
Effect of Nicotinic Acid on Exogenous Myocardial Glucose Utilization

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Clinical assessment of myocardial glucose uptake with ^{18}F -fluorodeoxyglucose (^{18}F -FDG) and PET requires the control of circulating substrates to achieve acceptable image quality. **Methods:** To determine the efficacy of the hypolipemic effect of oral niacin upon myocardial ^{18}F -FDG uptake, five volunteers were studied with ^{18}F -FDG and PET in the fasting state, with and without treatment with niacin. Levels of glucose, fatty acids, insulin and catecholamines were measured at baseline and before and after ^{18}F -FDG administration by programmed infusion. **Results:** No significant changes in glucose or insulin levels occurred with niacin. A significant decrease in fatty acid levels with niacin treatment was associated with a two- to three-fold increase in myocardial glucose utilization rates relative to the fasting state. Furthermore, regional variation in tracer distribution with greater uptake in the lateral wall than the septum or anterior wall in the fasting studies was not present after niacin treatment. **Conclusion:** As determined by programmed infusion of ^{18}F -FDG and PET imaging, niacin treatment in normal volunteers was associated with an increase in exogenous glucose utilization by the heart and a decrease in the cardiac regional variation of ^{18}F -FDG. Further studies are needed to compare the relative value of niacin therapy and oral glucose loading for determination of myocardial exogenous glucose utilization rates.

Key Words: glucose; cardiac metabolism; fluorine-18-fluorodeoxyglucose; positron emission tomography

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The rates of myocardial glucose uptake and metabolism depend on levels of circulating substrates and hormones, on membrane glucose transporters, and on the energy requirements of the heart (1-4). Glucose uptake by the human heart is increased by exercise (2), insulin (3-5) or by a fall in plasma free fatty acids (6), and decreased by an increase in circulating free fatty acids (5). Free fatty acids are thought to exert their effects by raising mitochondrial acetyl-CoA levels, inhibiting pyruvate dehydrogenase activity by increasing cytosolic citrate and inhibiting phos-

phofructokinase activity. Thus, a decrease in plasma free fatty acid concentration removes inhibitions on glycolysis and pyruvate oxidation and stimulates glucose uptake and oxidation.

Since the preferred substrate for the heart is fatty acids, imaging of the heart with ^{18}F -fluorodeoxyglucose (^{18}F -FDG) using PET has been performed in either the basal state or with alteration of glucose and insulin levels to promote glucose uptake (7). Oral glucose loading has been the preferred method of imaging at many centers because of the improvement in myocardial uptake (8). Variability in the absorption of glucose and the magnitude of the insulin secretory response to the glucose load has prompted the development of other means of stimulating myocardial glucose uptake to allow the comparison of ^{18}F -FDG uptake in different myocardial pathologic conditions. One means of standardization has been the use of an insulin/glucose clamp to rigorously control circulating insulin and glucose levels (9,10). Despite this technique intersubject variability in myocardial glucose utilization rates has persisted (11). As an alternative to controlling the levels of circulating glucose for the standardization of cardiac ^{18}F -FDG uptake, a second method may be the use of oral niacin to stimulate myocardial glucose uptake by decreasing free fatty acid availability (6,12). This second method for standardizing ^{18}F -FDG uptake would have the advantage over the insulin/glucose clamp technique of its ease of use.

The standard method of administration of ^{18}F -FDG has been as an intravenous bolus, causing rapid changes in plasma radioactivity concentration with the appearance and clearance of the bolus, and significant spillover of radioactivity signal from ventricular chambers to myocardial regions during the early frames when myocardial activity is low. These effects can be controlled by administration of ^{18}F -FDG by programmed infusion with rates intended to maintain constant plasma levels of radioactivity (13) and by graphical analysis of the time-course data for the estimation of glucose uptake rates (14).

