# Ipsilateral Thalamic Hypoperfusion on Interictal SPECT in Temporal Lobe Epilepsy

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Interictal brain SPECT is useful for the localization of a seizure focus. Concomitant hypoperfusion of the ipsilateral thalamus on interictal SPECT has been noted for temporal lobe epilepsy. In this study, we aimed to evaluate the prevalence of thalamic hypoperfusion ipsilateral to temporal hypoperfusion (ipsilateral thalamic hypoperfusion) and to assess the usefulness of this finding for the lateralization of epileptic foci on interictal SPECT for temporal lobe epilepsy patients. **Methods:** Forty-six patients with refractory temporal lobe epilepsy underwent interictal brain SPECT after intravenous injection of 555–740 MBq of $^{99m}$Tc-ECD. Perfusion impairments in the brain, especially the temporal lobe and thalamus, were evaluated. The localization of seizure foci was determined in conjunction with scalp, ictal and cortical electroencephalography, MRI and clinical outcomes. Ictal SPECT was performed for 5 of the 12 patients. **Results:** Concomitant decreased perfusion in both the temporal lobe and the ipsilateral thalamus was observed for 12 (26%) of 46 temporal lobe epilepsy patients on interictal brain SPECT. Seven patients showed hypoperfusion in the left temporal lobe and ipsilateral thalamus. Five patients showed hypoperfusion in the right temporal lobe and ipsilateral thalamus. In addition, hypoperfusion in the ipsilateral basal ganglia (ten patients) or contralateral cerebellum (four patients) was observed. **Conclusion:** Ipsilateral thalamic hypoperfusion is not uncommon in temporal lobe epilepsy. The exact mechanism causing ipsilateral thalamic hypoperfusion is uncertain; however, corticothalamic diaschisis may be an important factor. This finding may aid in the lateralization of seizure foci on interictal brain SPECT.

**Key Words:** temporal lobe epilepsy; diaschisis; interictal brain SPECT

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SPECT provides a unique opportunity to investigate cerebral blood flow on interictal scans. Temporal hypoperfusion is a characteristic finding for temporal lobe epilepsy. However, extratemporal hypoperfusion in temporal lobe epilepsy patients is not well described in the literature. Previous studies with interictal $^{18}$F-fluorodeoxyglucose (FDG) PET showed extratemporal hypometabolism in well-documented cases of unilateral mesial temporal lobe epilepsy (*1–4*). Recently, hyperperfusion within the cerebellum contralateral to seizure foci was reported for ictal SPECT, reflecting an alteration of blood flow via a neuronal connection in epilepsy patients (*5–7*). Won et al. (*5*) reported that this finding was common (75%) with ictal brain SPECT for seizure patients and that it may aid in the lateralization of epileptic foci despite lack of a typical uptake pattern on ictal SPECT or surface electroencephalography (EEG). Duncan et al. (*6*) also reported that a patient with focal epilepsy showed crossed cerebellar hyperperfusion on ictal SPECT and slight hypoperfusion in the ictal focus and symmetrically normal uptake in the cerebellum on interictal SPECT.

However, the significance of extratemporal hypometabolism in temporal lobe epilepsy still remains unclear. Moreover, the characteristics of the alteration of perfusion in the thalamus in temporal lobe epilepsy patients have not been well documented.

We observed decreased perfusion in the ipsilateral thalamus of patients with temporal lobe epilepsy on interictal brain SPECT. We assessed the usefulness of this ancillary finding for the lateralization of epileptic foci on interictal scans and attempted to define the role of thalamic dysfunction in the pathophysiology of temporal lobe epilepsy.

