde for 30 min and stored at 0°–4° C in an isotonic sucrose medium containing 0·1 molar *tris* maleate sodium hydroxide buffer at *p*H 7·2. After 3 days, tissues less than 0·5 mm³ were pre-incubated in a reaction medium without ATP to allow for ionic equilibration. The medium contained a final concentration of 0·08 molar *tris* maleate–sodium hydroxide buffer at *p*H 7·2, 0·10 molar sodium ions, 0·03 molar potassium chloride and 0·005 molar magnesium chloride, hexahydrate. Lead nitrate, 0·005 molar, was added just before 0·0025 molar ATP (sodium salt) was added. The reaction was run for 30 min at 30° C, both with and without substrate.

Ultramicrotome sections were taken from the region immediately below the surface of the tissue, which was post fixed in 1 per cent osmium tetroxide and embedded in 'Epon'. Electron micrographs were taken with a 'Siemens Elmiskop *I*'. Fig. 1 shows a section taken from an experimental 4 day embryonic ventricle. A lead deposit indicating "presumptive" ATP hydrolysis can be seen outlining the sarcolemmae. It is also closely associated with the inner nuclear membrane and the granules adjacent to it or with the granules alone, and is particularly dense in nuclear "pores".

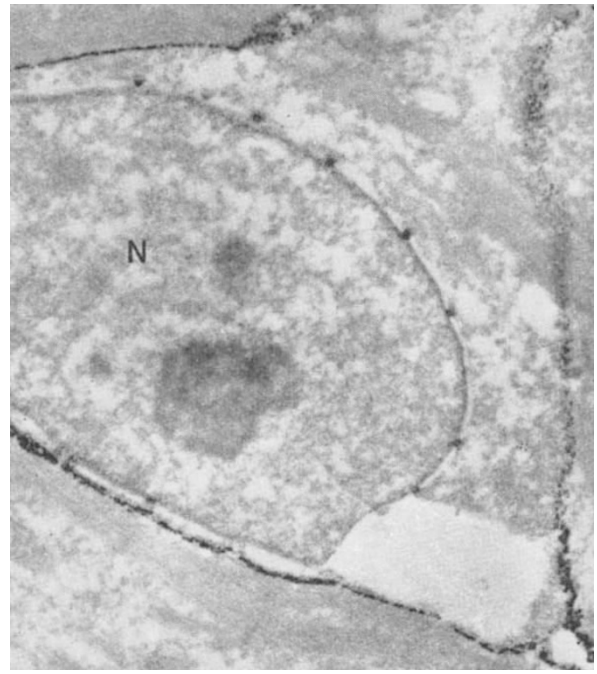

Fig 1. ATP reacted 4 day embryonic ventricle (× *c.* 16,550). *N*, Nucleus.

This observation may be relevant to nuclear membrane transport, but the methods used could give misleading results. For example, nucleoproteins have an especially high affinity for the lead marker, and tissue blocks can be predisposed to diffusion artefacts, but the fact that when ATP was absent the typical pattern of lead deposition was not observed gives some significance to this occurrence of deposition.

Current cytochemical studies on thin unfixed cryostat sections and biochemical studies on isolated nuclear fractions from embryonic myocardia substantiate the occurrence of true ATPase activity associated with these nuclei. The enzyme has some similarities to ATPases of liver cell nuclei reported by Siebert[2] and Bankowski[3]. More rigorously controlled investigations are needed to determine the extent to which present ultrastructural localization may be artefactual, involving the use of fixed and unfixed cryostat sections.

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Lactate Dehydrogenase Isoenzymes in Developing Human Muscle

THERE has recently been considerable interest in the changes which occur in the lactate dehydrogenase (LDH) isoenzyme pattern of skeletal muscle when the tissue is affected by a natural or experimental lesion, particularly with reference to human muscle disease. These changes have been regarded as a failure of maturation[1], or, alternatively, as a process of de-differentiation, for similar changes have been seen in human neurogenic