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Assessment of myocardial metabolism with ^{11}C-palmitate. Comparison with ^{123}I-heptadecanoic acid

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Carbon-11 (^{11}C)-palmitate is chemically identical to its physiological counterpart. After intravenous injection the myocardial distribution of ^{11}C-activity can be measured accurately by positron emission tomography. Regions of decreased ^{11}C-palmitate uptake can be readily identified and their size quantified. Results obtained in dogs with experimental coronary thrombosis and in patients with myocardial infarction indicate that positron emission tomography with ^{11}C-palmitate allows non-invasive assessment of the metabolic recovery of the myocardium after lysis of the occluding coronary thrombus.

There is experimental evidence that the rate of clearance of ^{11}C-palmitate activity from the myocardium is related to oxidative fatty acid metabolism. In dogs, a restriction of the oxygen supply to the myocardium results in a decrease in the rate of ^{11}C-clearance independently of whether myocardial perfusion is concomitantly reduced or not.

Similarities in myocardial uptake and clearance exist between iodine-123 (^{123}I)-heptadecanoic acid and ^{11}C-palmitate. However, interpretation of the kinetics of the radio-iodinated fatty acid analogue has to take into account the different intracellular fate of the iodine label compared with the fatty acid structure.

Efforts to 'metabolically image' the myocardium with labelled fatty acids were first suggested by Evans *et al.*[1]. The growing interest in imaging radio-iodinated fatty acid analogues over the past few years has closely paralleled the development of positron emission tomography (PET)[2–5]. The introduction of cyclotron produced carbon-11 (^{11}C; half-life 20·4 min) into biomedical research permitted for the first time the ability to label metabolic substrates such as palmitate with an isotope which is detectable externally, and which does not alter the biochemical behaviour of the molecule. The physiological properties of ^{11}C-palmitate together with the imaging characteristics of PET have provided a promising approach for non-invasive assessment of regional myocardial fatty acid metabolism in patients[2,4,6–12].

The hypothesis underlying this approach is that metabolic alterations are reflected in modifications of externally detectable uptake and clearance of radioactivity of ^{11}C-palmitate by the myocardium. It is obvious that factors other than oxidative metabolism, including tracer supply, circulating metabolites and washout of labelled molecules, may also influence the time course of myocardial radioactivity. Therefore ^{11}C-palmitate has been studied in experimental animals aimed at defining the interrelations between myocardial fatty acid metabolism and externally detectable tracer kinetics.

The following discussion of ^{11}C-palmitate is intended (1) to provide a review of experimental findings, (2) to summarize clinical applications and (3) to provide a comparison with studies employing iodine-123 (^{123}I)-heptadecanoic acid.

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Positron emission tomography versus single photon imaging

Positron emission tomography takes advantage of the decay characteristics of this class of isotopes: each positron 'annihilates' with an electron in close vicinity to the nuclear decay. As a result of this interaction two gamma photons are emitted in diametrically opposed directions. Recording of coincidence events (i.e. the detection of gamma photons at nearly the same instant) by multiple pairs of opposed detectors arranged around the chest permits reconstruction of the distribution of activity within the body in tomographic cross-sections. Sequential tomographic imaging allows analysis of tracer kinetics in well defined regions of interest without distortion by tracer in superimposed tissue, a limitation inherent to planar imaging techniques[2,12-15]. In addition, since the coincidence count rate, unlike the single photon count rate, is independent of the position of the radioactive source between the detectors, the local tissue tracer concentration can be measured in terms of microcuries per gram of tissue[2,12-15]. Difficulties in attenuation correction represent the principal limitation of quantification of tissue isotope concentration by single photon emission tomography[15].

Routine use of PET is limited by the costs involved in the tomographic instrument itself and in the production of the short lived tracers requiring an on site cyclotron[16].

