Myocardial Viability Assessed by Positron Emission Tomography in Infants and Children After the Arterial Switch Operation and Suspected Infarction

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OBJECTIVES

The aim of the study was to assess regional glucose metabolism and contractile function by gated positron emission tomography using fluoro-18-deoxyglucose (FDG-PET) in pediatric patients after the arterial switch operation and suspected myocardial infarction.

BACKGROUND

Morbidity and mortality after the arterial switch operation for transposition of the great arteries are often related to impaired coronary function. Justification of high-risk revascularization procedure in infancy requires thorough evaluation of myocardial viability. Although PET is state-of-the-art for evaluation of myocardial viability in adults there are no reports on its impact and feasibility in infants and children.

METHODS

We applied electrocardiogram-triggered FDG-PET for assessment of metabolic and functional status of the myocardium in seven infants and seven children. Glucose metabolism, wall motion and wall thickening were evaluated visually and quantitatively on the basis of parametric 3-D images. Additionally, single-photon emission computed tomography perfusion scan was performed in six children.

RESULTS

In two of seven infants, FDG-PET demonstrated viable myocardium in akinetic or hypokinetic regions corresponding to a coronary artery stenosis or occlusion. Therefore, indication for revascularization was derived from this finding. In six of the seven children, impaired glucose uptake reflecting myocardial scarring was present. Two patients had pathological findings on coronary angiography and signs of ischemia but were not suitable for revascularization.

CONCLUSIONS

Myocardial viability and contractile function can be assessed simultaneously by gated FDG-PET even in infant hearts. This method contributes pertinent information to guide further therapy after the arterial switch operation and suspected myocardial infarction. (J Am Coll Cardiol 2000;36:1676–83) © 2000 by the American College of Cardiology

Arterial switch operation is now the treatment of choice in patients with simple transposition of the great arteries (TGA) (1–4). The coronary arteries are transferred to the neo-aorta. Mortality and long-term outcome after arterial switch operation depend mainly on the adequate perfusion of the transferred coronary arteries (5–10). From the 5% to 10% early deaths within 24 h after surgery, 55% are related to myocardial ischemia or infarction resulting from stenosis, compression, kinking or occlusion of the transferred coronary arteries (11–13). It was reported that “late deaths” several years after operation are related (in 1% to 2%) to coronary stenosis or occlusion (11–12). Fibrocellular thickening of the intima at the proximal region of transferred coronary arteries resulting in stenosis or occlusion and sudden cardiac death due to myocardial infarction (MI) was reported by investigators in 6 (10%) of 59 patients within 10 months’ postoperation (14). Because revascularization by mammary bypass grafts and percutaneous transluminal coronary angioplasty (PTCA) are now therapeutic options in infancy, information on viability is mandatory (15–19).

Positron emission tomography using fluoro-18-deoxyglucose (FDG-PET) and single-photon emission computed tomography (SPECT) with thallium-201 or technetium-99m (Tc-99m) have been successfully used for detection of myocardial viability and ischemia in adults (20,21). Moreover, gated PET has the potential of simultaneous investigation of regional metabolism, wall motion and wall thickening. However, neither standard nor electrocardiographic (ECG) gated FDG-PET and SPECT have been established thus far in infants and children.

Therefore, the aim of this study was to demonstrate the feasibility and the clinical impact of gated FDG-PET in infants and children after the arterial switch operation and suspected or confirmed MI. Additionally, technetium-99m sestamibi SPECT was performed prior to FDG-PET. For technical reasons this was possible only in children above 10 kg of body weight. The study was approved by our ethics committee.

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PATIENTS AND METHODS

Among a group of 94 pediatric patients who underwent arterial switch operation since 1982, fourteen consecutive infants and children were evaluated. None of the patients who were intended to be investigated met exclusion criteria. We included seven infants age 1 to 6.5 months (median: 3 months), weight: 3.8 kg to 7.3 kg (median: 4.3 kg), and seven children age 6.5 to 16.6 years (median: 14 years), weight: 21.3 kg to 82 kg (median: 45 kg) who had undergone anatomic correction of simple TGA. Details of the patients are shown in Table 1. The anatomy of the coronary arteries was described according to the classification proposed by Yacoub (22). Anatomical status as defined by conventional left coronary angiography showed a usual pattern in four infants (type A). In three infants, coronary variations were found with the left circumflex artery originating from the right coronary artery (type D) in one infant, a single ostium in another patient (type B), and an additional ostium of the circumflex artery arising from the left-facing sinus of the third infant. In three of the seven children a type D, and in four a type A, coronary artery anatomy was present.

