Sensitising effect of sodium palmitate to an induced arrhythmia in the isolated perfused hypoxic rabbit heart

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A relation between serum free fatty acid (FFA) and arrhythmias after acute myocardial infarction has been proposed (Oliver, Kurien & Greenwood, 1968).

In order to determine whether FFA increases the vulnerability to cardiac arrhythmias, the effect of sodium palmitate on the ventricular arrhythmia threshold (VAT) in the isolated Ringer-perfused rabbit heart was studied. The VAT was determined by applying a single 10 ms square-wave pulse of current to the left ventricle during the vulnerable period of late systole and the minimal current required to produce fibrillation or a rapid tachycardia was measured. The duration of the induced arrhythmia was listed as persistent (>60 s) or non-persistent. The minimal time after the R wave at which the 10 ms pulse had to be applied to produce an arrhythmia indicated the vulnerable time (VT). Because the VAT varied among hearts the magnitude of change was expressed as the VAT change ratio i.e. VAT during treatment/VAT of preceding control(s). Albumin (bovine fraction V, Sigma) was dialysed and conjugated with sodium palmitate and incorporated in the perfusion fluid to give concentrations of sodium palmitate (0.6 mM) and albumin (0.15 mM).

Table 1 indicates that palmitate (0.6 mM) failed to alter the VAT or the duration or type of arrhythmia but significantly prolonged the VT. Because infarcted tissue is hypoxic the effect of combining hypoxia with the FFA was tested. Hypoxia was induced by gassing the Krebs-Henseleit solution with a mixture of 5% CO_2 in air instead of in oxygen. Although hypoxia alone significantly lowered the VAT, when combined with the FFA the effect was magnified as indicated by the significantly lower ($P < 0.05$) VAT change ratio. Both treatments significantly lengthened the VT but while hypoxia alone significantly increased the proportion of arrhythmias which were persistent, when combined with the FFA it had the converse effect.

Table 1 Effect of sodium palmitate and hypoxia on an induced ventricular arrhythmia

Treatment	*VAT change ratio mean ± s.e. mean*	*Arrhythmia Duration persistent/ non-persistent*	*Arrhythmia Type fibrillation/ tachycardia*	*Vulnerable time (ms) mean ± s.e. mean*
Control	1.0	86/137	106/117	82.9 ± 1.57 (223)
Sodium palmitate (0.6 mM)	1.03 ± 0.07 (16)	5/19	6/18	102.1 ± 5.14 (24)[3]
Sodium palmitate (0.6 mM) + hypoxia	0.57 ± 0.03 (13)[3]	3/18[1]	7/14	98.3 ± 6.03 (21)[2]
Hypoxia	0.72 ± 0.04 (16)[3]	13/3[2]	7/9	99.4 ± 7.24 (16)[1]

Number of trials in parentheses.
$P < 0.05$[1], 0.01[2], 0.001[3].

The results suggest that an increased FFA level when combined with hypoxia may predispose to cardiac arrhythmias.

Reference

OLIVER, M.F., KURIEN, V.A. & GREENWOOD, T.W. (1968). Relation between serum-free-fatty-acids (FFA) and arrhythmias and death after acute myocardial infarction. *Lancet*, **1**, 710–714.