Myocardial protection and cardiac surgery

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Since the start of cardiac surgery in the 1950s, multiple techniques have been used to protect the heart during the surgical requirement for elective global ischaemia (and the still, relaxed, bloodless field that this provides for the surgeon during repair of the lesion). Most of these techniques have been discarded. The current gold standard, established over 30 years ago, is hyperkalaemic (moderately increased extracellular potassium) cardioplegia. This technique revolutionized cardiac surgery, allowing significant surgical advancement with relative safety [1,2]. Hyperkalaemic cardioplegia induces a rapid depolarized arrest that is readily reversible. Recent patient demographic changes, with surgeons operating on older, sicker patients who have more severe and diffuse disease, potentially requires a more prolonged elective ischaemia; hence, an improved myocardial protection would be of benefit. Several areas of study have demonstrated that a new concept of myocardial protection, 'polarized' arrest, may provide this additional protection [3,4]. Many pharmacological agents have been shown (in experimental studies), to have the ability to induce a polarized arrest and to provide improved protection. However, the often-overlooked requirements of effect reversibility and systemic safety have meant that these agents usually remain experimental in nature [5-8]. This review attempts to highlight the cellular components that can be targeted, within the excitation–contraction coupling cascade, to induce cardiac arrest, and to provide an explanation for the mechanism of action of these agents. In this context, the agents are discussed in terms of their clinical potential for use during cardiac surgery, with particular reference to the safety aspects of the agents. In addition, the potentially cardioprotective properties of a number of endogenous mechanisms are discussed [9,10].

References

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