The objective of this study was to determine whether the acute hypolipemic effect of oral niacin in fasting normal volunteers was accompanied by an increase in ^{18}F -FDG uptake with PET imaging. The magnitude and regional variability of this uptake was compared to those in fasting

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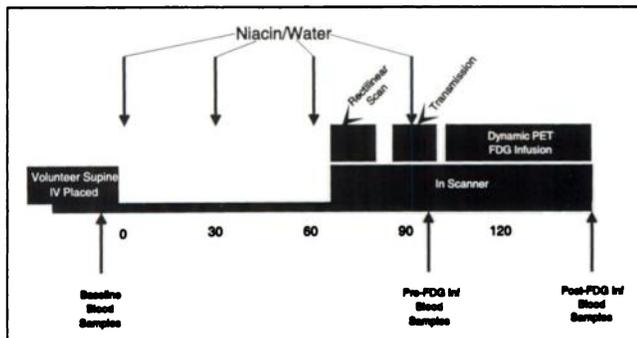


FIGURE 1. Protocol time line with timing sequence for niacin or water administration, blood sampling and ^{18}F -FDG scanning. Subjects received either water alone or water with 250 mg of niacin tablets at 0.5-hr intervals prior to the injection of ^{18}F -FDG. Blood was drawn at the time points shown for glucose, FFA, insulin and catecholamine levels.

studies performed in the same volunteers. Programmed infusion of ^{18}F -FDG was performed for the study. The standard clinical protocol of an oral glucose load and bolus administration of ^{18}F -FDG was performed in a second group of volunteers for a comparison with the niacin method.

METHODS

The study was reviewed and approved by the Human Subjects Committee of the University of Wisconsin-Madison. Volunteers were solicited through newspaper advertisements and were screened for any history of medical problems or current illnesses. Six normal volunteers (5 men; mean age 34 ± 4 yr) were recruited and studied with a paired protocol design. All participants gave informed consent. Following an overnight fast (9–14 hr), the volunteers underwent a brief history, physical exam and 12-lead electrocardiogram. Intravenous access was established with 20-G angiocaths in the dorsum of the right hand and the left antecubital fossa. Blood samples were drawn for the determination of glucose, catecholamines, free fatty acids and insulin levels (Fig. 1). At 30-min intervals, either niacin [250 mg with 100–150 cc of water (niacin intervention study)] or water alone (control study) was given orally. After the third dose of niacin or water, the volunteer was positioned in the ECAT 933/04 PET scanner (CTI, Knoxville, TN) using a transmission rectilinear scan. A 10-min transmission scan was obtained with a ^{68}Ge ring source for attenuation correction of the emission data. Blood sampling was repeated for substrate, hormone and catecholamine levels. ^{18}F -FDG infusion (10 mCi) was performed using programmed infusion. The infusion time course was calculated using conventional methods from a characteristic actual arterial time course previously measured in a fasted subject. The 10-ml infusate volume was infused at rates ranging from 1.5 ml/min at the start of the study down to 0.080 ml/min at the end. A dynamic PET image sequence of ten 5-min frames was collected during the 50-min infusion period.

Arterialized venous blood samples were drawn from the dorsal hand line with the hand and forearm placed in a hand warmer. Samples were taken at 1-min intervals for the first 5 min and at 5-min intervals thereafter. Samples were placed on ice and rapidly

centrifuged. Standard aliquots of plasma were used to determine the time course of radioactivity concentration. A final set of samples was drawn at the completion of scanning to determine insulin, substrate and catecholamine levels. These and the previous blood samples were stored at -70°C until analyzed.

One week later, the imaging protocol was repeated with the second intervention given (either niacin or water). Imaging protocol and blood sampling were performed as in the first study.

Image quality and glucose uptake rates with niacin treatment were compared to those from a second group of five fasting normal volunteers studied with the standard clinical protocol of bolus administration of ^{18}F -FDG following ingesting 50 g of glucose in solution.

Biochemical Analyses

Norepinephrine and epinephrine levels were determined by high-pressure liquid chromatography with electrochemical detection (15,16). Plasma insulin levels were determined by radioimmunoassay (Diagnostic Products Corp., Los Angeles, CA) (4) and nonesterified (free) fatty acid levels were measured by spectrophotometric enzymatic assay (Wako Chemicals USA, Inc., Richmond, VA). Plasma glucose levels were measured by a glucose oxidation assay (CX3-Delta Analyzer, Beckman Instruments, Inc., Brea, CA) (17).