All infants presented with acute signs of MI or ischemia peri- or postoperatively within two months. The children presented with regional wall motion abnormalities on echocardiography during routine follow-up 6 to 17 years after the arterial switch operation. Informed consent for each patient was obtained from the parents for the total procedure.

Clinical status. All infants had clinical signs of myocardial ischemia or infarction, with ECG changes and/or elevated enzymes (troponin I >0.5 U/liter, creatine kinase, MB fraction [CK-MB] >8% of total CK) and/or wall motion abnormality on echocardiography as given in detail in Table 1. A myocardial injury was diagnosed peri-operatively in three infants and within two months postoperatively in four infants. Only one of the seven children and only two of the seven infants developed symptoms. Two of these (patients 5 and 14) presented with signs of angina. One infant (Patient 2) had signs of congestive heart failure and died at the age of six months. None of the older children had intervening events such as MI between the patients’ arterial switch procedure and the PET imaging studies.

Operation. Six infants (Patients 1, 2, 4–7) and two of the children (Patients 8 and 12) had an arterial switch operation as a primary operation in the neonatal period within the first two weeks of life. One infant (Patient 3) with Taussig-Bing anomaly and coarctation of the aorta underwent a two-stage repair at the age of 4 and 13 weeks with additional resection of the coarctation. The children were operated between the age of 10 days and 2.2 years with a one- (Patients 8 and 12) or two (Patients 9–11, 13, 14) -stage repair. In one patient (Patient 14) a resection of a coarctation of the aorta was performed before corrective surgery.

Echocardiography. For wall-motion analysis, standard transthoracic parasternal short-axis and apical four-chamber views were used in all patients. Additionally, in all infants we used the subcostal four-chamber and short-axis views. The echocardiographic investigation was performed at the same date as the PET investigation. Commercially available equipment was used for the echocardiograms.

Coronary angiography. Selective injection into the coronary arteries was performed manually. In addition, a coronary root injection was done with particular attention to the coronary ostia and aortic valve. Biplane angiograms were performed in lateral and in right and left anterior oblique views to evaluate the coronary artery arising from the left and right side of the neoaoartic root.

Positron emission tomography. Patient preparation. To maximize F-18-FDG uptake into viable myocardium, FDG was administered following a combined oral and intravenous (IV) glucose loading for infants and children up to 10 years of age. Two hours before the PET investigation the oral glucose consumption (2 g/kg) was started. This was then followed by continuous IV glucose (10%) administration (4 ml/kg/h) 30 min before PET. Children above the age of 10 years received a continuous IV glucose (10%) solution (1.3 ml/kg/h) with insulin (1 U/50 ml) and KCl (1 mmol/50 ml). Blood glucose levels were obtained before and after the PET investigation. The radioactive tracer F-18-FDG (5 to 7 MBq/kg) was injected 30 min before the PET investigation intravenously. Sedation was achieved by oral application of 40 mg/kg to 60 mg/kg chloral hydrate or IV administration of 0.1 mg/kg to 0.2 mg/kg midazolam.

Data acquisition. The ECG-gated data acquisition (7 to 12 gates per heart cycle) was performed with a conventional CTI/Siemens PET-scanner (ECAT EXACT 47) 30 min after injection of FDG. An emission scan of 20 min was followed by a 10-min transmission measurement for attenuation correction.

Data analysis. Transaxial slices of $128 \times 128$ pixels were reconstructed by iterative reconstruction and re-angulated for display according to the left ventricular (LV) long axis. For further evaluation, end-diastolic, end-systolic, and summarized nongated sets of slices were considered. Analysis of

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**Abbreviations and Acronyms**

ECG = electrocardiogram, electrocardiographic
FDG-PET = fluor-18-deoxyglucose positron emission tomography
IV = intravenous
LAD = left anterior descending artery
LCA = left coronary artery
LV = left ventricular, left ventricle
MI = myocardial infarction
PTCA = percutaneous transluminal coronary angioplasty
SPECT = single-photon emission computed tomography
TGA = transposition of the great arteries
Table 1. Comparison of Clinical and Radionuclide Data of Pediatric Patients After Arterial Switch Operation

