# Cerebral Perfusion During Transient Global Amnesia: Findings with HMPAO SPECT

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The aim of this study was to investigate the pattern of regional cerebral blood flow changes associated with transient global amnesia (TGA). **Methods:** HMPAO SPECT was performed in six consecutive patients during the acute phase of TGA. A follow-up SPECT was performed 3–20 wk later in four of the six patients. Semiquantitative analysis of 14 regions of interest, including the basal ganglia and the basal section of the temporal lobes, was performed by comparing patient data with control data obtained from a matched group of healthy subjects. **Results:** During TGA, unilateral or bilateral hypoperfusion of the temporo-basal region was observed in four patients. Variable hypoperfusion of further cortical areas was observed in five patients. Two patients who exhibited the most marked cortical hypoperfusion also showed striatal and thalamic hypoperfusion. These changes were normalized in the control studies obtained in four patients. **Conclusion:** It remains unclear whether hypoperfusion during TGA represents a primary feature or a sequel of regional brain hypometabolism. Because hypoperfusion is not confined to the temporo-basal region or to the territory of the posterior cerebral artery, it is suggested that the origin of TGA-related changes lies at the level of subcortical structures that project diffusely to the cerebral cortex.

**Key Words:** transient global amnesia; cerebral blood flow; HMPAO SPECT

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Transient global amnesia (TGA), a benign condition of unknown etiology, is characterized by sudden onset of anterograde amnesia and variable retrograde amnesia. Clinical resolution occurs within 1–24 hr; the average duration of the attacks is 6–8 hr. The minimum annual incidence of TGA is about three per 100,000. The peak incidence occurs between the ages of 50 and 60 yr. Recurrence is observed in approximately 10% of those affected. In about half of the cases, TGA is precipitated by an emotionally or physically stressful event. An epidemiological relationship with migraine has been established, but the pathophysiological link remains unclear (*1,2*). In vivo imaging studies of cerebral perfusion performed during episodes of TGA may contribute to the understanding of the disorder. However, due to the relative rarity and brevity of TGA, few cases have been documented to date.

We studied cerebral blood flow (CBF) changes during attacks of TGA with HMPAO SPECT in six patients. A delayed control SPECT was performed in four patients. A brief review of previous radionuclide imaging findings is given, and the location, time course and pathophysiological implications of TGA-related CBF abnormalities are discussed.

## MATERIALS AND METHODS

### Patients

Six consecutive patients who presented at our clinic during the acute phase of a TGA episode were studied. In all cases, the clinical picture was typical of TGA and fit the accepted diagnostic criteria (*3,4*). None of the patients had a history of epilepsy or cerebral ischemic events. A history of migraine was present in three patients (Patients 2, 5 and 6). During TGA, neurological examination disclosed no physical abnormalities or cognitive deficits other than amnesia. The beginning of the resolution of amnesia was assessed by repeatedly probing patients' orientation and memory for the course of the examination. At the time of the SPECT study, amnesia was fully expressed in Patients 1–3, whereas some vague memories for preceding events were present in Patients 4–6. In all cases, amnesia had resolved by the following day. In Patients 1–4, a control SPECT was performed 3–20 wk after the attack. Clinical data are summarized in Table 1, and information on the timing of SPECT examinations is given in Table 2.

### Control Group

Five men and 10 women (mean age 65 yr; range 50 yr–75 yr) who were free of current or previous neuropsychiatric disease were selected from our control database.

### SPECT Imaging Protocol

All patients were examined in an identical setting. SPECT studies were done on a dual-headed rotating gamma camera system (Dyna Digital, Picker International, Cleveland, OH), equipped with low-energy, all-purpose collimators (FWHM 9.2 mm). Image acquisition was initiated 20 min after injection of 550 MBq $^{99m}$Tc-HMPAO (Ceretec, Amersham International, Buckinghamshire, UK) into an anterior cubital vein. Patients were examined in a supine position, with their heads fixed in a head holder on an extension board so that the cantho-meatal line was perpendicular to the longitudinal axis of the gamma camera heads. The room was darkened 15 min before injection. The imaging protocol acquired 60 frames at 20 sec per frame with a 360° rotation of the camera, using a matrix size of 64 × 64 pixels. A filtered backprojection technique was applied for image reconstruction, using a Butterworth filter of sixth order and a cutoff frequency of 0.5 $cm^{-1}$. Images were analyzed without attenuation correction. Two-pixel-wide (14.6-mm) transaxial and coronal slices were obtained.

