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MYOCARDIAL CARNITINE DEFICIENCY IN CHRONIC HEART FAILURE

SIR,—Carnitine is essential for fatty acyl derivatives to penetrate across the inner mitochondrial membrane and be transported to the sites of oxidation in the mitochondria. A reduction in free carnitine and an accumulation of long-chain acyl CoA and long chain acylcarnitine, which are thought to exaggerate myocardial ischaemic damage,[1] have been demonstrated in ischaemic myocardium.[2-5] Replacement of carnitine has been reported to alleviate the accumulation of long chain acyl CoA and long chain acylcarnitine,[4,5] and result in the improvement of energy metabolism and mechanical performance.[3-5]

A reduced level of carnitine has been also reported in the failing guinea pig heart.[6] We have looked for carnitine deficiency in the heart muscle of patients with heart failure.

Tissue levels of carnitine and its derivatives were determined[5] in sixteen biopsy specimens of heart papillary muscle from patients undergoing valve replacement surgery because of chronic heart failure due to mitral valve disease and compared with levels in five post-mortem specimens from patients (controls) without heart disease. The patients with chronic heart failure had less free carnitine, more short and long chain acylcarnitine, and similar total carnitine levels compared with controls (table).

LEVELS OF CARNITINE AND ACYLCARNITINES IN HUMAN LEFT VENTRICULAR PAPILLARY MUSCLE*

	Free carnitine	Acylcarnitine		Total carnitine
		Short chain	Long chain	
Controls	868(±372)	162(±116)	231(±32)	1261(±446)
Heart failure	382(±168)	373(±160)	697(±355)	1452(±363)
p	p<0·001	p<0·05	p<0·01	NS

*Values expressed as nmol per gram wet tissue, as mean±SD.

Because control specimens were obtained 5–10 h (mean 7 h) after death, post mortem changes in the levels of carnitine and its derivatives were studied in five dog hearts incubated at 37°C after death. 6–10 h after death free carnitine levels rose accompanied by a corresponding decrease in short chain acylcarnitine, whereas long chain acylcarnitine and total carnitine remained unchanged. In other words, the significant differences detected in human heart failure (see table) are not likely to be due to post mortem changes in the control samples.

These data point to a decrease in myocardial free carnitine and an increase in long chain acylcarnitine in patients with chronic heart failure, whereas the increase in short chain acylcarnitine may be not significant. Perhaps administration of exogenous carnitine would be worth trying in the treatment of chronic heart failure.

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