MYOCARDIAL CARNITINE DEFICIENCY IN ACUTE MYOCARDIAL INFARCTION

SIR,—Dr Suzuki and colleagues (Jan. 9, p. 116) reported decreased levels of free L-carnitine in the myocardium of chronic heart failure patients and a concomitant rise in acylcarnitine. These changes are related to a removal of accumulated free fatty acid (FFA) and long-chain acyl-CoA esters secondary to chronic hypoxia which in turn inhibits adenine nucleotide translocase.[1] This metabolic device, also observed in dog hearts during the reversible phase of ischaemia[2] seems to act as a servo mechanism tending to relieve myocardial injury. Suzuki et al. suggested the administration of exogenous L-carnitine during acute and chronic cardiac ischaemia. In acute experimental ischaemia, however, progressively more total L-carnitine is lost the longer the ischaemia lasts.[2] The two findings strongly suggest that the maintenance of physiological levels of L-carnitine and the acylcarnitine to free carnitine ratio play an important role in the control of the metabolism of the injured myocardium.

We measured[3,4] heart L-carnitine levels at necropsy in seven patients who had had acute myocardial infarctions and in four who had died from causes other than heart disease. In the first group the tissue samples were removed from the necrotic area, from the border zone, and from the healthy myocardium; whereas in the controls L-carnitine levels were separately determined in specimens taken from the left ventricular walls. The necrotic myocardial areas had lower L-carnitine levels, while the border zone tissue showed intermediate values between necrotic and healthy surrounding tissue levels. There was no discrepancy between the myocardial L-carnitine values in the controls and those found in the healthy surrounding tissue of those who had died from myocardial infarction.

We did not observe short and long chain carnitine esters in the tissue fragments of either group, presumably because in specimens removed 24 h after death all the L-carnitine content is present in the free isomer form, as a result of hydrolysis during the period since death and during storage at −25°C for 10–15 days. Therefore, in our experimental model free L-carnitine corresponds to total carnitine. This statement is supported by the fact that the tissue L-carnitine levels we found in the healthy myocardium were identical to the total L-carnitine levels mesured by Cederblad in the myocardium of heart surgery patients.[5]

LEVELS OF L-CARNITINE IN HUMAN MYOCARDIUM OF THE LEFT VENTRICLE

Site	Free L-carnitine (μmol/g non-collagenous protein): mean ± SEM
Controls	
Anterior wall	6·30 ± 0·47
Posterior wall	7·18 ± 0·38
Septal wall	6·29 ± 0·19
Myocardial infarction	
Infarction area	1·74 ± 0·16*
Peri-infarction area	3·55 ± 0·31*
Healthy area	6·16 ± 0·29

*$p<0·001$

Our data confirm L-carnitine depletion in necrotic areas of myocardium and suggest that the intermediate L-carnitine values (see table) found in the infarct border zones reflect an area of reversible metabolic injury for which restoration of adequate L-carnitine levels could protect the myocardium from damage caused by accumulation of FFA and long-chain acyl-CoA esters.

Institute of Anatomy
and Pathological Histology II,
Department of Ultrastructural Pathology,
University of Rome,
Rome, Italy 00161

LUIGI GIUSTO SPAGNOLI
MARCO CORSI
SERGIO VILLASCHI
GIAMPIERO PALMIERI
FRANCO MACCARI

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