### Data Analysis

Semiquantitative evaluation was done in the transaxial plane using anatomically defined regions of interest (ROIs), as described elsewhere (*5,6*). In brief, six ROIs in consecutive slices were assigned to each frontal lobe, five ROIs were assigned to each of the temporal and parietal lobes, and four ROIs were assigned to each occipital lobe. In addition, one thalamic ROI and one striatal ROI were assigned to each hemisphere. Two ROIs served as cerebellar references. Figure 1 shows the delineation of the 46 ROIs.

Average counts per pixel were calculated for each ROI. To improve the correlation between $^{99m}$Tc-HMPAO distribution and CBF, as measured by $C^{15}O_2$ PET, a linearization correction was applied to the $^{99m}$Tc-HMPAO images (*7–9*). The corrected data were normalized by the average counting rate of the cerebellum, which is usually not involved in TGA. The resulting value was designated as the perfusion index (*7*):

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**TABLE 1**
Clinical Data

| Patient no. | Age (yr) | Sex | Precipitants and circumstances of TGA | Physical complaints and behavior during TGA |
|---|---|---|---|---|
| 1 | 55 | M | Stress electrocardiography | No complaints, appears calm and cooperative, asks repetitive questions |
| 2 | 68 | F | Shopping in an overheated boutique | Complains of nausea, appears anxious and restless, asks repetitive questions |
| 3 | 70 | M | History taking (admission for hypertension, exhaustion and depression) | Heart rate and blood pressure elevated, appears anxious and perplexed, unable to retain new information |
| 4 | 57 | F | Long bicycle ride in the course of a sports event | Complains of headache, appears anxious and restless, asks repetitive questions |
| 5 | 63 | F | Onset of TGA upon awakening in the early morning | No complaints, appears calm and cooperative, asks repetitive questions |
| 6 | 48 | F | Hot shower | No complaints, appears perplexed and slightly slowed, but cooperative, unable to retain new information |

$$\text{Perfusion index} = (a \times C/C_r)/(1 + a - C/C_r),$$

where a is a constant of 1.5, C is the counts/pixel of a given ROI and $C_r$ is counts/pixel of the cerebellar reference. Patients' perfusion indices were then transformed to z-scores on the basis of controls' means and s.d. A z-score of $\geq +2$ or $\leq -2$ was applied as a criterion of abnormal regional CBF.

The visual analysis of SPECT images was done by two investigators who were aware of the diagnosis but unaware of clinical details when reading the images.

## RESULTS

Table 3 presents patients' perfusion indices. Cortical values were averaged on a lobe-by-lobe basis. The basal temporal lobe section, the thalamus and the striatum were evaluated separately.

### Cortical Perfusion During Transient Global Amnesia

All perfusion indices were reduced below $-2$ or were in the normal range. Only two scores were greater than $+0.7$ (Patient 1). Unilateral or bilateral hypoperfusion of the temporo-basal section, as indicated by scores below $-2$, was present in four patients (Patients 2–5) and approached the criterion in a fifth patient (Patient 6). Figure 2 exemplifies this finding with a coronal section through the temporal lobes of Patient 5. Unilateral hypoperfusion of the entire temporal lobe was observed in Patient 3 and approached the criterion in Patient 2. Furthermore, five patients showed unilateral or bilateral perfusion deficits in other ROIs, i.e., the parietal and occipital lobes (Patients 1, 2, 3 and 6) and the frontal lobes (Patients 2, 3 and 5). Figure 3 demonstrates parietal and occipital hypoperfusion in one of these patients (Patient 6).

### Cortical Perfusion at Follow-Up

The semiquantitative data were now unremarkable; z-scores ranged from $-1.3$ to $+1.8$. This finding conforms to the assumption of normalized perfusion. In Patients 1, 2 and 4, SPECT images were normal on visual inspection. An example is shown in Figure 4: the temporo-basal, parietal and basal ganglia hypoperfusion present during TGA is normalized at follow-up, 4 wk later (Patient 2). An exception was Patient 3, in whom persistent fronto-lateral hypoperfusion was observed, although it was less marked than that observed during TGA and was confined to the left hemisphere. This finding could be related to the coexistence of depression in this patient because imaging studies in depressed patients have shown a variety of abnormalities, including global reductions in blood flow or metabolism, as well as changes confined to frontal, temporal and subcortical regions (*10–12*). A magnetic resonance imaging performed 10 days after TGA in Patient 3 was normal.