Experimental studies with ^{11}C-palmitate

MYOCARDIAL UPTAKE

After intravenous injection in dogs, ^{11}C-palmitate accumulates in normal myocardium and disappears rapidly from blood permitting clear delineation of the myocardium in tomographic images[8]. During complete coronary occlusion[5,12] or after production of critical coronary narrowing followed by atrial pacing[17], a defect in ^{11}C-palmitate uptake is apparent in the ischaemic region. ^{11}C-palmitate uptake is determined by the arterial input function, myocardial perfusion and the extraction fraction. Since the arterial tracer concentration is identical for the entire myocardium, the latter two factors have to be considered to explain the regional differences noted in ^{11}C-palmitate uptake during ischaemia. It is obvious that myocardial blood flow and hence tracer supply is reduced in ischaemic compared with normal myocardium. However, in the same region, myocardial work and oxidative metabolism, two major determinants of fatty acid uptake, are reduced concomitantly[18]. In isolated perfused rabbit hearts maintained at a constant work load and oxygen consumption, the extraction fraction of ^{14}C-palmitate is inversely related to flow resulting in constant net uptake over a flow range between 0·5 and 1·5 ml min^{-1} g^{-1}[19]. However, at a given flow rate uptake is increased if work load is augmented. Thus, the myocardial distribution of activity after administration of ^{11}C-palmitate does likely not primarily reflect myocardial perfusion, but rather delineates zones of altered fatty acid uptake independent of whether flow is altered.

Because of the capability of PET to accurately quantify the size of uptake defects[20] and the dependence of myocardial ^{11}C-palmitate uptake on metabolic activity, this approach is promising for the non-invasive assessment of the extent of myocardial salvage achieved by reperfusion of ischaemic regions[21]. In a dog model of coronary thrombosis intracoronary streptokinase infusion resulted in a marked increase of ^{11}C-palmitate uptake in the region of the previous defect if perfusion was re-established between 1 and 2 hours after occlusion (Fig. 1)[9]. Recovery of ^{11}C-palmitate uptake was progressively attenuated with prolongation of occlusion and was virtually absent if reperfusion was instituted later than 6 h after occlusion despite an average recovery of myocardial perfusion to 65% of normal[9,22]. The time course of the development of irreversible depression of ^{11}C-palmitate uptake is similar to the progression of irreversible damage determined by histochemical criteria[23]. Nevertheless, ^{11}C-palmitate may underestimate the mass of viable myocardium since enhanced accumulation of ^{18}F-2-fluoro-2-deoxyglucose and ^{11}C-glucose has been observed in some regions of depressed ^{11}C-palmitate uptake[2,3].

CLEARANCE OF MYOCARDIAL ACTIVITY

In dogs clearance of ^{11}C-palmitate remaining in the myocardium after the initial vascular transit typically exhibits a biexponential pattern with an early rapid phase between 5 and 10 min and a slow phase, evident later than 20 min after tracer injection (Fig. 2)[8,24,25]. This behaviour is compatible with incorporation of tracer into at least two different lipid compartments[26]. In normal myocardium more than 90% of activity appearing during the early rapid component in the coronary venous blood is in form of $^{11}CO_2$[27]. Furthermore in the fasting state,