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Diagnosis</th>
<th>Age at Surgery</th>
<th>Coronary Artery Type*</th>
<th>Coronary Angiography</th>
<th>Echocardiography (hypo-/akinesis)</th>
<th>Age at PET</th>
<th>Weight at PET (kg)</th>
<th>PET Metabolism</th>
<th>PET Wall Thickening</th>
<th>PET Motility</th>
<th>SPECT Type of Defect</th>
</tr>
</thead>
<tbody>
<tr>
<td>Infants</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>TGA</td>
<td>10 d</td>
<td>D</td>
<td>LAD occlusion</td>
<td>LV ant, sept, apex</td>
<td>3 m</td>
<td>4.3</td>
<td>Nontransmural: apex</td>
<td>Impaired: apex</td>
<td>Akinesis: LV ant, sept, apex</td>
<td>n.a.</td>
</tr>
<tr>
<td>2</td>
<td>TGA, VSD</td>
<td>15 d</td>
<td>B</td>
<td>LCA, RCA kinking</td>
<td>LV ant, RV ant, sept</td>
<td>4 m</td>
<td>5.9</td>
<td>Transmural: apex, anterobasal</td>
<td>Negative: apex, ant, apex</td>
<td>Akinesis: ant, apex</td>
<td>n.a.</td>
</tr>
<tr>
<td>3</td>
<td>Taussig-Bing CoA</td>
<td>2.5 m</td>
<td>3 Orifices</td>
<td>Normal</td>
<td>Septal</td>
<td>3.5</td>
<td>4.8</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
</tr>
<tr>
<td>4</td>
<td>TGA</td>
<td>14 d</td>
<td>A</td>
<td>Refused</td>
<td>Transient: ant</td>
<td>1 m</td>
<td>3.8</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
<td>n.a.</td>
</tr>
<tr>
<td>5</td>
<td>TGA</td>
<td>17 d</td>
<td>A</td>
<td>LCA: prox. stenosis</td>
<td>LV ant, sept, Apex</td>
<td>6.5 m</td>
<td>7.3</td>
<td>Normal</td>
<td>Normal</td>
<td>Impaired: ant, sept, apex</td>
<td>n.a.</td>
</tr>
<tr>
<td>6</td>
<td>TGA</td>
<td>12 d</td>
<td>A</td>
<td>Normal</td>
<td>Transient: ant</td>
<td>29 d</td>
<td>3.8</td>
<td>Normal</td>
<td>Normal</td>
<td>Impaired: ant, lat</td>
<td>n.a.</td>
</tr>
<tr>
<td>7</td>
<td>TGA</td>
<td>12 d</td>
<td>A</td>
<td>Normal</td>
<td>Ant, sept</td>
<td>3 m</td>
<td>6.2</td>
<td>Nontransmural: ant, lat</td>
<td>Normal</td>
<td>Impaired: ant, lat</td>
<td>n.a.</td>
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<tr>
<td>Children</td>
<td></td>
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<td></td>
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<td></td>
</tr>
<tr>
<td>8</td>
<td>TGA, CoA</td>
<td>38 d</td>
<td>A</td>
<td>Normal</td>
<td>sept</td>
<td>14.8 y</td>
<td>82</td>
<td>Nontransmural: ant, sept</td>
<td>Impaired: sept</td>
<td>Impaired: sept</td>
<td>Irreversible: ant, sept</td>
</tr>
<tr>
<td>9</td>
<td>TGA</td>
<td>11 m</td>
<td>D</td>
<td>Not done, (LV dilation)</td>
<td>sept, (LV dilation)</td>
<td>13.1 y</td>
<td>44</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
</tr>
<tr>
<td>11</td>
<td>TGA</td>
<td>1.5 y</td>
<td>D</td>
<td>Normal</td>
<td>LV: sept, apex (LV dilation)</td>
<td>16.3 y</td>
<td>80</td>
<td>Nontransmural: ant, transmural: apex, ant, sept</td>
<td>Negative: apex, ant, sept</td>
<td>Impaired: apex, ant, sept</td>
<td>n.a.</td>
</tr>
<tr>
<td>12</td>
<td>TGA</td>
<td>10 d</td>
<td>A</td>
<td>Normal</td>
<td>sept</td>
<td>6.5 y</td>
<td>21.3</td>
<td>Nontransmural: sept, ant, lat</td>
<td>Normal</td>
<td>Normal</td>
<td>Refused</td>
</tr>
<tr>
<td>13</td>
<td>TGA, VSD</td>
<td>2 y</td>
<td>A</td>
<td>Refused</td>
<td>sept</td>
<td>16.6 y</td>
<td>73</td>
<td>Transmural: apex, ant, sept</td>
<td>Transmural: apex, ant, sept</td>
<td>Impaired: apex, ant, sept</td>
<td>Irreversible: apex, ant, sept, reversible: apex, lat, apex</td>
</tr>
<tr>
<td>14</td>
<td>Taussig-Bing</td>
<td>2.2 y</td>
<td>D</td>
<td>Hypoplastic LAD</td>
<td>Apex, ant, lat</td>
<td>12.2 y</td>
<td>54.3</td>
<td>Transmural: apex</td>
<td>Impaired: apex</td>
<td>Impaired: apex, lat</td>
<td>Irreversible: apex, reversible: sept, ant, apex</td>
</tr>
</tbody>
</table>