**TABLE 2**
Timing of SPECT Studies

| Patient | HMPAO injection (hr after onset) | Initiation of TGA resolution (hr after onset) | Follow-up study |
|---|---|---|---|
| 1 | 4 | 7 | 3 wk later |
| 2 | 5 | >8 | 4 wk later |
| 3 | 5.5 | 7 | 20 wk later |
| 4 | 6 | 3 | 10 wk later |
| 5 | 8.5 | 8 | — |
| 6 | 3.5 | 3 | — |

### Acute Stage/Follow-Up Differences

Differences between acute stage and follow-up perfusion indices were separately evaluated because it may be assumed that they are less affected by interindividual variations. There was a general tendency toward cortical perfusion increases at follow-up. Decreases exceeding 0.5 s.d. were not observed. Patients 1–3 exhibited a global increase of 1 s.d. or more in all ROIs, except for the temporo-basal section of Patient 1. Temporo-basal increases in Patients 2–4 ranged from 1.7 to 4.3 s.d.

### Striatal and Thalamic Perfusion

Two patients showed clearly subnormal striatal perfusion during TGA. Thalamic perfusion was also subnormal or approaching the criterion in these patients (Patients 2 and 3). At follow-up, the corresponding perfusion indices increased throughout by 2.2–5.1 s.d. The other patients exhibited no significant striatal and thalamic hypoperfusion during TGA and no increases at follow-up. Patient 6 showed hyperperfusion during TGA.

## DISCUSSION

The present findings show that transient hypoperfusion of the temporo-basal region is a prominent feature in TGA. First, temporo-basal perfusion deficits of $\geq 2$ s.d. were observed in four patients. Patient 6 showed a borderline deficit on one side. In most instances, these deficits were more pronounced than the deficits found in any other ROI. However, one patient showed no temporo-basal hypoperfusion (Patient 1).

A second finding is that cortical hypoperfusion extended beyond the temporo-basal region, affecting the frontal, parietal and occipital lobes with similar frequency but variable distribution. Neocortical hypoperfusion was more extensive in those

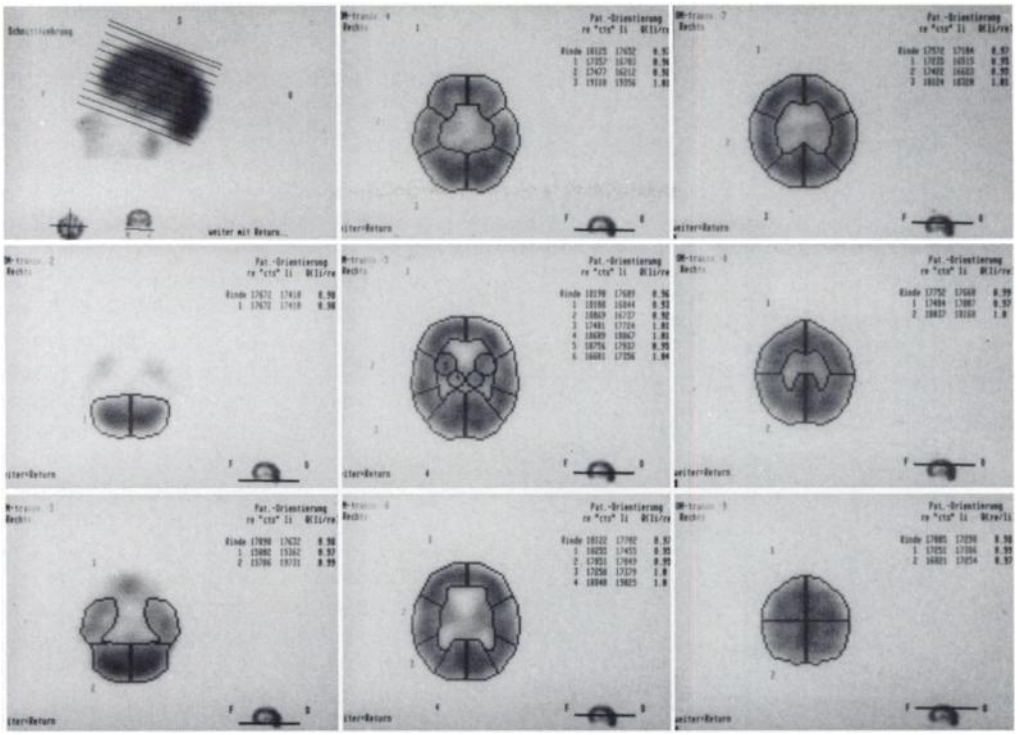

**FIGURE 1.** Delineation of the cortical, cerebellar, thalamic and striatal ROIs.

three patients whose amnesias were fully expressed during SPECT. Of the corresponding 24 perfusion scores, 16 were lower than −2 s.d. in this subgroup (Patients 1–3). At follow-up, 5 of these scores increased by ≥1 s.d., and 19 scores increased by ≥2 s.d. In contrast, only 5 of 24 scores were lower than −2 s.d. in the three patients whose amnesias had begun to remit at the time of study (Patients 4–6; Table 3).