Data Analysis

Estimation of glucose uptake rates from the PET time course data was performed with graphical analysis (2,14,18). The myocardial uptake rates K_i for ^{18}F -FDG were first estimated from the relation:

$$\frac{C_i}{C_p} = K_i \frac{\int_0^T C_p dt}{C_p} + b,$$

where C_i is the myocardial radioactivity concentration and C_p is the plasma ^{18}F radioactivity concentration at time T . Plots of C_i/C_p versus “stretch time,” the integral of C_p from 0 to time T divided by C_p at time T , were fitted to straight lines by conventional least-squares methods, and the slopes of the best-fit lines taken as estimates of K_i . Frames ranging from 15 to 50 min after the start of infusion were included in the fit. The myocardial glucose uptake rate (GUR) was calculated from the K_i values by

$$\text{GUR} = \frac{P_{\text{Glu}} K_i}{LC},$$

where P_{Glu} is the plasma glucose concentration and LC the lumped constant. A LC value of 0.67 was assumed (19).

For determination of C_i , regions of interest (ROIs) within the myocardial border were drawn in the septum, anterior wall and lateral wall from three contiguous midventricular transaxial slices in the niacin study of each subject. Region boundaries were determined from the iso-intensity contour corresponding to 50% of the peak value. The ROI contours were transported to the control study and overlaid on the matched myocardial sections. Position of the regions was confirmed on the control study by review of the last dynamic frame where myocardial radioactivity was maximal. No correction was made for the roughly 30% reduction in average radioactivity concentration expected for regions of this size due to partial volume effects. These effects are expected to be identical for both the control and niacin groups. C_p was obtained from the

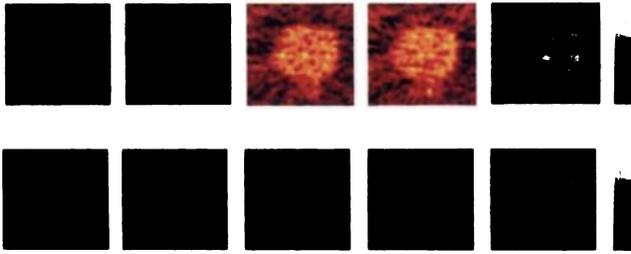


FIGURE 2. Five-minute dynamic PET scans at the same mid-ventricular level in the same fasting volunteer after receiving water (top row) or niacin (bottom row) prior to ^{18}F -FDG infusion. The five columns correspond to frames starting at 5, 15, 25, 35 and 45 min after the start of infusion. Orientation of the scans is anterior of the thorax at the top of the image and the right side of the subject on the left side of the image.

blood samples. GUR were averaged over the three axial slices to yield average values corresponding to the septal, anterior and lateral wall regions. A parametric image of glucose uptake rate was calculated in one midventricular transaxial plane by performing the estimate of K_i in each image pixel.

Statistical Analyses

Uptake rate data were expressed as mean \pm s.e.m. The paired Student's t-test was used to compare values within the niacin intervention study, and the unpaired t-test was used to compare the GUR values between the niacin intervention study and the separate group of glucose-fed subjects. A p level of less than 0.05 was accepted as significant.

RESULTS

Serial 5-min dynamic images at the midventricular level demonstrated little myocardial uptake of ^{18}F -FDG in the fasting state with an increase in uptake after niacin. Representative dynamic image sequences from one subject are presented in Figure 2. Example data analysis from the same subject is shown in Figures 3 and 4. Plasma radioactivity (C_p) from the arterialized venous blood samples demonstrated the expected plateau with the programmed infusion of ^{18}F -FDG. This plateau of plasma radioactivity was also seen in the ventricular chambers in the dynamic im-

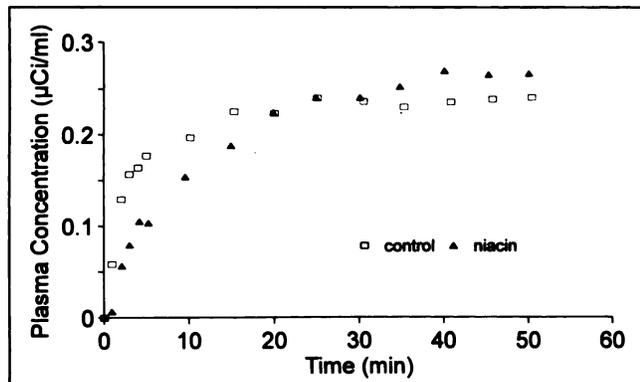


FIGURE 3. Plasma radioactivity concentration curves from the studies shown in Figure 2. These curves demonstrate the plateau of radioactivity with the programmed infusion of ^{18}F -FDG.