## MATERIALS AND METHODS

### Clinical Data

Interictal brain SPECT images obtained with $^{99m}$Tc-ECD in 67 patients with epilepsy were included in this study. The localization of seizure foci was determined in conjunction with scalp, ictal and cortical EEG, MRI and clinical outcomes. The seizure foci were right temporal in 22, left temporal in 24, right frontal in 1, left frontal in 1, left occipital in 2 and unlocalized in 17. Of the 17 patients with unlocalized foci, 13 had generalized seizures and 4 had partial seizures. Twelve patients were chosen for inclusion in the final study group on the basis of the finding of concomitant decreased perfusion in the temporal lobe and the ipsilateral thalamus on interictal brain SPECT. All patients had temporal lobe epilepsy; 4 of 12 patients had secondary generalized epilepsy as a prominent clinical problem. The seizure foci were right temporal in five and left temporal in seven. The patients were 10–34 yr old. MRI was performed for all patients and included fast spinecho T2-weighted coronal and oblique axial images but not quantitative volumetric measurement. A selective intracarotid sodium amobarbital injection test to assess hemispheric language dominance and memory and bilateral carotid and vertebral angiography were performed for five patients who underwent surgical therapy for the epileptic foci and showed mesial temporal sclerosis in four patients and hippocampal dysgenesis in one. Ictal EEG and SPECT were performed for five patients. Electrocorticography was performed for four patients.

### SPECT Procedures

After an intravenous injection of 15–20 mCi (555–740 MBq) $^{99m}$Tc-ECD, interictal brain SPECT was performed by use of a brain-dedicated gamma camera (CERASPECT; Digital Scintigraphic, Inc., Waltham, MA) equipped with high-resolution, low-energy, parallel-hole collimators. A total of 120 projections were acquired with a 3° angular increment. Transaxial images were obtained by filtered backprojection methods with a Butterworth filter (Nyquist frequency, 1.1 cycles per cm at order no. 10), and attenuation correction was performed by Chang's method. The reconstructed slices were displayed on a 128 × 128 matrix (1.67 × 1.67 mm) as a set of 64 slices (1.67-mm slice thickness). Coronal and sagittal images were generated from the original transaxial

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**TABLE 1**
Demographic Data and MRI and Interictal SPECT Findings

| Patient no. | Sex/ Age (yr) | Age at onset (yr) | Seizure type | Surface EEG result | MRI result | Interictal SPECT result |
|---|---|---|---|---|---|---|
| 1 | F/26 | 1 | CP to 2'GTC | LT | LHA | LT, Th, BG |
| 2 | M/33 | 9 | CP | LT | LHA | LT, Th |
| 3 | F/23 | 10 | CP to 2'GTC | RT | NL | LT, Th, BG |
| 4 | M/34 | 12 | CP | BT | RHA | RT, Th |
| 5 | F/17 | 14 | CP to 2'GTC | RT | RHA | RT, Th |
| 6 | M/35 | 14 | CP | RT | LHA | LT, Th |
| 7 | F/24 | 21 | CP | BT | LHA | LT, Th |
| 8 | F/30 | 22 | CP | RT | RHA | RT, Th, BG |
| 9 | M/10 | 8 | CP | LT | NL | LT, Th |
| 10 | M/31 | 18 | CP to 2'GTC | LT | NL | LT, Th, BG |
| 11 | F/22 | 9 | CP | LT | RHA | RT, Th |
| 12 | F/16 | 11 | CP | NL | NL | RT, Th |

CP = complex partial seizure; GTC = generalized tonic clonic seizure; LT = left temporal; RT = right temporal; BT = bitemporal; NL = normal; LHA = left hippocampal atrophy; RHA = right hippocampal atrophy; Th = thalamus; BG = basal ganglia. Locations for interictal SPECT indicate the areas of decreased perfusion.

images (parallel to the orbitomeatal line). Additionally, transaxial images parallel to the long axis of the temporal lobes were obtained for evaluation of the temporal lobes. The slice thickness was 1.67 or 3.34 mm. Ictal scans were obtained for 5 of the 12 patients.

SPECT findings were qualitatively evaluated by two experienced nuclear medicine specialists who were blinded to EEG findings, clinical information or other imaging findings and who evaluated cortical and subcortical regions, including the thalamus and basal ganglia.

Interictal PET findings were available for two patients and were compared with interictal SPECT findings.

## RESULTS

A concomitant decrease in perfusion in both the temporal lobe and the ipsilateral thalamus was observed for 12 (26%) of 46 patients with temporal lobe epilepsy on interictal brain SPECT. No structural abnormality was observed by MRI, except for mesial temporal sclerosis in all patients. Bilateral carotid and vertebral angiography for five patients showed no gross vascular abnormality. Hypoperfusion in the ipsilateral thalamus was not found for patients with extratemporal lobe epilepsy.