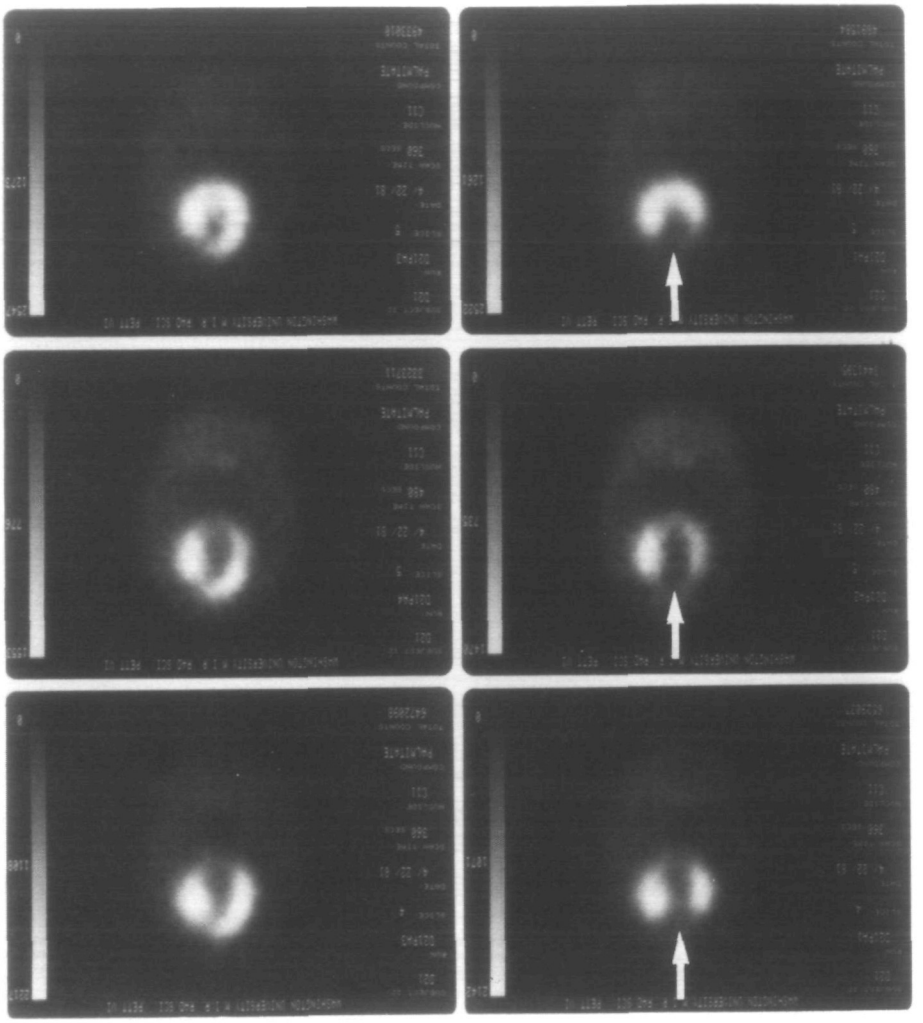

Figure 1 Midventricular transverse positron emission tomograms of a dog heart after intravenous injection of ^{11}C-palmitate. The images on the left hand side were obtained 1·5 h after occlusion of the left anterior descending coronary artery by an experimentally induced thrombus. There exists a large uptake defect in the anterior wall (arrow). Streptokinase was then infused intracoronarily and a second ^{11}C-palmitate injection followed by a second tomogram performed one and a half hours after thrombolysis (images on the right). After reperfusion the defect fills in substantially indicating myocardial salvage (from Bergmann *et al.*, Am J Med 1983; 73: 573–81. Reproduced with permission).

the fraction of extracted activity released as $^{11}CO_2$ is closely related to myocardial oxygen consumption[25,27]. Therefore oxidative metabolism is responsible for the bulk of clearance of activity from the normal myocardium.

The effect of ischaemia on myocardial time–activity curves was assessed in open chest dogs with controlled perfusion of a coronary artery and continuous monitoring of myocardial activity by a positron detector after intracoronary ^{11}C-palmitate injection[24]. After reduction of myocardial perfusion by 76%, sufficient to induce lactic acid production, the slope of ^{11}C-clearance during the interval of the early component was consistently decreased com-