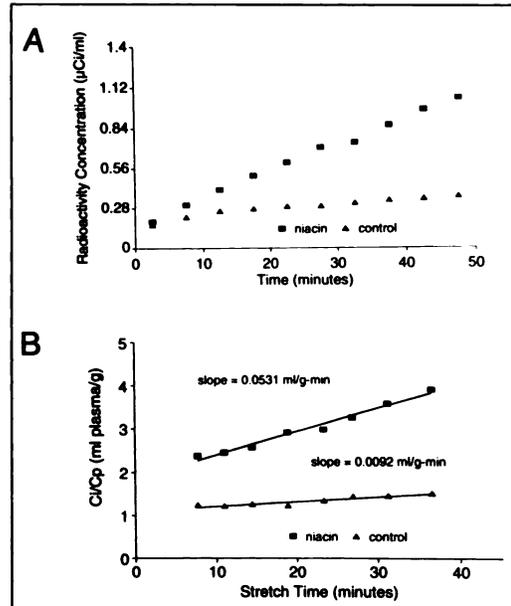


FIGURE 4. Representative myocardial region of interest time courses from the control and niacin studies shown in Figures 2 and 3. (A) Region radioactivity concentration and (B) graphical analysis plot. Linear regression was performed to determine the slope on the graphical analysis plot.

ages in both the control and niacin groups (Fig. 2). Because the same infusion schedule was used for all studies, the approach to plateau was slower on the average after niacin (time to plateau: 10 ± 2 min for control studies versus 16 ± 5 min following niacin), presumably due to the more avid peripheral uptake of the tracer. In the myocardium, increases were consistently observed in both radioactivity concentration (Fig. 4A) and in K_i values (Fig. 4B) after niacin. For example, ratios of radioactivity concentrations in septum to the plasma for the tenth dynamic frame (47.5 min infusion time) increased from 1.5 ± 0.1 to 3.1 ± 0.3 ($p < 0.01$) following niacin treatment. Image acquisition was unsatisfactory in one volunteer due to motion during the scan and inadequate ^{18}F -FDG uptake, and his data were excluded from final analysis of glucose utilization rates.

For the five subjects, a two- to three-fold increase in glucose uptake occurred with niacin treatment (Fig. 5). In the fasting state, glucose utilization was significantly higher in the lateral wall than the septum (0.16 ± 0.01 versus 0.12 ± 0.01 $\mu\text{mole/g/min}$, $p < 0.05$). With niacin treatment, a significant increase in glucose utilization occurred in all three myocardial regions. Uptake rates showed the greatest increase in the septum, to 0.36 ± 0.03 $\mu\text{mole/gm/min}$ ($p < 0.01$) for an increase of $230\% \pm 40\%$. Lateral wall utilization also increased to 0.39 ± 0.03 ($p < 0.01$) for an increase of $160\% \pm 40\%$. After niacin treatment, there was no significant difference between septal and lateral wall glucose utilization rates. Thus, the regional variation of ^{18}F -FDG uptake present in the fasting control studies was not present with niacin treatment (Fig. 5).

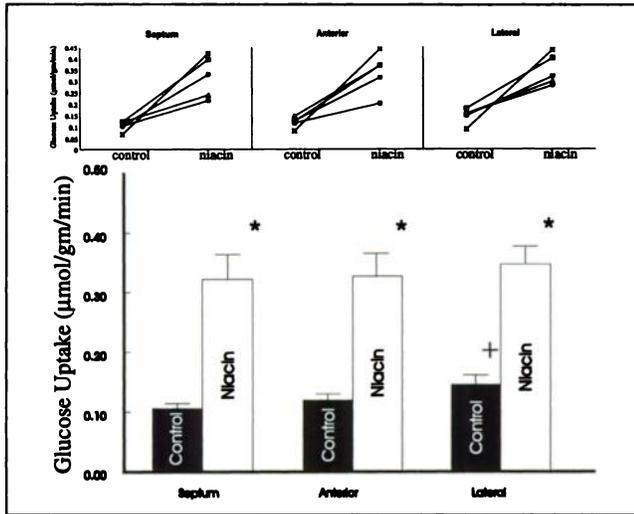


FIGURE 5. Glucose uptake rates (GUR) in control and niacin intervention studies by graphical analysis. A significant increase in GUR was seen in all three myocardial regions after niacin (* $p < 0.01$ niacin versus control GUR within region). Although GUR was decreased in the septum relative to the lateral wall in the control group ($^+p < 0.05$ lateral wall versus septum), this difference was abolished after niacin.