Demographic data and MRI findings for these 12 patients are summarized in Table 1. Brain SPECT of seven patients revealed hypoperfusion in both the left temporal lobe and the ipsilateral thalamus (Fig. 1). Of those seven patients, three also showed hypoperfusion in the ipsilateral basal ganglia. On MRI, mesial temporal sclerosis was found for four patients, but hippocampi were normal in the remaining three. In four of the seven patients showing thalamic hypoperfusion, localization by interictal SPECT was concordant with that by surface EEG, it was discordant in two patients and unlocalized in one. Localization by electrocorticography was concordant with localization by interictal SPECT and MRI in two patients.

Five patients showed hypoperfusion in both the right temporal lobe and the ipsilateral thalamus (Fig. 2). On MRI, mesial temporal sclerosis was found in four patients, but normal findings were obtained for one. For these five patients, localization by interictal SPECT was concordant with that by surface EEG in two patients, it was discordant in one patient and unlocalized in two. Localization by electrocorticography was concordant with localization by interictal SPECT and MRI in two patients.

Five of the 12 patients for whom interictal SPECT showed hypoperfused areas in the temporal lobe and thalamus were shown to have hyperperfused areas on ictal SPECT (Fig. 3).

Interictal PET studies with FDG, which were available for two patients, revealed temporal, thalamic and/or basal ganglion hypometabolism; thus, the PET findings were concordant with the interictal SPECT findings.

Ipsilateral thalamic hypoperfusion was not observed in the remaining 34 of 46 patients with temporal lobe epilepsy; however, hypoperfusion in ipsilateral basal ganglia (six patients) and the contralateral cerebellum (four patients) was observed. Hypoperfusion of the contralateral thalamus was not seen in any patients.

Between patients with thalamic hypoperfusion and those without it, clinical differences were not observed. Twelve patients with ipsilateral thalamic hypoperfusion did not show any neurologic deficit corresponding to thalamic hypofunction, such as delayed speech, memory impairment and/or neglect.

## DISCUSSION

Concomitant hypoperfusion in the ipsilateral thalamus was observed in 26% of temporal lobe epilepsy patients. The exact etiologic factor contributing to thalamic hypoperfusion remains to be elucidated, but diaschisis and thalamic dysfunction related to temporal lobe epilepsy are possible contributing factors.

Diaschisis is defined as hypoperfusion and hypometabolism in a portion of the brain distant from the site of damage because of an interruption of its afferent axonal supply (*8*), originally used by Von Monakow in 1914 (*9*). The classical definition of diaschisis is a reversible, functional phenomenon without structural changes. Primary metabolic suppression from diaschisis causes reductions in regional cerebral blood flow, rate of oxygen metabolism and rate of glucose metabolism. However, the normal oxygen extraction rate is crucial for differentiating primary metabolic suppression attributable to diaschisis from the ischemic condition (*10*). Crossed cerebellar diaschisis (CCD) is well known in supratentorial ischemic lesions or other clinical circumstances, including brain tumors, vascular malformation and hemorrhages (*11,12*). In patients with cerebral infarction, the location, rather than the extent and severity of the lesion, may be the major determinant for the occurrence and magnitude of CCD. Although once CCD occurs, the infarct size may play a potential role in determining the magnitude of CCD (*13*).

