## Crossed Cerebellar Diaschisis: Related to Lesion Location or Disease Duration?

**TO THE EDITOR:** The article by Kim et al. (*1*) draws the conclusion that the location, rather than the extent and severity, of the lesion may be the major determinant for the occurrence and magnitude of crossed cerebellar diaschisis (CCD) in patients with cerebral infarction. They reported that both the frequency and severity of CCD are greater in patients with infarction in either the frontoparietal lobes (FP) or the deep middle cerebral artery (MCA) territory including basal ganglia and internal capsule, as compared to patients with infarcts of other regions. Their results were based on 26 patients who suffered from a single supratentorial infarction. However, the interval between onset of stroke and examination time of $^{99m}$Tc-HMPAO SPECT and MRI varied widely (between 5 and 1825 days after stroke). This may pose a major problem.

Although some authors have reported an unchanged CCD in older stroke patients as compared with acute ones, many studies do report that CCD can be resolved spontaneously sometime after stroke (*2,3*). In Kim et al.'s (*1*) study, of the 14 patients without CCD, 12 (85.7%) patients had stroke duration over 30 days. In contrast, of the 12 patients with CCD, only 4 (33.3%) patients had duration over 30 days. The p value was 0.009 by a Fisher test.

We tabulated the relationship between CCD and time of stroke according to the locations of stroke based on Kim et al.'s (*1*) data as follows:

| | Location of stroke | No. | CCD | No CCD |
|---|---|---|---|---|
| Group 1 | Fronto-parietal lobes and deep MCA territory | | | |
| | Disease duration <30 days | 8 | 7 | 1 |
| | Disease duration >30 days | 11 | 4 | 7 |
| Group 2 | Other regions | | | |
| | Disease duration <30 days | 2 | 1 | 1 |
| | Disease duration >30 days | 5 | 0 | 5 |

In Group 1, most of the patients with CCD had disease duration less than 30 days. In contrast, 7/11 (63.6%) patients with disease duration over 30 days had no CCD even with the lesions located in the FP and deep MCA territory. In Group 2, 50% (1/2) patients with disease duration within 30 days had CCD even with lesions located in the so-called other region. All of the patients without CCD had disease duration over 30 days. The disease duration seemed to play a more important role than lesion location in causing a CCD phenomenon.

Based on these data, it does not seem reasonable to draw any conclusion.

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## Septal Perfusion in Left Bundle Branch Block

**TO THE EDITOR:** We read with interest the recent article by Sugihara et al. (*1*) concerning septal perfusion in patients with left bundle branch block (LBBB). The authors found that reduced septal wall thickening in patients with LBBB may mimic hypoperfusion and they conclude that an apparent hypoperfusion on nongated images is the result of reduced wall motion rather than true hypoperfusion at rest.

We agree with Sugihara et al. (*1*) that the assessment of septal perfusion in patients with LBBB is difficult. As could be anticipated from previous studies, any wall motion abnormality causes artifactual defects in the apparent tracer distribution in myocardial SPECT (*2,3*). Consequently, the presented results are not surprising. However, we would like to comment on the relevance of the observed phenomenon.

During data acquisition in myocardial SPECT, the patients are in a resting condition. The image acquired is the "frozen image" of myocardial perfusion at the time of injection. This generally applies to all myocardial perfusion tracers, even if the time interval between injection and imaging may be shorter (as with $^{201}$Tl) or longer (as with $^{99m}$Tc-sestamibi). Since during acquisition the patients are equally in the resting condition, artifacts due to reduced septal wall thickening should be the same irrespective of the type of study (stress versus rest). Any difference in septal tracer uptake between stress and rest acquisitions can, therefore, not be related solely to an artifact caused by reduced septal wall motion.

In patients with LBBB, septal redistribution has been described when using $^{201}$Tl and a stress-rest protocol. There is controversy about the pattern found with $^{99m}$Tc-sestamibi (fixed defect in stress and rest studies, completely normal uptake, or a fill-in in the rest study). The observed phenomenon does not explain studies showing a normal septal uptake of $^{99m}$Tc-sestamibi at rest and does not contribute to the understanding of the frequently reported septal redistribution of $^{201}$Tl.