No change was seen in insulin or plasma glucose levels after administration of niacin (Table 1). An increase in epinephrine levels relative to initial baseline levels occurred by the end of the 50-min scanning period in both groups while an increase in norepinephrine occurred in the niacin intervention group alone (Fig. 6). Facial and/or truncal flushing was noted after niacin but not with water alone.

Circulating free fatty acid levels decreased in the niacin group relative to control group (Fig. 7). In the control study, fatty acid levels gradually increased throughout the study from 0.36 ± 0.06 to $0.50 \pm 0.13 \mu\text{M/ml}$; with niacin therapy, fatty acid levels declined to $0.24 \pm 0.06 \mu\text{M/ml}$ prior to the start of imaging, and $0.27 \pm 0.06 \mu\text{M/ml}$ at the end of the procedure, a significant depression compared to the control study at the same time points ($p < 0.05$). An inverse correlation of glucose utilization rates and fatty acid levels prior to infusion of $^{18}\text{F-FDG}$ was present ($R = -0.54$, $p < 0.05$) for the control and niacin studies. There

TABLE 1
Insulin and Glucose Levels

	Control	Niacin
Insulin levels ($\mu\text{U/ml}$)		
Baseline	22.3 ± 1.2	19.6 ± 3.6
Pre-FDG Inf	21.4 ± 0.7	18.5 ± 3.1
Post-FDG Inf	21.1 ± 0.7	18.3 ± 3.0
Glucose levels (mg/dl)		
Baseline	91.5 ± 3.9	95.5 ± 3.4
Pre-FDG Inf	91.8 ± 3.0	89.8 ± 4.7
Post-FDG Inf	91.0 ± 2.3	95.7 ± 2.6

Values are mean \pm s.e.m. Inf = infusion.

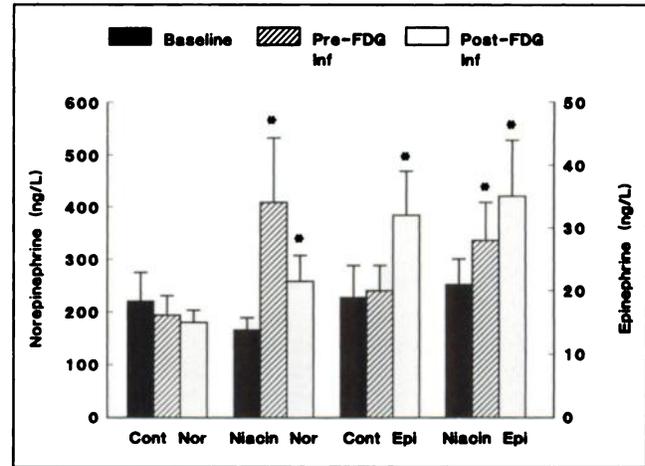


FIGURE 6. Plasma catecholamine (norepinephrine and epinephrine) levels in control and niacin intervention studies. Pre-FDG inf = pre- $^{18}\text{F-FDG}$ infusion; post-FDG inf = post- $^{18}\text{F-FDG}$ infusion; nor = norepinephrine; epi = epinephrine; * $p < 0.05$ value versus corresponding baseline value. A significant increase in epinephrine levels occurred in both studies, while norepinephrine was increased only in the niacin study.

was no correlation of glucose utilization rates with pressure rate product ($R = 0.08$), glucose level ($R = 0.24$) or insulin level ($R = -0.01$).