Diaschisis and its clinical importance in regions other than

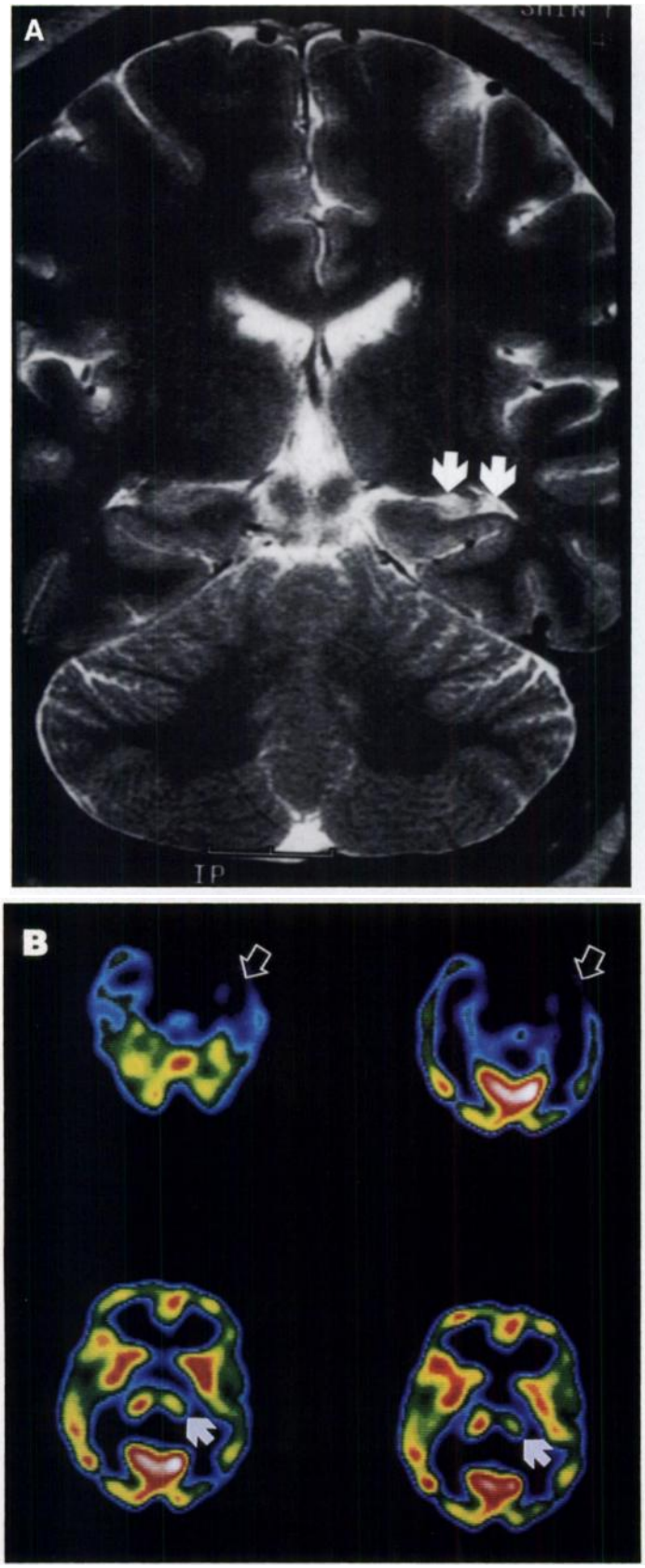


**FIGURE 1.** Scans of a 26-yr-old female patient with left temporal lobe epilepsy. (A) Coronal T2-weighted MRI shows increased signal intensity and atrophy of left hippocampus (arrows). (B) Transaxial reconstructed SPECT images show decreased perfusion of left temporal lobe (open arrows) and left thalamus (closed arrows).