Furthermore, the diagnostic accuracy of myocardial SPECT in patients with LBBB has been reported to be superior with dipyridamole as compared with physical exercise using either $^{201}$Tl (*4*) or $^{99m}$Tc-sestamibi (*5*). This improvement in accuracy is related to the dipyridamole effect at the time of tracer injection and not to any effect at the time of imaging. The septal wall motion abnormality does not seem to be of major importance in this context.

Experimental studies with right ventricular pacing in dogs have shown that LBBB itself may reduce myocardial perfusion and glucose uptake in the septum presumably due to asynchrony of left ventricular contraction with an augmented intramyocardial pressure (*6*).

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## Septal Perfusion in Left Bundle Branch Block

**REPLY:** We thank Drs. Richter and Munz for their interest in our article. Their comments can be summarized in two issues: (*1*) septal perfusion for diagnostic accuracy for CAD detection and (*2*) pathophysiological considerations in patients with left bundle branch block (LBBB).

We agree with their comments on stress perfusion abnormality in septal regions. Many authors pointed out the limited values of exercise perfusion imaging in LBBB patients. Stress-induced perfusion defect is often seen in septal regions in these patients without organic coronary stenosis (*1–3*), but the specificity of perfusion imaging may be significantly increased by use of dipyridamole stress instead of exercise stress (*4,5*). On the other hand, caution is needed to interpret perfusion abnormality, since stress-induced septal hypoperfusion and LBBB is often caused by left anterior descending artery stenosis which improved after coronary angioplasty (*6*).

Our article did not intend to improve diagnostic accuracy by use of gated SPECT acquisition in stress myocardial perfusion study in these patients. All of our data were collected at resting condition. Our study indicates a decrease in tracer uptake in septal region mimicking "hypoperfusion" in nongated perfusion study due to decreased wall thickening in this area. But, this artifact can be eliminated by use of end-diastolic images with gated acquisition. Thus, gated perfusion studies can assess perfusion and function separately. This technique can be applied for evaluation of pathophysiological conditions in the patients with various cardiac disorders, such as stunned myocardium where regional dysfunction persists after recovery of perfusion.

Another issue is the potential mechanism of a decrease in regional wall thickening in septal region. Ono et al. (*7*) showed a decrease in perfusion and metabolism in experimental LBBB induced by right ventricular pacing. Such decrease may be considered as a result of increased intramyocardial pressure. A decrease in glucose metabolism is often observed in LBBB patients as well on FDG-PET study (Fig. 1), indicating a decrease in energy metabolism without evidence of organic stenosis of coronary arteries, probably due to decreased contraction rather than myocardial ischemia. Further study with more cases is necessary. Echocardiographic studies indicated a reduced magnitude of systolic septal motion which may cause reduced left ventricular ejection fraction in these patients (*8,9*). Grines et al. (*8*) showed the displacement of the septum due to relative increase in right ventricular volume increase or pressure. This displacement may cause paradoxic systolic motion of the septum. However, precise assessment of regional function in relation to perfusion and metabolism may be required with radionuclide studies.

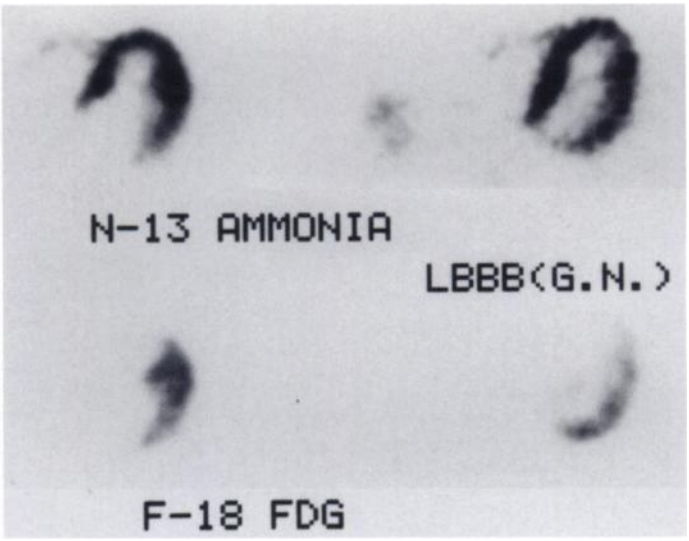


**FIGURE 1.** Two representative transverse slices of $^{13}$N-ammonia perfusion and $^{18}$F-FDG images of a patient with LBBB. While myocardial perfusion is normal, FDG uptake is significantly decreased in septal region, indicating a decrease in glucose utilization.

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