Third, it was found that two patients whose cortical perfusion deficits during TGA were most pronounced also showed transient striatal and thalamic hypoperfusion and substantial perfusion increases at follow-up (Patients 2 and 3). In the other patients, consistent striatal and thalamic perfusion changes were not observed.

### Synopsis of Previous Radionuclide Imaging Studies

Six previous single-case reports of SPECT scanning during TGA have been published. Two patients each displayed bilateral temporal hypoperfusion (*13,14*) and patchy hypoperfusion in the territory of the posterior cerebral arteries, including the medial temporal regions (*15,16*). One patient exhibited asymmetric hypoperfusion of the medial temporal regions, the left frontal lobe and both thalami (*1*). The sixth case showed global

**TABLE 3**
Perfusion Indices

| Subject | Frontal lobe (right/left) | Temporal lobe (right/left) | Temporo-basal section (right/left) | Parietal lobe (right/left) | Occipital lobe (right/left) | Thalamus (right/left) | Striatum (right/left) |
|---|---|---|---|---|---|---|---|
| Control, mean | +1.02/+1.01 | +0.99/+0.98 | +0.89/+0.88 | +1.01/+1.01 | +1.18/+1.17 | +0.92/+0.90 | +1.09/+1.07 |
| Control, s.d. | +0.077/+0.077 | +0.081/+0.086 | +0.060/+0.060 | +0.066/+0.066 | +0.085/+0.076 | +0.096/+0.086 | +0.080/+0.086 |
| Patient 1 | | | | | | | |
| During TGA | −0.1/−0.4 | −0.9/−0.6 | +1.2/+1.8 | **−2.0**/−1.8 | **−2.4/−2.5** | +1.1/+0.6 | +1.3/+0.9 |
| Follow-up | +0.9/+0.8 | +0.6/+0.8 | +0.7/+1.3 | +0.9*/+0.6* | −0.4*/−0.5* | +0.7/−0.4 | −1.1/+0.8 |
| Patient 2 | | | | | | | |
| During TGA | **−2.7/−2.1** | −1.9*/−1.6* | **−4.2/−4.5** | **−2.7/−2.6** | **−2.2/−2.5** | −1.8/−1.6 | **−2.6/−2.4** |
| Follow-up | +1.4*/+1.4* | +0.7/+0.9 | −0.7*/−0.3* | +1.5*/+1.4* | +0.8*/+1.2* | +1.2*/+1.3* | **+2.4*/+2.3*** |
| Patient 3 | | | | | | | |
| During TGA | **−2.2/−3.1** | **−2.0**/−1.0 | **−3.5/−3.0** | **−3.0/−2.0** | **−4.1/−2.6** | −1.7/**−2.2** | **−3.0/−2.4** |
| Follow-up | 0*/−0.5* | **+2.3*/0** | −1.2*/−1.3 | **+2.9***/+1.4* | +0.8*/+1.8* | +0.5*/**+2.1*** | **+2.1***/+1.9* |
| Patient 4 | | | | | | | |
| During TGA | +0.1/−0.3 | 0/+0.7 | **−2.5/−2.2** | −0.2/+0.3 | 0/+0.5 | −0.6/−0.3 | −1.5/+0.7 |
| Follow-up | −0.1/−0.1 | +0.2/+0.6 | +1.8*/+0.3* | −0.3/−0.2 | −0.4/0 | +0.8/+1.5 | −0.9/−0.7 |
| Patient 5 | | | | | | | |
| During TGA | **−2.6/−2.6** | −1.2/−1.0 | −1.2/**−3.3** | −1.4/−1.1 | −1.3/−0.9 | +0.1/−0.2 | −1.7/−1.3 |
| Patient 6 | | | | | | | |
| During TGA | −0.6/+0.4 | −0.7/+0.2 | −1.8/+0.3 | **−2.7**/−1.5 | **−2.2/−2.0** | **+2.4/+3.0** | **+2.4/+2.4** |

*ROI for which differences between follow-up and acute stage values are ≥2 s.d.