pared to control conditions[24]. A reduction of early ^{11}C-clearance of similar magnitude was observed when oxygen supply was restricted to a comparable extent at normal flow by hypoxic perfusion (Fig. 2)[24]. Therefore decreased washout of labelled metabolic products appears not to be primarily responsible for the decreased clearance of activity from ^{11}C-palmitate during ischaemia. Decreased release of myocardial activity is mostly compatible with impairment of fatty acid oxidation and predominant incorporation of tracer into storage lipids such a triglycerides[26]. Further evidence for the predominant role of oxidative metabolism for ^{11}C-clearance has been provided by dog experiments demonstrating decreased clearance of activity during demand induced ischaemia at unaltered perfusion[17] and during suppression of fatty acid oxidation by infusion of glucose and insulin[28]. Thus, reduced fatty acid oxidation is reflected in reduction of the externally detectable clearance of activity from ^{11}C-palmitate. However, limitations for quantification of fatty acid oxidation in absolute terms from time–activity curves include variable back-diffusion of non-metabolized fatty acids which becomes prominent during ischaemia and hypoxia[27].

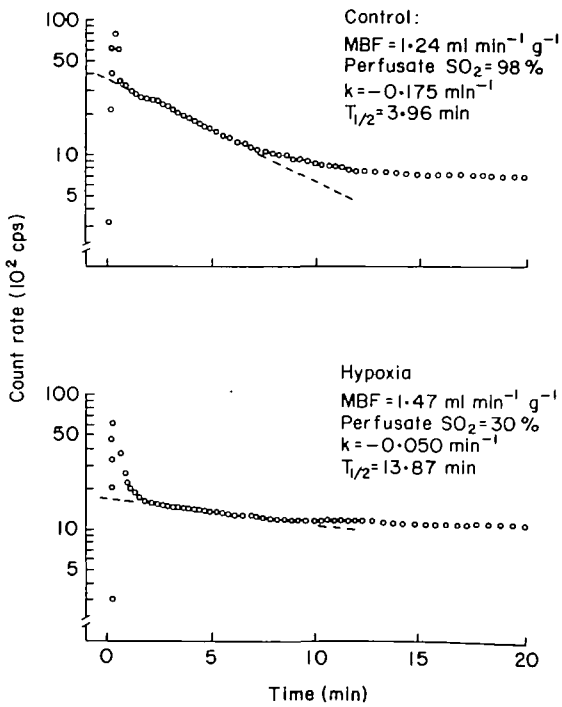

Figure 2 Semilogarithmic plots of regional time–activity curves obtained with a positron detector in an open chest dog with controlled perfusion of the left anterior descending coronary artery by an extracorporeal circuit. Under control conditions (top panel) clearance of myocardial activity exhibits three components: (1) vascular transit of non-extracted tracer; (2) early rapid myocardial phase evident between 5 and 10 min after injection; (3) late slow phase of clearance. During perfusion at normal flow rate with venous blood (bottom panel) exhibiting a haemoglobin saturation with oxygen (SO_2^1) of 30% the rate of early monoexponential ^{11}C-clearance is markedly decreased indicating impaired fatty acid oxidation. MBF = myocardial blood flow; $t_{\frac{1}{2}}$ = half-time of rapid early monoexponential clearance; k = rate constant of clearance (from Lerch *et al.*, Circulation 1982; 65: 731–8).

Patient studies with ^{11}C-palmitate

PET with ^{11}C-palmitate has permitted detection of infarcted regions in patients with recent or remote myocardial infarction[4,10,11]. In the case of electrocardiographically defined non-transmural infarction the sensitivity of PET is considerably higher than conventional scintigraphy with thallium-201 in the same patients[10]. Tomographic defect size correlated closely with infarct size estimated from serial plasma MB-CK determinations[11]. Among infarct patients undergoing ^{11}C-palmitate studies before and after administration of a thrombolytic agent, defect size decreased significantly in the group with successful myocardial reperfusion but remained unchanged in the group without thrombolysis[6]. Finally, inhomogeneous ^{11}C-palmitate uptake has been observed in patients with dilated cardiomyopathy[7].

Recently developed positron emission tomographic devices for cardiac studies in patients permit scanning times short enough for the determination of myocardial ^{11}C-palmitate time-activity curves in human hearts[29]. Initial results indicate that clearance of myocardial activity is biexponential as previously observed in dogs[29,30]. In addition, the rate of early clearance decreases markedly after glucose administration[30] which in view of the previous dog experiments is most likely an expression of decreased fatty acid oxidation.