*Coronary artery classification as described by Yacoub (22).

ant, anterior; CoA, coarctation of the aorta; d, days; inf, inferior; LAD, left anterior descending coronary artery; lat, lateral; LV, left ventricle; m, months; n.a., not available; RCA, right coronary artery; RV, right ventricle; sept, septal; TGA, transposition of the great arteries; VSD, ventricular septal defect; y, years.
regional wall motion was performed by a three-dimensional model of the LV surface (23). Therefore, the basal 3/4 of the LV were modeled as a cylinder, and the apical 1/4 was modeled as a hemisphere. For the cylindrical part of the model, the surface points were determined by radial searching started from the ventricular long axis, and searching started from the center toward the radial direction for the hemispherical part of the model (Fig. 1).

Amplitude of regional wall motion was estimated from pixels with maximal FDG uptake at each searching direction. The distance between these points at end-diastole and end-systole was considered as the amplitude of the ventricular contraction and displayed color-coded onto the end-diastolic ventricular surface (Figs. 1 and 2).

For the analysis of ventricular thickening, the same surface points, end-diastolic and end-systolic, were consid-

Figure 1. Systolic (yellow) and diastolic (red) maximal indicated intensity was detected and defined as wall center points for wall-motion analysis (vla = vertical long axis; sa = short axis).

Figure 2. Color-coded wall-motion analysis of the LV (Patient 1) was derived from gated PET (red = max. amplitude). The anteroseptal and inferoseptal view showed akinesia of the entire anteroseptal and apical region.
Parametric imaging of myocardial viability and function was achieved by dedicated software developed in Hanover (Medical School, PET center) (23).

Myocardial perfusion scintigraphy. Stress myocardial perfusion scintigraphy was performed corresponding to a two-day protocol after injection of 5 to 7 MBq/kg Tc-99m-MIBI each, following a bicycle stress test. The SPECT data acquisition was performed using a single-headed gamma camera (Orbiter, Siemens). Thirty-two images were acquired every 40 s over 180° in a 64×64 matrix with an appropriate acquisition zoom. For reconstruction a Butterworth filter (order 5, cutoff 0.4) was used. Re-angulated short- and long-axis views were displayed in the same manner as described for FDG-PET. Evaluation was performed visually using a dedicated color-table for heart studies.

RESULTS

Echocardiography and ECG. The echocardiographic image quality was sufficient in all patients to evaluate regional wall motion in all myocardial territories. All infants and children presented with regional wall-motion abnormalities as described in Table 1. Left ventricular shortening fraction was below the normal range in two infants (Patients 1 and 2) and three children (Patients 9–11). Additional findings were mild-to-medium supravalvular pulmonary artery stenosis in five patients (Patients 1, 2, 5, 11, 12), right ventricular outflow tract obstruction in two (Patients 3 and 10), second-degree aortic regurgitation in one (Patient 13) and severe stenosis of aortic homograft in position of the pulmonary valve in another child (Patient 14).

On the resting ECG, six infants (Patients 1, 2, 4–7) presented with ST-T changes. An abnormal Q wave was found in two children (Patients 13 and 14). All patients had normal sinus rhythm.

Coronary angiography. Accurate imaging of the anatomical pattern of the coronary arteries was possible in all patients by selective coronary angiography and aortic root injection before primary surgery and in 11 patients on follow-up. A coronary artery stenosis in three patients (Patients 2, 5, 10), an occlusion in one patient (Patient 1) and a hypoplastic left anterior descending artery (LAD) were diagnosed in another child (Patient 14). The clinical and functional status in relation to the coronary angiography is listed in Table 1.