Patients' z-scores are given (differences between patients' values and control means, divided by controls' s.d.). Values ≥ 2 s.d. are highlighted by boldface print.

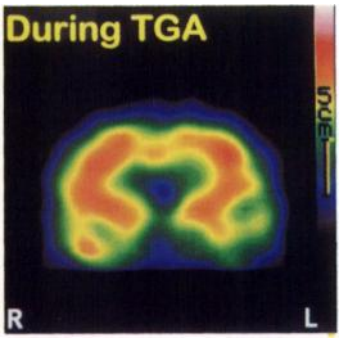


**FIGURE 2.** Circumscribed hypoperfusion of the left temporo-basal and temporo-lateral region during TGA (Patient 5). A small area of hypoperfusion is also present in the right temporo-medial region.

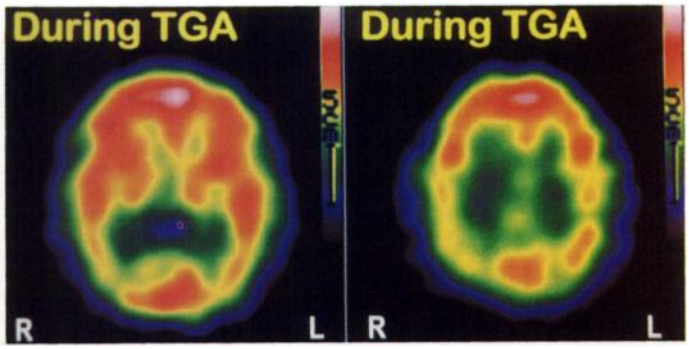


**FIGURE 3.** Bilateral, slightly asymmetric (left > right) hypoperfusion of the temporo-parieto-occipital region (left panel) and of the parietal lobe (right panel) during TGA (Patient 6).

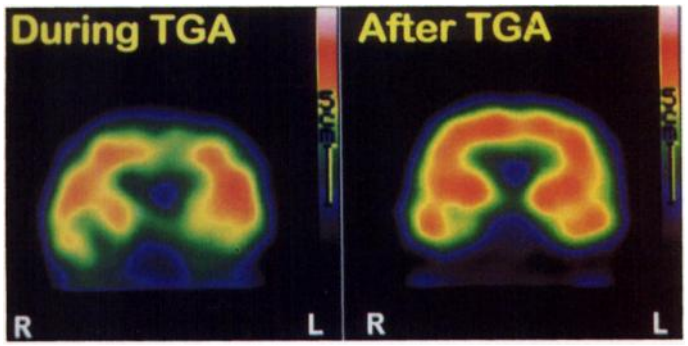


**FIGURE 4.** During TGA, Patient 2 exhibits a circumscribed hypoperfusion of the right temporo-medial region and of the entire left temporo-basal region. Less-pronounced hypoperfusion also is seen in the apical region and the basal ganglia (left > right). The follow-up SPECT of the same patient shows a normalized, homogeneous perfusion.

hypoperfusion and a more marked, asymmetric thalamic deficit (*17*). This case is atypical, in that abnormalities of perfusion and cognition persisted at follow-up studies performed 12 and 40 days later. Left temporo-medial hypoperfusion was reported in three further cases, but clinical data were not given (*18*).

Further studies were performed at a time when TGA had resolved. SPECT studies performed within 1 day after recovery showed transient left temporo-basal/temporo-medial hypoperfusion in two patients (*19,20*) and transient (possibly reactive) left temporo-medial hyperperfusion in another (*21*). Xenon-133 brain scans were normal in three patients studied 2, 24 and 38 hr after clinical recovery (*22*). Xenon-133 scans of five patients, three of whom had migraine attacks during the episode, showed an abnormal vasomotor response to the inhalation of $CO_2$ 1–5 days after TGA. Three cases showed additional variable cortical hypoperfusion (*23*).

### Implications for Transient Global Amnesia Pathophysiology

*Location.* The comparability between studies is limited by differences in the methods applied. However, some of the present results are comparable to those of previous single-case studies. It is confirmed that transient temporo-basal/temporo-medial hypoperfusion represents a hallmark of TGA, although not all cases show this pattern. Because disturbances of the hippocampal system that elicit amnesia are usually bilateral, the lack or unilaterality of perfusion abnormalities in a subset of cases may be explained by registrations performed at a time when previously bilateral disturbances had begun to resolve.