^{123}I-heptadecanoic acid and ^{11}C-palmitate

SIMILARITIES

Among several radiohalogenated fatty acid analogues, ^{123}I-heptadecanoic acid has initially been

selected for clinical investigation because of similar uptake and clearance of activity compared to ^{11}C-palmitate in the myocardium of mice[31]. Studies in patients employing scintigraphy or limited angle tomography show clear visualization of the left ventricle 5 minutes after intravenous injection of ^{123}I-heptadecanoic acid[32-35]. Zones of infarction are readily detectable [32, 35]. Clearance of myocardial activity has been reported to exhibit a biexponential pattern[35] with a half-time of the early phase[33] that closely agrees to the one observed in patients studied with ^{11}C-palmitate[30]. The slope of the early component was decreased during stress in regions corresponding to stenosed coronary arteries in patients with ischaemic heart disease[33] and in normal myocardium after glucose/insulin infusion[35]. Furthermore, inhomogeneous uptake and clearance of activity from ^{123}I-heptadecanoic acid has been observed in patients with dilated cardiomyopathy[36]. These findings are very similar to those obtained in comparable studies with ^{11}C-palmitate despite the intrinsic differences in the radiopharmaceuticals used as well as in the imaging methods. Other observations with ^{123}I-heptadecanoic acid include delayed clearance of activity at rest in patients with coronary artery disease[35] and increased rate of clearance in regions of myocardial infarction[32]. Furthermore, in dogs, the rate of clearance was decreased during beta blockade[37] and after administration of toxic doses of doxorubicin[38]. Studies with ^{11}C-palmitate comparable to these latter investigations have not been published so far.

THE DIFFERENCE

Because of the similarity of myocardial uptake and clearance of activity between ^{123}I-heptadecanoic acid and ^{11}C-palmitate, it is tempting to speculate that the kinetics of both tracers are dominated by identical physiological, metabolic processes. Nevertheless, in contrast to ^{11}C-palmitate, ^{123}I-heptadecanoic acid is not a natural substrate of fatty acid metabolism. Therefore differences in the chemical and physical properties of iodinated intermediates or end products of metabolism compared with their physiological counterparts may modify the response to metabolic alterations and findings between the two tracers cannot directly be related.

For example, production of iodide-123 released into the coronary circulation from extracted ^{123}I-heptadecanoic acid may respond differently to metabolic alterations than formation $^{11}CO_2$ from ^{11}C-palmitate. Deiodination independent of metabolic degradation of the fatty acid structure has been suggested by some authors based on observations with structurally modified terminally iodinated fatty acid analogues which are not substrates for beta-oxidation[39]. Such non-specific deiodination would limit the use of ^{123}I-heptadecanoic acid to probe oxidative fatty acid metabolism based on analysis of myocardial clearance curves. However, in isolated perfused rat hearts, myocardial release of iodide-123 from ^{123}I-heptadecanoic acid was reduced to a similar extent as $^{14}CO_2$ production from ^{14}C-palmitate during cardioplegia or inhibition of fatty acid oxidation by phenylalkyloxirane carboxylic acid (POCA)[40]. Thus, deiodination of ^{123}I-heptadecanoic acid appears to be related to oxidative fatty acid metabolism.

Another factor which may preclude recognition of variations in cellular deiodination of ^{123}I-heptadecanoic acid is slow membrane transport leading to prolonged retention of produced iodide-123 in the myocyte. Based on several experimental studies supporting this possibility[41,42], it has been proposed that observed alterations in myocardial ^{123}I-clearance reflect altered membrane properties rather than altered fatty acid metabolism[41,43]. Nevertheless, the relative importances of factors potentially modifying externally detectable myocardial kinetics of ^{123}I-heptadecanoic acid under conditions such as ischaemia, which include myocardial perfusion, metabolism and membrane integrity, remain to be defined.

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