Figure 3. The corresponding picture of glucose metabolism (Patient 1) shows normal-to-increased glucose metabolism in the akinetic anterosepal region (red = max. intensity). In the apical region the reduced intensity indicates a nontransmural infarction.
In one child without symptoms (Patient 9) we did not perform a heart catheterization, because there was no evidence for stress-induced ischemia or myocardial scarring detected by SPECT and PET. The parents of one infant (Patient 4) and one child (Patient 13) refused heart catheterization.

**PET.** The mean age at the time of PET investigation was 3.1 ± 1.7 months for the infants (Patients 1–7) and 13.7 ± 3.3 years for the children (Patients 7–14).

**REGIONAL MYOCARDIAL METABOLISM.** All PET investigations demonstrated good image quality. Both regional metabolism and regional wall motion/wall thickening could be completely evaluated. In one (Patient 2) of seven infants and in four of seven children (Patients 10, 11, 13, 14) there was no glucose metabolism in the echocardiographically detected akinetic/hypokinetic areas indicating scar tissue. In one infant (Patient 1) and two children (Patients 8 and 12) there was a partially decreased glucose uptake consistent with a nontransmural infarction in the region of interest. In six infants (Patients 1, 3–7) and in one child (Patient 9) we found normal glucose uptake in akinetic or hypokinetic areas. The relation of regional glucose metabolism, coronary angiography and wall motion is listed in Table 1.

**MYOCARDIAL WALL MOTION AND WALL THICKENING.** The results of PET wall-motion analysis were in concordance with the echocardiographically detected wall-motion abnormalities of the main areas of focus in all patients (Table 1). By contrast to the findings on echocardiography and PET wall-motion analysis, we found preserved septal wall thickening in two infants (Patients 1 and 5). A detailed comparison of regional glucose metabolism and wall motion was possible only by using the PET images.

**Myocardial perfusion scintigraphy.** Both stress and rest myocardial perfusion were studied in six children. However, myocardial perfusion imaging with SPECT was not performed in infants owing to the small size of their hearts and the expected poor image quality. In one child (Patient 12) the parents refused the SPECT investigation. The resting study demonstrated perfusion defects corresponding to the echocardiographically detected wall-motion abnormalities in five children (Patients 8, 10, 11, 13, 14). Normal metabolism of these areas was preserved only in one child (Patient 9). No additional perfusion defects under stress were observed in this patient. Three children (Patients 10, 11, 14) presented with additional small perfusion defects during stress tests, indicating ischemia. Localization of the stress-induced perfusion defect is shown in Table 1.

**DISCUSSION**

This study reports the first experiences with simultaneous evaluation of myocardial glucose metabolism and contractile function by FDG-PET in pediatric patients. An MI was suspected in seven infants and seven children after arterial switch operation for TGA. A stenosis or occlusion of the coronary arteries was detected by coronary angiography in three infants and one child. Therefore, information on regional myocardial viability was mandatory to streamline subsequent therapeutic procedures, especially in infants, considering the higher associated procedural risk.

To obtain high spatial and temporal resolution in very small hearts we used ECG-gated data acquisition. This technique was described and validated for adults in past studies (24,25). Additionally, data acquired by state-of-the-art PET scanners permit analysis of ECG-gated images for regional myocardial contractile function (e.g., wall motion and wall thickening [26]). In small infants and children, geometric resolution may be impossible due to the technical limits of the current imaging device. However, in our experience, FDG-PET can be performed even in infants with a body weight of 3.8 kg.

**Findings.**

**INFANTS.** In our series we found viable myocardium in two infants with akinetic regions on echocardiography. In one infant with severely impaired LV function and LAD occlusion, the glucose metabolism in the akinetic anterior wall and the septum was normal to increased (Fig. 1). Wall-motion analysis of the gated PET images showed no motility in the anterior and septal regions (Fig. 2), but contrary to echocardiography, wall thickening in the septal region was still present. The fact that in this infant there was preserved wall thickening in the septal area demonstrated by PET is somewhat surprising. Because of the impaired LV function, the septal area was obviously not able to contribute to systolic function. The color-coded three-dimensional analysis of the glucose uptake during the complete heart cycle made it possible to visualize changes of wall thickening indicated by a higher count of recovery per voxel.

The second infant presented with a proximal stenosis (>90%) of the left coronary artery (LCA). Considering the fact that there was decreased coronary flow as detected by coronary angiography but normal-to-increased glucose uptake in akinetic or hypokinetic areas, we determined this was hibernating myocardium (20,21).

