extensive myofilament degradation was always found in both mammalian and amphibian preparations which closely resembled that described in skeletal muscle[7–9]. Typical damage included intensely contracted filaments with blurring of the Z-lines (see figures 1 and 2); in other areas, extensive myofilament dissolution was seen, with the myosin filaments being particularly affected. Finally, areas could be found with complete degradation of the myofilament apparatus. Control preparations similarly treated but lacking ionophore were normal.

We conclude that marked rises in $[Ca^{2+}]_i$ in cardiac muscle are able to initiate myofilament degradation, similar to that reported in skeletal muscle. However, A23187 rarely causes damage in cardiac muscle except when its action is augmented by partial depolarization or stimulation. The ionophore is effective with skeletal muscle in the absence of extracellular Ca^{2+} and is believed to act primarily by releasing Ca^{2+} from the sarcoplasmic reticulum (SR). We suggest that this difference between skeletal and cardiac muscle may lie in the relative importance of the different systems controlling $[Ca^{2+}]_i$. The bulk of the intracellular Ca^{2+} is stored in the SR in skeletal muscle which is therefore particularly sensitive to A23187; in cardiac muscle the mitochondria contain (Patriarca and Carafoli[11]) the Ca^{2+} required for contraction enters from outside the cell at excitation. We conclude that release of Ca^{2+} from the SR by A23187 does not usually raise $[Ca^{2+}]_i$ to sufficiently high levels in cardiac muscle to cause rapid myofilament degradation, unless accompanied by an influx of extracellular Ca^{2+} induced by the depolarization of the plasma membrane.

Exposure of amphibian or mammalian skeletal muscle strips to either 2,4-dinitrophenol (DNP) (10^{-4} M, 25 min) or to ruthenium red (30 µM, 40 min) also rapidly causes typical myofilament degradation with the same characteristics as those described above. So far, we have not found any evidence of reversibility of this rapid degradative effect in our in vitro systems.

Equally, perfusion of the isolated frog heart with saline containing 10^{-4} M DNP caused cessation of contractile activity in 3–5 min and electron microscopy of the muscle at this stage again revealed myofilament degradation. Contractile activity continued on perfusion with 10^{-5} M DNP, but examination again revealed extensive damage. DNP is a mitochondrial uncoupling agent, and ruthenium red inhibits intracellular Ca^{2+}-uptake, and we conclude that ultrastructural damage can be rapidly produced in cardiac muscle when the functional integrity of the mitochondria is seriously impaired and that the consequent release of Ca^{2+} from this major intracellular store[11] is a potent means of triggering damage.

From these studies, we suggest that 1. Cardiac and skeletal muscles are similar in that myofilament damage can be rapidly induced by a variety of treatments in both. 2. The common feature in these experiments appears to be a marked rise in $[Ca^{2+}]_i$ in the myoplasm. 3. The 2 tissues differ in the relative importance of the different intracellular systems controlling $[Ca^{2+}]_i$. The SR constitutes the main store of Ca^{2+} in skeletal muscle; in cardiac muscle the mitochondria (as is shown in ischaemia or DNP treatment) and the plasma membrane (as in the calcium paradox) are of particular importance. The sensitivity of mitochondrial Ca^{2+} accumulation to ischaemia in cardiac muscle is well documented. 4. A marked rise in $[Ca^{2+}]_i$ probably promotes lysosomal breakdown in cardiac and skeletal muscle, the enzymes so released being responsible for this form of rapid myofilament degradation[10]. Methyl prednisolone is a known lysosomal stabilizing agent[12] which has been shown in some studies to have a protective effect in the damage induced by ischaemia in cardiac muscle[3]. 5. These findings could be of importance for our understanding of cellular injury in cardiac muscle following ischaemia.

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Photoreceptor properties of an ectopic eye in the fleshfly, *Sarcophaga bullata*[1]

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Summary. Ectopic eyes were produced on the fleshfly, *Sarcophaga bullata* by transplantation of imaginal eye discs. Electrophysiological and histological observations of these supernumerary eyes indicate the absence of synaptic connections between retinular cells and higher order neurons.

The compound eye of insects consists of several thousand ommatidia, and each ommatidium is composed of photoreceptor cells called retinular cells. Determining the properties of these sensory cells by extracellular recording has been complicated by potentials originating from synaptic connections with second order neurons. Studies with surgically isolated ommatidia[3] and with eyes cultured in vivo (and hence lacking synapses)[4] have indicated that photoreceptor cell depolarizations contribute a monophasic negative response to the electroretinogram (ERG, sometimes called the retinal action potential). In this study we have investigated the retinular cells by using an ectopic eye in the fleshfly, *Sarcophaga bullata.* These eyes closely resemble the normal eyes in their external appearance.