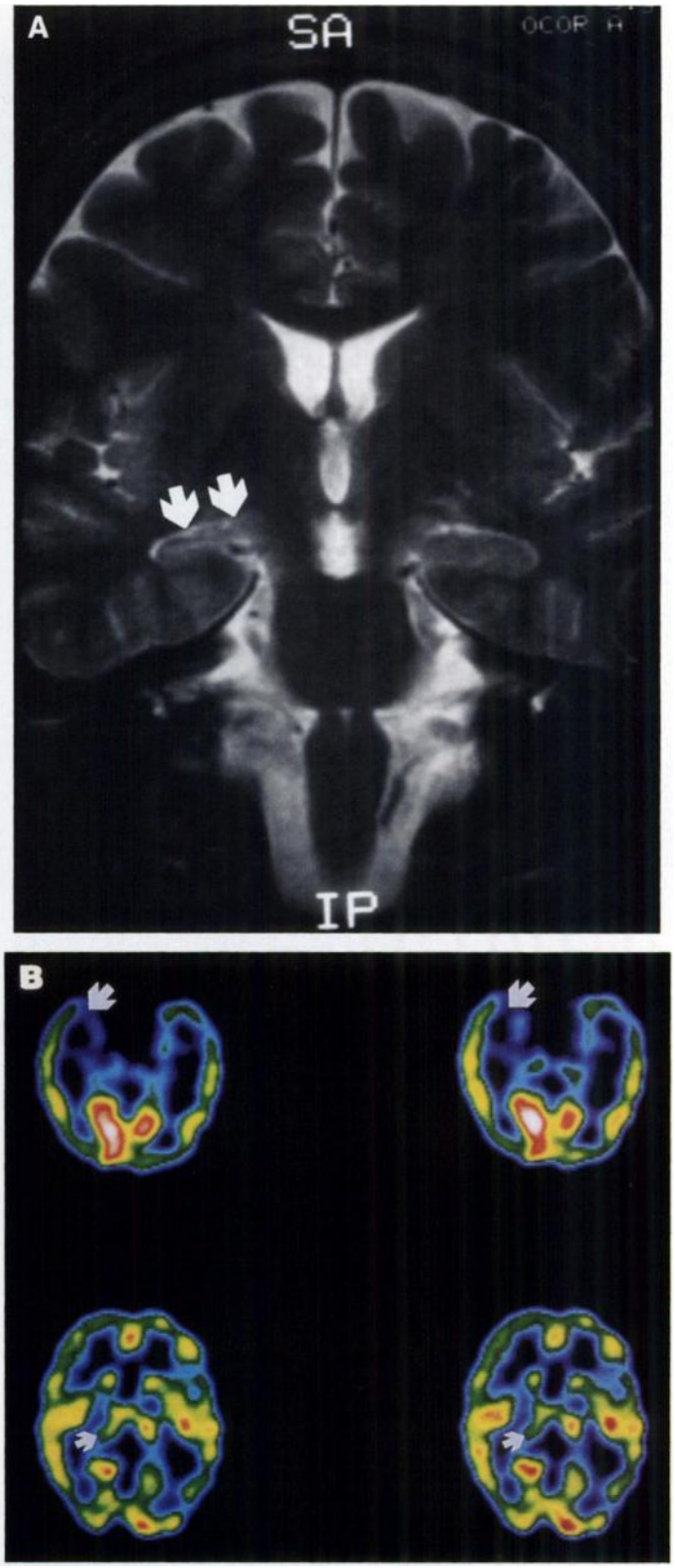


**FIGURE 2.** Scans of a 17-yr-old female patient with right temporal lobe epilepsy. (A) Coronal T2-weighted MRI shows increased signal intensity and atrophy of right hippocampus (arrows), consistent with mesial temporal sclerosis. SA = superoanterior; IP = inferoposterior. (B) Transaxial reconstructed SPECT images show decreased perfusion of right temporal lobe (large arrows) and ipsilateral thalamus (small arrows).

the contralateral cerebellum have yet to be documented. Wise et al. (*14*) observed a 30%–40% decrease in cerebral blood flow and cerebral rate of oxygen metabolism in the thalamus ipsilateral to a cerebral infarction. In contrast, Baron et al. (*15*) reported that thalamocortical diaschisis occurred as a depression in bilateral cortical metabolism as a result of a unilateral thalamic lesion on PET scans. The mechanism of thalamocor-

