

THE EFFECT OF PHOSPHATE BUFFERED RS-I SALINE ON THE CONTRACTILITY OF ISOLATED PERFUSED RAT HEART PREPARATIONS
D. Rees, Department of Physiology, Victoria University of Wellington, Wellington, New Zealand.

There have been several studies indicating the deleterious physiological affects of phosphate buffered salines on perfused isolated mammalian cardiac and skeletal organ/tissue systems (1,2). The biochemical basis for this effect is thought to reside in the inhibition of the glycolytic enzymes, hexokinase and phosphofructokinase, the latter being regulated by the level of fructose 1,6, bis-phosphate and the intracellular hydrogen ion (ph.) concentration (3).

In this study, the effect on the heart rate and contractility of isolated, non-paced rat hearts simultaneously perfused (via the aorta and/or left atrium) and superfused with aerated (95% O₂/5% CO₂) RS-I saline at 35.0 ± 0.3°C in a Res-Del perfusion system were examined following the substitution of a di/hydrogen phosphate buffer system with and without hydrogen bicarbonate in the normally BES/HCO₃ buffered RS-I saline (modified after Rees (4), see Table)

KCl	- 5.0mM	BES	-5.00mM	cocarboxylase(TPP)-40nM
NaCl	- 110.0mM	L-glutamine	-0.40mM	dl-carnitine -50uM
CaCl ₂ ,2H ₂ O	- 1.20mM	L-glutamate (Na ⁺)	-0.30mM	insulin(Porcine)-25mIU/l
MgCl ₂ ,6H ₂ O	- 0.45mM	Laspartate (Na ⁺)	-0.02mM	choline chloride -10µM
NaHCO ₃	- 25.0mM	Glycerol	-0.11mM	(pH 25-35°C -7.38±0.06)

A significant decrease was observed in the heart rate (68%) and force of contraction (77%) after 3 h of perfusion in phosphate/bicarbonate buffered RS-I in comparison to the heart rate (18%) and contractile force (8 %) recorded in hearts maintained in BES/bicarbonate buffered RS-I. In rat hearts perfused with RS-I containing physiological levels of epinephrine (0.5 nM) and ethyl acetoacetate (40-110µM), there was a 15-27% increase in the force of contraction with the hearts (non-paced) achieving 270-320 b/min at 38.1°C.

The results of this investigation will be discussed with respect to the role of the glycolytic pathway in the functioning of cardiac myofibres.

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