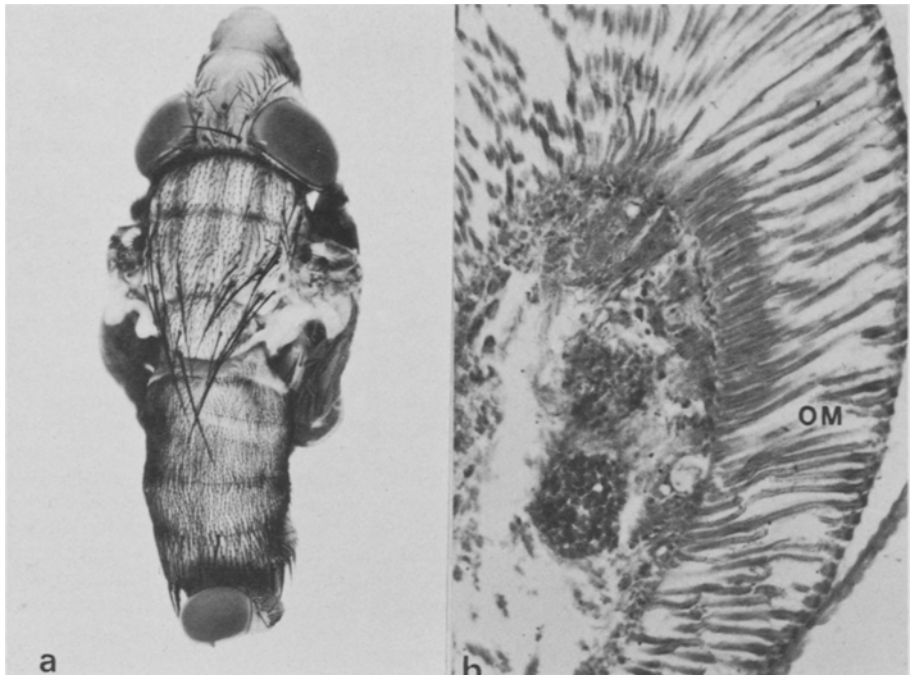

Fig. 1. The ectopic eye. *a* Host with the ectopic eye at the tip of its abdomen. *b* Paraffin section of the ectopic eye showing ommatidia (OM). × 320.

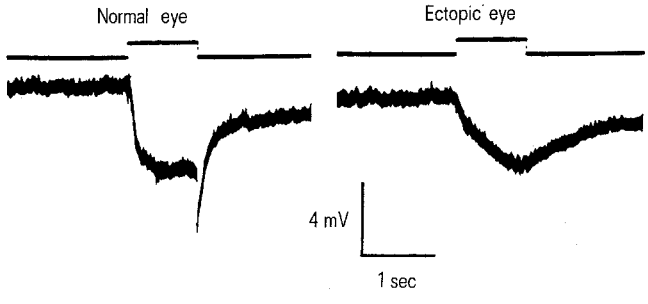

Fig. 2. Electroretinograms recorded from normal and ectopic eyes are shown on the lower trace. The upper trace represents the light monitor, a bright 470 nm light stimulus (10^{-16}–10^{-17} quanta/cm^2 · sec). A small on-transient and a large off-transient response was recorded from the normal but not from the ectopic eye.

Material and methods. Supernumerary eyes, and other appendages can be developed in flies by a simple transplantation technique[5]. In our application of this procedure, eye imaginal discs from mature 3rd instar larvae were transplanted onto the dorsal posterior end of the freshly formed prepupae. When the host pupae metamorphosed into adult flies, the transplanted eye discs had differentiated into supernumerary ectopic eyes (figure 1, a). Electroretinograms (ERG's) of these eyes were recorded with a subcorneally placed, NaCl filled microelectrode. Monochromatic light stimuli were from a 150 W Xenon arc and a Bausch and Lomb 500 mm monochromator (adjusted for a 13 nm maximum band pass) focused onto the eye with an achromat 10× objective. The ERG's were displayed on a Tektronix (5100 series) oscilloscope and photographed with a Tektronix camera. For histological observations, the specimens were fixed in alcoholic Bouin's solution, sectioned in paraffin and stained with haematoxylin and eosin.

Results and discussion. ERG's from at least 6 ectopic eyes were recorded and in all these there was only a sustained corneal negativity during illumination while the characteristic on- and off- transient responses exhibited by normal eye were absent (figure 2). Treatments that affect the synaptic activity have also been shown to eliminate the on- and off-transients from ERG's[6,7]. Furthermore, *Drosophila* mutants with impaired synaptic transmission from retinular cells to second order neurons in the lamina ganglionaris fail to show the on- transients at the onset of illumination[8,9]. From these studies it has been concluded that the on- and off-transients originate in the lamina and the sustained negative response from retinular cell depolarizations[10]. Histological sections of the ectopic eye examined under light microscope reveal the presence of well formed ommatidia with retinular cells (figure 1, b) whose axons pass through the basement membrane and penetrate a conspicuous neuropile-like mass which appears relatively unorganized compared to the real lamina ganglionaris. Based on these light microscope examinations it is difficult to establish unequivocally the presence or absence of synaptic connectivity of retinular cells with higher order neurons. However, from the lack of on- and off-effects in the ERG's of ectopic eyes we suggest that the retinular cells are not synaptically connected with lamina ganglionaris. Thus our observations using ectopic eye also confirm the view that the sustained monophasic negative response in the ERG is contributed by the retinular cells while the on- and off-transients originate in the lamina ganglionaris.

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