《招待講演》

Cardiac Applications of PET

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Myocardial blood flow

The non-invasive nature of PET and the low radiation exposure allow the measurement of absolute myocardial blood flow (MBF) in normal subjects.

$H_2{}^{15}O$ and $^{13}NH_3$ are the most widely used tracers for quantification of MBF.[1–2] Measurements of MBF in normal volunteers both during rest and following maximal drug-induced vasodilatation are similar using either tracer.[3] Both tracers have short half-lives, thus allowing repeated measurements of MBF within the same scanning session. The high reproducibility of such measurements using $H_2{}^{15}O$ has been clearly established.[4]

The ratio of absolute MBF during near-maximal vasodilatation (adenosine or dipyridamole) to resting MBF (the coronary vasodilator reserve, or CVR) permits the assessment of the functional significance of coronary stenoses in patients with CAD.[2,5] CVR starts to fall when stenosis severity rises above 40% and is exhausted for stenoses ≥80%. In the absence of significant stenoses CVR mainly reflects the function of the coronary microcirculation. For instance CVR is severily blunted, in patients with hypertrophic cardiomyopathy or secondary LVH despite angiographically normal epicardial coronary arteries.[6]

Identification of hibernating myocardium

The term ‘hibernating myocardium’ was originally used to describe dysfunctional segments of the left ventricle, which recovered contractility after revascularisation of the coronary arteries subtending such dysfunctional segments.[7–9] The greatest clinical benefit is seen in those patients with the most severe dysfunction. It is therefore important to use techniques that offer a high sensitivity for identifying hibernating myocardium. The identification of hibernating myocardium using PET requires the recognition of myocyte viability i.e. preserved glucose uptake (using ^{18}FDG) in dysfunctional myocardial segments.[10] Glucose uptake depends upon several factors

including dietary state, cardiac workload, sensitivity of the myocardium to insulin, sympathetic drive, and the presence and severity of ischaemia.[2] This variability confuses the interpretation of ^{18}FDG uptake data. Insulin resistance is common in patients with CAD and reduces ^{18}FDG image quality. These problems are partly overcome by use of the '*hyperinsulinaemic euglycaemic clamp*'[10] which results in optimal image quality and allows comparison of the absolute rates of glucose uptake between different patients and centres.

Using PET with ^{18}FDG during hyperinsulinaemic euglycaemic clamp, we have shown that a metabolic rate of glucose $\geq 0.25\ \mu mol/g/min$ is the best predictor of improvement in functional class after revascularisation.[11]

Function of the Autonomic Nervous System

The non-selective β-adrenoceptor antagonist ^{11}C-CGP 12177 has a high affinity for its receptor and a low lipophilicity, thus enabling the cell surface receptor pool to be quantified (pmol/g). It has been used to demonstrate diffuse down-regulation of myocardial β-adrenoceptors in hypertrophic cardiomyopathy and congestive cardiac failure.[12] PET has also been used to investigate the integrity of cardiac pre-synaptic sympathetic innervation with ^{11}C-HED which competes with noradrenaline for transport into the pre-synaptic sympathetic nerve terminal via uptake-1 mechanism. Decreased retention of ^{11}C-HED has been demonstrated in patients following heart transplantation.[13] Both pre and post synaptic myocardial autonomic function can be assessed noninvasively by combining different tracers, e.g. ^{11}C-HED and ^{11}C-CGP 12177.[14]

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