the questions (*a*) why, in Leydig cell proliferation, adequate numbers of new capillaries are not formed, and (*b*) what factors limit growth along the existing vascular tree.

In (*a*) a recent preliminary report[7] on the possible reduction in the number of small blood vessels in the seminal vessels of the castrate rat is in part suggestive that the local action of Leydig cell secretion may effect capillary proliferation. However, the process of infiltration of the hyaline substantia is usually diffusely distributed. In (*b*) if it is assumed that endocrine epithelium must have a thin-walled capillary blood supply, it follows that limitation of proliferation along the capillary bed, away from the tubular capillaries, will be effected by increasing thickness of the vessel wall, be it arteriole or venule. The point of importance in these observations is that the same capillary supplies Leydig cells and tubule.

Because of the extreme tortuosity of the tubules and the close application of many of the Leydig cells to the tubular wall, it is difficult to demonstrate clearly on which side of the tubular capillary bed the Leydig cells are situated. In the spermatic cord the Leydig cells are situated close to the arterioles and the same relations probably exist within the testicular parenchyma.

On the basis of these findings, I advance the hypothesis that Leydig cells are situated between the arterioles and the testicular tubules and have the same capillary blood supply as the tubules. This hypothesis may usefully be applied to several aspects of testicular function, notably the gonadotrophic effect of steroid hormones and their varying effect at different dosage-levels, the irregular segmental distribution of atrophic changes in mature testes, pubertal development and abnormal segmental development as in cryptorchidism, as well as possible mechanisms in male infertility and its therapy.

J. B. W. Halley

Department of Pathology,
The Medical School,
Birmingham 15.

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HISTOLOGY

Histochemical Demonstration of Succinic Dehydrogenase Activity in Osteoclasts

A review of the literature has revealed but few studies on enzyme characteristics of osteoclasts. None of these, however, related to dehydrogenase activity.

In the present work frozen sections of rat and hamster knee joints and jaws, 8–10μ thick, were prepared according to the Adamstone-Taylor cold knife technique and mounted on glass microscope slides. In addition, pieces of calvarium from newborn hamsters were also employed. Slides and specimens were then incubated for 60–90 min. in the succinic dehydrogenase substrate solution as described by Pearson[1]. This contained nitroneotetrazolium. Representative sections were also incubated in a substrate solution containing nitro blue tetrazolium[2] as

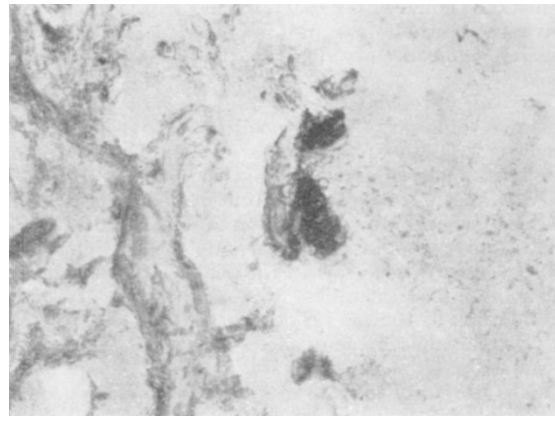

Fig. 1. Succinic dehydrogenase activity of osteoclasts (knee joint from 2-day-old hamster). (× *c.* 214)

described by Nachlas *et al.* With both substrates an intense selective staining reaction was observed in osteoclasts (Fig. 1). Sections incubated in tetrazole without the co-factor (succinate) were unstained, as were heat-treated sections. Sodium malonate was inhibitory. The staining of osteoclasts associated with the remodelling resorption of the jaws was particularly striking. Osteoblasts by contrast showed little if any reaction.

Osteoclasts associated with both intramembranous and intracartilagenous bone resorption showed a similar reaction. The cytoplasm was evenly stained, and it was difficult to assess any mitochondrial reaction.

Osteoclasts have also been observed to exhibit high acid phosphatase[3] and aminopeptidase activity[4]. The present study would further tend to confirm the hypothesis that these cells exhibit high metabolic activity. Localization of succinic dehydrogenase to osteoclasts may be considered as presumptive evidence that the Krebs cycle is involved in the resorptive process.

M. S. Burstone

National Institute of Dental Research,
National Institutes of Health,
Public Health Service,
U.S. Department of Health,
Education and Welfare,
Bethesda 14, Maryland.

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Histochemical Localization of Oxidase Activity in the Mitochondria of the Heart in Some Lower Vertebrates

With the aid of some new methods[1–3] the histochemical localization of oxidase activity was investigated in the mitochondria of the heart in some lower vertebrates, namely, the viviparous cyprinodonts, *Lebistes reticulatus*, *Xiphophorus helleri* and *Xiphophorus maculatus*, the carp, *Cyprinus carpio*, the frog, *Rana temporaria*, the toad, *Bufo calamita* and the lizard, *Lacerta agilis*.

To permit a satisfactory comparison with the results of Burstone[3] obtained with the mitochondria