Although the stability of C_p with the programmed infusion protocol simplifies the graphical analysis for determination of glucose utilization rates, the higher plasma levels of $^{18}\text{F-FDG}$ lessens the myocardial-to-blood pool radioactivity ratio which degrades image quality in the late dynamic frames. Parametric slope images were calculated to decrease the effect of the constant blood-pool activity of $^{18}\text{F-FDG}$ and highlight the increase in C_i over time (Fig. 8). The quality of these parametric slope images after niacin treatment was similar to that of the summed dynamic images of a separate group of normal volunteers studied with a standard clinical protocol of bolus administration of $^{18}\text{F-FDG}$ after 50 g of glucose. Absolute glucose utilization

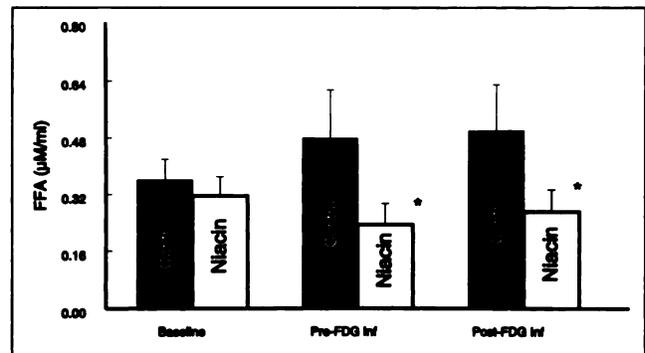


FIGURE 7. Free fatty acid levels after water and niacin administration. Although fatty acid levels were the same at baseline prior to the two interventions, fatty acid levels increased in the control group and decreased with niacin, yielding a significant difference in levels before and after the PET dynamic scan * $p < 0.05$ versus control value at the same time point.

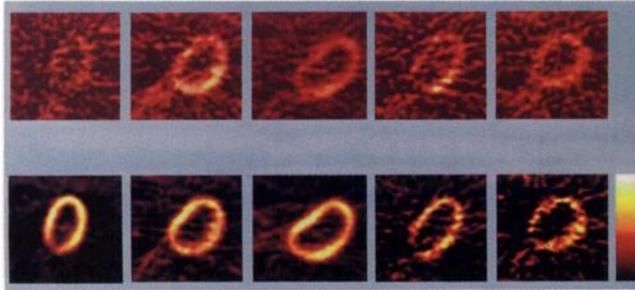


FIGURE 8. Parametric images of glucose uptake rates by pixel-by-pixel graphical analysis for the five subjects. Image intensities in the control (top row) and niacin (bottom row) studies were displayed with a common scale for each subject. Maximal myocardial glucose uptake for all five studies was in the niacin image, which is consistent with a significant increase in uptake rate in the niacin study compared to the control (water only) study for each subject.

rates after niacin, however, were less than rates obtained with the standard clinical protocol; for instance, septal uptake was $0.36 \pm 0.03 \mu\text{mole/g/min}$ for the niacin group versus 0.49 ± 0.03 for the glucose-fed volunteers ($p < 0.05$). The glucose-fed volunteers were studied at a similar cardiac workload as the niacin subjects (8175 ± 1370 versus $6561 \pm 176 \text{ mmHg} \cdot \text{b/min}$). As expected, plasma glucose levels were significantly elevated prior to FDG infusion in the glucose-fed volunteers compared to the niacin-treated subjects ($152 \pm 19 \text{ mg/dl}$ versus $90 \pm 5 \text{ mg/dl}$, $p < 0.05$). All other hormonal (norepinephrine, $176 \pm 24 \text{ pg/ml}$; epinephrine, $12 \pm 3 \text{ pg/ml}$; insulin, $52 \pm 21 \text{ IU/ml}$) and substrate (free fatty acids, $0.33 \pm 0.04 \mu\text{M/ml}$) levels for the glucose-fed volunteers prior to FDG infusion were similar to the niacin-treated subjects.

DISCUSSION

These results demonstrate an increase in exogenous glucose utilization after niacin administration. This increase appears to be due to the hypolipemic effect of niacin since no change in circulating insulin or glucose levels was seen. Two changes in catecholamines occurred. An increase in epinephrine occurred in both the control and niacin groups, probably due to the stress of remaining supine in the PET gantry for 90 min of positioning, transmission imaging and dynamic imaging. An increase in norepinephrine levels occurred only with niacin treatment, suggestive of an autonomic response to the cutaneous vasodilatation induced by niacin. Infusion of dopamine in dogs has been shown to increase circulating fatty acids with accelerated lipolysis, leading to a decrease in exogenous glucose utilization in myocardium (20). Thus, it is important to note that the observed increase in glucose uptake with niacin treatment occurred in spite of elevated plasma norepinephrine levels.