It also has become clear that TGA-related hypoperfusion can affect widespread neocortical areas. This finding may relate to differences in the expression of retrograde amnesia, in that the retrieval of existing contents of memory requires the integrity of neocortical, specifically, frontal and temporal association areas, whereas the establishment of new episodic memories depends predominantly on the function of the hippocampal system (*20*). The variability of perfusion changes can explain why some patients exhibit behavioral abnormalities during TGA, as well as cognitive deficits outside the domain of memory (*24*). The presence of transient thalamic and striatal hypoperfusion in two of our cases and one earlier case (*17*) shows, furthermore, that TGA is not a purely cortical phenomenon. The observation of striatal and thalamic hyperperfusion in one case (Patient 6) remains unexplained. Whether it represents a reactive change after preceding hypoperfusion is speculative.

*Time Course.* Previous findings suggest that perfusion changes usually normalize shortly after TGA. However, transient temporo-basal/temporo-medial hypoperfusion can outlast clinical recovery for up to 1 day, which conforms to neuropsychological findings of significant anterograde memory impairment persisting for many hours after clinical recovery (*2,15*). Abnormalities that persist for more than 1 day appear to be confined to patients with coexisting vascular risk factors or migraine (*19,23,25*).

Comparison of our Patients 1–3 to Patients 4–6 shows that hypoperfusion outside the temporo-basal region was less extensive in the latter subgroup, whose amnesia had begun to remit at the time of study. This finding suggests that global hypoperfusion, if present, tends to remit early.

## CONCLUSION

CBF changes observed during TGA are not uniform across studies or within our own case series. This may be due, in part, to differences in the timing and methodology of imaging. However, it seems clear that substantial hypoperfusion, particularly in the temporo-basal/temporo-medial region, is regularly present during the attack. As in the case of the migraine aura, it is unknown whether hypoperfusion represents the primary event or whether it is secondary to decreased metabolism. Although the single-case PET study of Baron et al. (*24*), performed at a time of advanced recovery, showed a matched reduction of neocortical perfusion and oxygen metabolism, which argues against primary ischemia, Volpe et al. (*26*) observed a disproportional decrease of temporo-medial oxygen metabolism during TGA. However, it may well be the case that both vasomotor and neuronal dysfunction play a role during the evolution of a TGA attack.

The finding of widespread neocortical hypoperfusion in a subset of patients argues against a vasomotor dysfunction restricted to the posterior territory. It also argues against a neuronal event that is confined to the hippocampi, such as spreading depression (*27*). One possibility is that TGA-related perfusion changes are caused by a perturbation within subcortical structures, such as the thalamus or aminergic upper brainstem nuclei, that project diffusely to the hippocampi and neocortex. The present finding of striatal and thalamic hypo-

perfusion in a subset of patients conforms to this hypothesis. Because the use of HMPAO precludes the determination of absolute perfusion, the distinct possibility remains that a global CBF deficit, including the cerebellum, can occur during TGA. Further imaging studies, specifically with PET, are needed to elucidate the chain of events that elicit TGA.


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# Correlations Between Uptake of Technetium-99m Q12 and Thallium-201: Myocardial Perfusion and Viability in a Model of Acute Coronary Reperfusion


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To investigate whether Q12 uptake is affected by myocardial viability, as has been noted for $^{201}$Tl and sestamibi, we analyzed the initial and delayed distribution patterns of Q12 in a rat coronary artery occlusion–reperfusion model. **Methods:** Animals were intubated and ventilated, and their arterial pressures were monitored. A left thoracotomy was performed. After a 1-hr occlusion and a 1-hr reperfusion of a major branch of the circumflex artery, $^{201}$Tl and Q12 were injected intravenously. Radiolabeled microspheres were used to document the areas of risk and reperfusion. The animals were killed at 5 min or 1 hr after administration of the diffusible tracers. Tracer distribution was determined by segmental tissue analysis, and tissue viability was determined by histochemical staining. **Results:** Both the initial uptake and delayed retention of Q12 are sensitive to myocardial viability as shown by significantly lower uptake (28% ± 8%) and retention (41% ± 13%) of Q12 in the nonviable as compared to the viable segments ($p < 0.001$). In addition, the myocardial retention of Q12 was significantly less in the nonviable tissue when compared to the initial uptake ($p < 0.01$). **Conclusion:** The clinical implication of these observations suggests that initial and delayed imaging after Q12 administration would reflect both the initial regional blood flow pattern and myocardial viability. Also, delayed imaging of Q12 may reflect viability better than the initial imaging.



**Key Words:** technetium-99m Q12; myocardial viability; ischemia; coronary artery disease; thallium-201

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