Another infant showed signs of severe anterior MI in the early postoperative period due to kinking of the LCA and right coronary artery. In the areas with impaired function, FDG-PET showed no glucose uptake, motility or wall thickening. Therefore, this patient was not suitable for any revascularization procedure.

In four infants, PET demonstrated basically normal glucose uptake. None of these patients evidenced any coronary stenosis or occlusion (Table 1). Wall-motion akinesis/dyskinesis may suggest temporary perioperative impaired coronary flow, despite open coronary arteries. The segmental LV function in each of these patients recovered to normal at follow-up, detected by echocardiography. Therefore, these findings support the notion of myocardial stunning as the etiology for the regional dysfunction (27,28).

**CHILDREN.** In six of the seven children we found small areas of decreased FDG uptake corresponding to the results
of wall-motion analysis by PET and echocardiography. In five of them SPECT showed an irreversible defect in the same segments, and three of them had additional reversible defects in other segments (Table 1). In one instance the parents refused SPECT. Only one child had normal glucose uptake in all myocardial areas. Echocardiography showed borderline global LV function and flat septal movement. Coronary angiography demonstrated a proximal LCA stenosis in one and a hypoplastic LAD in another child. The latter was the only child with angiina-like symptoms. The fact that three children had no pathological coronary angiograms but a small defect detected by PET and SPECT is consistent with previously reported results (29,30).

**Treatment. INFANTS.** These findings of impaired coronary function in the three infants gave rise to the question of the ideal treatment. The proof of myocardial viability justifies myocardial reperfusion (28,31). However, therapeutic options for such an infant are limited. Only case reports and studies with small numbers of infants who had undergone PTCA or bypass grafting are available (15–19,32). Pediatric coronary artery bypass graft has been done mostly for ischemic complications of Kawasaki disease (17). Internal mammary artery bypass was considered the treatment of choice for our patient with LAD occlusion, whereas PTCA was the treatment of choice for the patient with the LCA stenosis. Follow-up echocardiography demonstrated a full recovery of the LV function in both infants.

**CHILDREN.** In the group of seven children we found no indication for catheter-based intervention or coronary surgery. Two children with pathological findings in coronary angiography had evidence of small ischemic areas detected by SPECT and PET. One child with a proximal LAD stenosis (>80%) had no symptoms. The other child had a hypoplastic LAD, which was considered not to be suitable for any revascularization. The patient was treated medically.

**Clinical impact.** Based on our results, FDG-PET is helpful for detecting irreversible myocardial injury in infants and children after surgical correction of TGA and other operations with coronary artery transfer (e.g., Ross operation or coronary artery transfer for anomalous origin of the LCA). Gated acquisition and the simultaneous analyses of regional wall motion/wall thickening and metabolism are especially helpful to localize jeopardized or irreversibly injured myocardium, including estimation of the extent of damage. This technique does not prolong acquisition time significantly. The co-registration of images of metabolism and regional kinesis compensates for possible discrepancies between echocardiographic and scintigraphic results. This is because of differences in the localization of myocardial territories and assessment of wall thickening, especially of the septal area. Limited acquisition resolution makes assessment of regional wall thickening, using the partial volume effect, possible. Compared with echocardiography, PET wall-motion analysis also has the advantage of providing precise information about the apical region.

**Study limitations.** The study was limited by the lack of systematic investigation of myocardial perfusion reserve. This is possible only in children with a body weight of more than 10 kg using SPECT. It should be taken into account that, owing to the finite spatial resolution of SPECT imaging, detection of small perfusion defects is limited by partial volume effects. Therefore, PET with perfusion markers as N-13-NH3 or O-15 H2O would have been preferred. However, analysis of wall motion/thickening in gated FDG-PET can partly compensate for the lack of perfusion studies because regional wall thickening correlates with the degree of hypoperfusion (25,26,33). Identification of viable myocardium using gated FDG-PET as a solitary indicator was reported previously (33,34). For pediatric patients in particular, further prospective studies with healthy control groups are required before FDG-PET without a perfusion marker can be recommended for routine clinical use.

**Conclusions.** These results indicate that myocardial viability and contractile function can be assessed simultaneously by ECG-gated FDG-PET even in infant hearts. This method contributes pertinent information to guide further therapy in pediatric patients after the arterial switch operation and suspected MI.

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**REFERENCES**