Although insulin is known to have a profound impact upon myocardial glucose uptake (21), the current results highlight the independent impact of circulating fatty acid levels upon myocardial glucose utilization. The glucose/fatty acid cycle, first proposed by Randle et al. (22), implies an inverse relation between glucose and fatty acid

metabolism. The primary mechanisms of the glucose/fatty acid cycle are the regulation of the pyruvate dehydrogenase complex (PDH) by acetyl-CoA levels and of phosphofructokinase (PFK) by citrate. The inhibition of PDH and PFK leads indirectly to negative feedback inhibition of hexokinase. It is the inhibition of hexokinase that accounts for the decrease in myocardial uptake of exogenous glucose. The effect of increases in circulating fatty acid levels upon exogenous glucose uptake by the heart has been demonstrated in humans with arteriovenous sampling (1) and PET ^{18}F -FDG (5). Nuutila et al. (5) further demonstrated that increases in circulating fatty acids may suppress exogenous glucose utilization even in hyperinsulinemic conditions.

In the current study, the effect of decreased circulating fatty acid levels was observed. Noninvasive determination of exogenous myocardial glucose utilization was performed with ^{18}F -FDG and PET, demonstrating an increase in glucose uptake after acute niacin therapy. Correlation of myocardial glucose utilization rates in the control and niacin studies with free fatty acid, insulin and glucose levels demonstrated a significant correlation with fatty acid levels only. Our results are in agreement with the previous study of Lasser et al. (23) in which the reduced levels of circulating fatty acid induced by niacin infusion corresponded to increases in myocardial glucose uptake as determined by arteriovenous differences. Our results also agree with the recent study of Knuuti et al., in which relative myocardial ^{18}F -FDG uptake and absolute glucose utilization were determined both during hyperinsulinemic euglycemic clamp and after a dose of acipimox, a potent nicotinic acid derivative (24). In that study, there was no difference either in ^{18}F -FDG uptake or in glucose utilization between the two experimental conditions. Given the known stimulation of ^{18}F -FDG uptake with the euglycemic hyperinsulinemic clamp (10), the similarity of the glucose uptake rates for the two treatment approaches is consistent with augmentation of glucose uptake with suppression of fatty acid levels. These studies clearly demonstrate an increased utilization of circulating glucose in vivo with a reduction in plasma fatty acid levels, without facilitation of glucose transport by augmentation of insulin levels.

The increase in exogenous glucose utilization by the heart occurred in the setting of no change in cardiac workload. It is unclear from this study which myocardial substrate had a decline in utilization rate since we did not measure the metabolic rate of the other substrates. We presume with the change in plasma FFA levels compared to the control studies, the lack of change in plasma glucose and insulin levels, and the lack of a direct effect of niacin upon the heart, that there was a decrease in exogenous FFA utilization. Without directly measuring the metabolic rate of exogenous FFA, it is also plausible that there may have been both a decrease in the myocardial metabolic rates of cardiac endogenous lipid stores and exogenous FFA.

The current study also suggests a regional variation in

the susceptibility of myocardial glucose uptake to circulating fatty acid levels. A previous study by Hicks et al. found a relative decrease in septal ^{18}F -FDG uptake despite rigorous control of the circulating carbohydrate/insulin milieu with the clamp technique, implying regional differences in metabolic substrate preference (9). In the current study, a similar significant difference between uptake in the septum and lateral wall was seen in the control group. With the administration of niacin, however, the regional variation in cardiac ^{18}F -FDG uptake, was abolished, with similar rates of glucose utilization rates in the septum, anterior wall and lateral wall after niacin. This regional change in ^{18}F -FDG uptake after niacin treatment, compared to the control state, may be related to a difference in metabolic preferences among the different myocardial regions, with fatty acids more avidly utilized in the septum than in the other regions. Moreover, the suppression of this preference with a decrease in fatty acid levels suggests that the regional difference is related to the inhibitory effect of acetyl-CoA upon PDH.