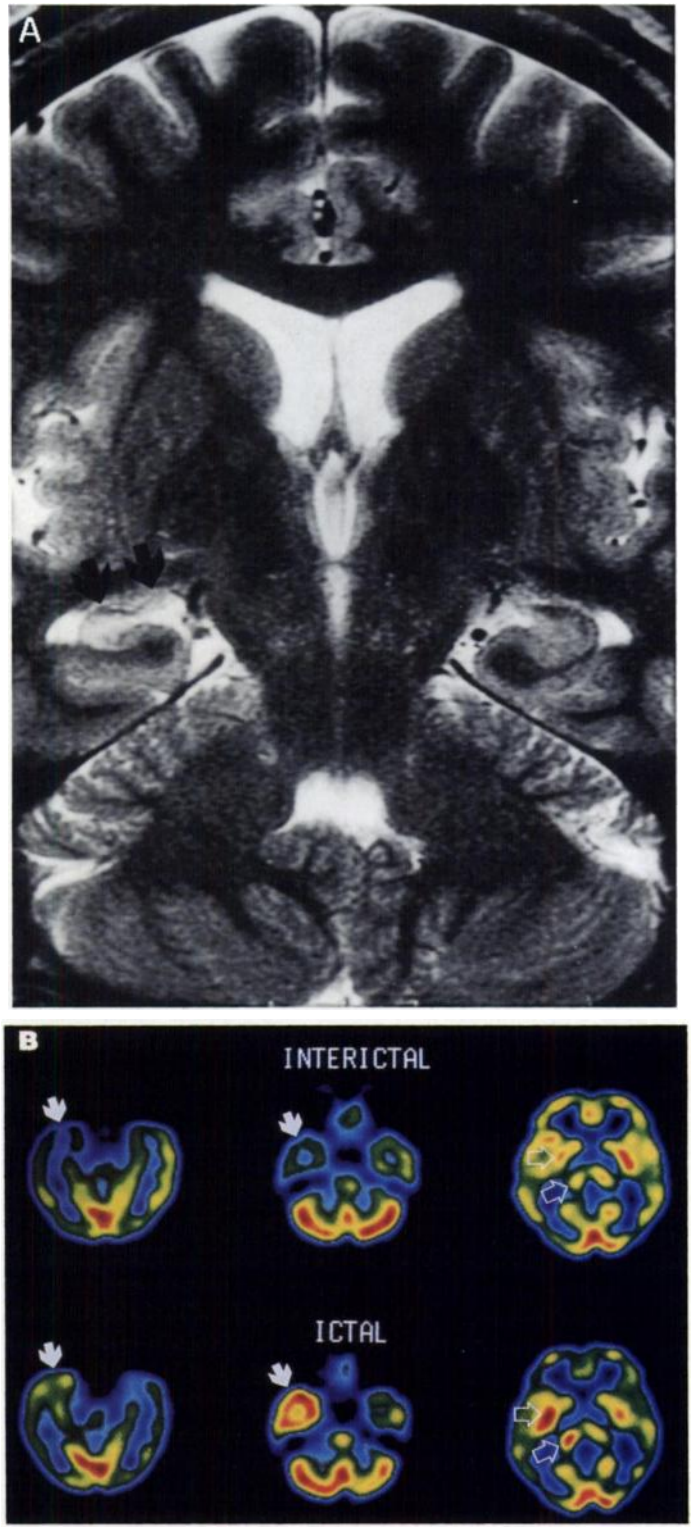


**FIGURE 3.** Scans of a 34-yr-old female patient with right temporal lobe epilepsy. (A) Coronal T2-weighted MRI shows increased signal intensity and atrophy of right hippocampus (arrow), suggesting mesial temporal sclerosis. (B) Transaxial reconstructed interictal SPECT images show decreased perfusion of right temporal lobe (closed arrows) and ipsilateral thalamus and basal ganglia (open arrows). On ictal SPECT, right temporal lobe (closed arrows) and right thalamus and basal ganglia (open arrows) show increased perfusion when compared with the left side.

tical diaschisis was thought to be an interruption of cortical afferents originating from the thalamus.

Several studies done with interictal FDG-PET have found evidence of thalamic hypometabolism ipsilateral to temporal hypometabolism in temporal lobe epilepsy patients (*1–4*). Henry et al. (*3*) reported abnormal regional asymmetry of the thalamus in 63% of 27 patients and of the basal ganglia in 41%. The prevalence of thalamic hypometabolism suggested a pathophysiologic role for the thalamus in the initiation or propagation of temporal lobe seizures or in the interictal cognitive dysfunction of temporal lobe epilepsy (*3*). Other reports have suggested that subcortical hypometabolism is secondary to decreased efferent activity from the temporal lobe structures, especially the amygdala and hippocampus, to subcortical nuclei. Moreover, diminished subcortical activity may lead to defective regulation of cortical excitability in the temporal lobe, increasing the likelihood of seizure development and spread (*2*).

Studies of monosynaptic pathways in primates have shown dense ipsilateral, reciprocal connections among the amygdala, hippocampal formation and entorhinal cortex (*16–18*). Primate midline thalamic nuclei, including the nucleus reuniens, are reciprocally connected to the ipsilateral amygdala and hippocampal formation; the hippocampal formation is also reciprocally connected to the lateral dorsal and anterior thalamic nuclei (*16,19–21*). In primates, the inferior and lateral temporal neocortices also have dense monosynaptic, reciprocal connections ipsilateral to the amygdala and the hippocampal formation. Thus, anterograde and retrograde axonal tracing techniques disclose unilateral monosynaptic, reciprocal projections among widespread mesial and lateral temporal areas and the thalamus in primates (*16,18,19*). Furthermore, the amygdala and anterior portions of the hippocampal formation (which are most often involved in both the electrographic ictal onset zone and the neuronal loss of mesial temporal lobe epilepsy) are included in all of these reciprocal connections (*22*).

