Ultrastructural changes in atrial sympathetic nerves during perfusion with solutions deficient in sodium and compensated by potassium or urea

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Perfusion of rabbit hearts with solutions deficient in sodium results in the release of noradrenaline into the perfusion fluid (Muscholl & Ritzel, 1976; Ritzel & Muscholl, 1976) and the mechanism depends on whether the absence of sodium is compensated either by potassium or urea. With potassium, the release is calcium dependent but independent of temperature between 16 and 36°C whilst with urea, release occurs independent of calcium but is greatly dependent on temperature. The present report compares the histochemical and ultrastructural changes which result from these two perfusion procedures.

Hearts were perfused with Tyrode solution depleted of sodium but containing compensating amounts of either potassium (high K–low Na) or urea (urea–low Na) (see Ritzel & Muscholl, 1976 for details). Samples of right atrial tissue were removed for electron and fluorescence microscopy after 5, 15 and 120 minutes. In separate experiments, the endogenous atrial and ventricular noradrenaline concentrations were estimated at these times by spectrofluorimetric techniques.

When compared to hearts perfused with normal Tyrode solution, atrial noradrenaline concentrations declined by 12, 41 and 63% (high K–low Na) and by 21, 52 and 86% (urea–low Na) after 5, 15 and 120 minutes. After 5 min perfusion with high K–low Na, little change was apparent in the ultrastructure of either noradrenergic nerve terminal regions or myocardial cells and there was no apparent reduction in the specific fluorescence attributable to noradrenaline. After 15 min both the endogenous noradrenaline and the intensity of fluorescence were reduced and there was fine structural evidence that the dense cores contained within 50 nm diameter axonal vesicles were smaller than in controls and that the number of dense cored vesicles per axon profile was reduced. This trend was continued until at 120 min specific noradrenaline fluorescence was markedly reduced and most axon profiles contained very few vesicles. Of the vesicles which remained, the majority were devoid of electron dense cores although evidence of fine structural damage to myocardial cells was minimal.

With urea–low Na, myocardial cells suffered damage after 5 min which was more extensive after longer perfusion times. However, at 5 and 15 min, despite the reduced noradrenaline concentrations, no marked differences were observed in fluorescence intensity or axonal ultrastructure between urea perfused and control hearts. Even at 120 min, when atrial cells were extensively damaged, axons containing 50 nm diameter dense cored vesicles were frequently seen, although fluorescence at this time was greatly reduced.

Thus, in keeping with the different mechanisms of noradrenaline release, the ultrastructural changes resulting from perfusion with either high K–low Na or urea–low Na solutions also differ. Whereas depletion of noradrenaline by high K–low Na is accompanied by progressive depletion of dense cored vesicles and minimal damage to the surrounding muscle cells, the noradrenaline depletion following urea–low Na is accompanied by minimal changes in axonal ultrastructure, despite the obvious extensive damage to surrounding myocardial tissue.

References

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