For this study, we used a programmed infusion method for administration of ^{18}F -FDG. This method offers the technical and analytic benefits of a constant plasma ^{18}F -FDG level. The technical complexity of the study, particularly of blood sampling, is significantly reduced. Accurate estimates of blood concentrations, whether by conventional blood sampling or from image data, are attained more easily when those concentrations are varying only slowly. Furthermore, the method provides stronger assurance that the assumptions required by the graphical method are met. Exchangeable tissue radioactivity approaches true rather than transient equilibrium with the plasma radioactivity. Moreover, the requirement that the time dependence of plasma radioactivity be slower than any exchangeable process in tissue is assured. The lack of blood-pool activity clearance, however, does lead to a decrease in the ratio of final myocardial-to-blood pool radioactivity. Use of a parametric image mapping the graphical slopes computed in each image pixel resolves this problem, retaining the image quality seen with a standard bolus of ^{18}F -FDG.

CONCLUSION

Although image quality of the parametric slope image with niacin is similar to that from the more widely used glucola protocol with bolus administration of ^{18}F -FDG, absolute glucose rates were lower with niacin-induced alteration in myocardial glucose uptake, suggesting that the stimulus for myocardial glucose uptake with 1 g of niacin is less than that with 50 g of glucose. Future studies of a direct comparison of niacin and glucola as well as the effect of higher doses of niacin on absolute glucose utilization are planned.

Further studies are also needed to determine the rate of uptake with niacin treatment in diabetic patients and to compare the effect of niacin and glucola utilizing the same

^{18}F -FDG administration protocol prior to adoption of acute niacin treatment for routine clinical studies of myocardial glucose uptake.

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(continued from page 11A)

FIRST IMPRESSIONS

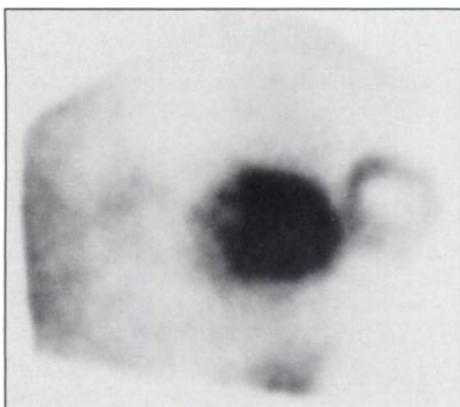


FIGURE 1. Anterior stress thallium image.

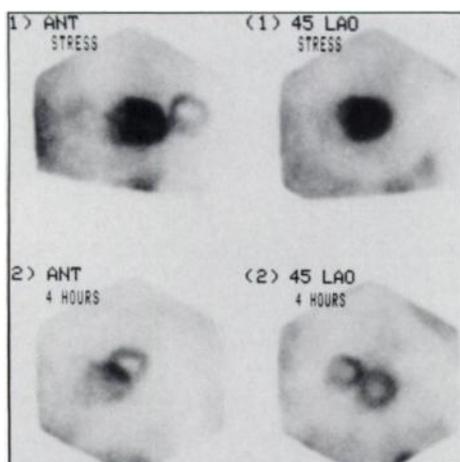


FIGURE 2. Anterior and 45° LAO views. Top row: Stress images. Bottom row: Four-hour redistribution images.

PURPOSE

A 70-yr-old woman with an ulcerated left breast tumor had metastases to bone. Stress and redistribution thallium scintigraphy was performed to rule out coronary artery disease. The anterior view stress-thallium image (Fig. 1) shows uniform activity in the left ventricle. A large area of increased thallium activity with central photopenia is seen adjacent to the left ventricle. The anterior and 45° LAO views at stress and 4 hr (Fig. 2) show a change in the lesion's position in accordance with the left breast location. These images are unique because thallium activity in the lesion resembles the left ventricle. The images might also be interpreted as a thallium-avid lesion in the chest wall or in the left lung, illustrating that examination of the patient is an essential component of nuclear medicine practice.

TRACER

Thallium-201-chloride (3.5 mCi)

ROUTE OF ADMINISTRATION

Intravenous injection at peak exercise

TIME AFTER INJECTION

Immediately after injection and 4 hr later

INSTRUMENTATION

Gamma camera

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