Findings of thalamic hypoperfusion ipsilateral to temporal hypoperfusion without any evidence of morphologic abnormalities on MRI and cerebral angiography may reflect decreased efferent activity from the temporal lobe structures as well as the alteration of a reciprocal connection between the temporal lobe structures and the thalamus. It is of interest to note that the hypoperfused thalamus and basal ganglia on interictal SPECT were hyperperfused on ictal SPECT; this finding is related to the functional alteration of subcortical structures. This ipsilateral thalamic hypoperfusion has not been reported in studies of interictal brain SPECT up to now, although several PET investigations have shown subcortical alteration of metabolism in interictal studies. Our results from interictal PET studies for two patients were almost the same as those from earlier PET studies. Improved detection of thalamic hypoperfusion on interictal SPECT may have been attributable to the improved spatial resolution of the brain-dedicated annular crystal gamma camera used in our study (*23*).

Recent studies have shown that the thalamus may play a primary role in the initiation and/or propagation of seizures in several types of epileptic disorders (*24–30*). Urich (*24*) presented the idea of thalamic lesions representing ictal brain damage. Mori et al. (*25*) performed a neuropathological study on 14 older patients who died after status epilepticus. They suggested that the thalamus was damaged by the seizures themselves and not by secondary degeneration from the cortex, as the corresponding cortices were little affected in these patients; ischemic changes in the neurons of the thalamus also supported this concept (*25*). Lin et al. (*29*) suggested that a selective effect of presynaptic $GABA_B$ receptors on GABA release in the neocortical and thalamic nuclei of lethargic mice may have contributed to the mechanism underlying the absence of seizures. Yan et al. (*30*) suggested that the thalamic deficiency in norepinephrine release detected via intracerebral microdialysis may have contributed to seizure predisposition

through a failure to provide a normal level of protection against initiation and spread. Although the thalamus plays a role in the initiation, propagation and/or symptoms of seizures, it is uncertain whether thalamic dysfunction is related to alterations in thalamic blood flow, and thalamic perfusion is reduced in an interictal state. Further investigation of thalamic dysfunction and alterations in blood flow is needed.

## CONCLUSION

Although ipsilateral thalamic hypoperfusion on interictal SPECT is too small to assist in seizure localization, it may aid in the localization of seizure foci. This finding occurs ipsilateral to epileptic foci in the temporal lobe, not in the contralateral thalamus. This finding may reflect either thalamic hypometabolism secondary to decreased efferent activity from the temporal lobe structures or functional alteration of thalamic metabolism by the regulation of cortical excitability and seizure propagation or both.

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# SPECT Brain Imaging in Epilepsy: A Meta-Analysis


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A meta-analysis of SPECT brain imaging in epilepsy was performed to derive the sensitivity and specificity of interictal, postictal or ictal rCBF patterns to identify a seizure focus in medically refractory patients. **Methods:** Papers were obtained by pooling all published articles identified by two independent literature searches: (a) Dialnet (EMBASE) or Radline by CD-ROM and (b) Current Contents searched manually. Literature inclusion criteria were: (a) patients had a localization-related epileptic syndrome; (b) more than six patients were reported; and (c) patients had at least an interictal EEG-documented epileptiform abnormality. Of 46 papers meeting these criteria, 30 contained extractable data. SPECT results were compared to localization by standard diagnostic evaluation and surgical outcome. Meta-analytic sensitivities for SPECT localization in patients with temporal lobe seizures relative to diagnostic evaluation were 0.44 (interictal), 0.75 (postictal) and 0.97 (ictal). Similar results were obtained relative to surgical outcome. False-positive rates were low relative to diagnostic evaluation (7.4% for interictal and 1.5% for postictal studies) and surgical outcome (4.4% for interictal and 0.0% for postictal studies). **Results:** The results were not dependent on tracer used (or dose), the presence of CT-identified structural abnormalities, blinding of image interpretation or camera quality (although data were more variable with low-resolution cameras). There were insufficient data for conclusions regarding extratemporal-seizure or pediatric epilepsy populations. **Conclusion:** Insights gained from reviewing this literature yielded recommendations for minimal information that should be provided in future reports. Additional recommendations regarding the nature and focus of future studies also are provided. The most important of these is that institutions using SPECT imaging in epilepsy should perform ictal, preferably, or postictal scanning in combination with interictal scanning.



**Key Words:** epilepsy; meta-analysis; SPECT

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