(2%) in all hypoperfused segments (n = 87). These 2 segments were in ischemic but noninfarcted, remote, myocardium. Thus, even the number of segments with relatively increased fatty acid uptake was less in our study compared with the study of Schulz et al. (2), using their threshold, instead of more, which is the major point raised by Buell et al.

Another major resemblance between both studies is the finding that a higher correlation was found between fatty acid uptake and perfusion: BMIPP(%) = 0.75*Tl(%)+23, r = 0.87 and n = 273, than FDG versus perfusion: FDG(%) = 0.70*Tl(%)+24, r = 0.75 and n = 273.

In addition to the differences in methods pointed out by Buell et al. (vide supra), the following differences in study design should be mentioned: the myocardial segments in our study were 13 in each heart versus 33 in the study of Schulz et al. (2). Furthermore, we used a reference database of healthy individuals to define normal or abnormal perfusion by $^{201}$Tl (1), whereas Schulz et al. (2) did not use a normal reference database but defined hypoperfusion by $^{99m}$Tc-hexakis-isobutyl isonitrile (MIBI) uptake <70% of peak uptake. Finally, they used an oral glucose load before FDG imaging, which results in lower target-to-background ratio compared with the glucose clamp or Acipimox (Byk, The Netherlands) protocol, as we applied it (5,6). By coincidence, both study groups consisted of only 21 patients, and therefore one may not be surprised that the relative numbers are not exactly the same. Still, the principle finding is the same.

The protocol we used has been proven to discriminate viable from nonviable myocardial segments, hence, satisfactorally predicting functional outcome after revascularization (7,8). Furthermore, it has been demonstrated in our institution that there is good agreement between the detection of viability in dyssynergic myocardium with FDG/$^{13}$N-ammonia PET and FDG/$^{201}$Tl SPECT (9). Therefore, we feel that their suggestion that our results may be influenced by the study design is incorrect. Obviously, the outcomes of both studies are to some extent influenced by study design and evaluation methods.

Our study did not contain patients with a left bundle branch block (LBBB), so we cannot comment on the issue of LBBB and substrate utilization.

Thus, the differences in methods, especially the threshold of a metabolism/perfusion difference to define a matched defect or mismatch, is largely responsible for the different numerical outcomes of both studies. Nevertheless, it is an important observation that the principle outcome is the same: increased fatty acid uptake relative to perfusion can be found in chronic ischemic myocardium.

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## Moyamoya Disease and Pregnancy

**TO THE EDITOR:** I read with great interest the article by Kume et al. (1) reporting on the usefulness of cerebral blood flow (CBF) mapping under hyperventilation for prediction of the risk of vaginal delivery of pregnant women with moyamoya disease.

My colleagues and I have recently reviewed the literature describing pregnant women with moyamoya disease (2). In the literature, there have been 53 pregnant women with moyamoya disease: 30 patients (group A) who had been diagnosed with moyamoya disease before pregnancy and were capable of delivery, and 23 patients (group B) who were symptomatic and diagnosed for the first time as having moyamoya disease associated with pregnancy. In group A, delivery could be performed safely either by cesarean or vaginal delivery, and any anesthetic method could be used, as long as special attention was given to avoid hypocapnia, hypotension and hypertension. Poor prognosis for the mother or the neonate was generally caused by cerebral hemorrhage in group B and not by cerebral ischemia in group A. In fact, neither cerebral ischemia nor cerebral hemorrhage developed during delivery in patients in group A, although only 11 patients from the group had undergone extracranial-intracranial bypass surgery when diagnosed with moyamoya disease. As Kume et al. (1) stated, hyperventilation challenge may be dangerous for patients with moyamoya disease. Thus, CBF mapping under hyperventilation could be dangerous and may give little information on the safety of vaginal delivery for the patients in group A. Accordingly, I do not believe that evaluation of cerebral vascular reserve using $^{99m}$Tc-hexamethyl propylenamine oxime (HMPAO) is necessary even if its radiation dose to the fetus is negligible. Instead, we should find the best method of delivery (vaginal or cesarean delivery) and anesthesia (general, epidural or spinal) which are familiar to the obstetric and anesthetic teams in each hospital to avoid hypocapnia (hyperventilation), hypotension and hypertension.

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**REPLY:** Dr. Komiyama has commented that it is not necessary to understand the risk associated with childbirth during brain SPECT for moyamoya disease, because patients have never complained of neurological symptoms.

We examined the relationship between complications of childbirth and neurological symptoms by reviewing our patients with moyamoya disease who had undergone surgery over the past 20 y. A preliminary analysis revealed that only 1 of 11 patients had a difficult delivery. They may or may not have had neurological symptoms associated with moyamoya disease. A study from the perspective of neurological surgery will be completed next year.

Although we feel that Dr. Komiyama's opinion may be correct, it is necessary to prove that abnormalities in cerebral blood flow (CBF) are not induced during childbirth. This is particularly true for natural delivery, because obstetricians in Japan recommend cesarean delivery rather than natural delivery to avoid complications during childbirth.

Brain SPECT should be performed during rehearsals of natural delivery using a minimal dose of technetium to show that CBF does not change. The results of this procedure should help convince obstetricians to opt for natural childbirth in patients with moyamoya disease.

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## Retreatment of Graves' Disease with Radioiodine $^{131}I$

**TO THE EDITOR:** Leslie et al. (*1*) reported their experience with retreatment of Graves' disease with similar deposited doses of radioiodine $^{131}I$. They found that 15.6% of their patients required a second dose, and there was no significant difference between the percentage of patients who remained hyperthyroid after either dose. In an important paper published in the *Journal* in 1971, not referenced by Leslie et al., Spencer (2) examined the outcomes in three large clinical series in which multiple doses (4–9) were administered, each dose yielding the same $^{131}I$ concentration per gram of thyroid. When the percentage uncured (i.e., still hyperthyroid) was plotted against number of doses on semilog paper, the data in each series fell in a straight line, indicating that the fraction of patients who remain hyperthyroid is constant for a given deposited dose.

In Figure 1, I have replotted the data of two of these series (*3,4*) and have added a third (*5*) from a more accessible publication. In each series, the same $^{131}I$ concentration was deposited at successive dosings, which in one series was carried out to 11 dose administrations. Spencer (2) concluded that the uncured fraction with any dose could be described by $H = H_0e^{-\lambda D}$, where H is the number of patients not cured after any dose, $H_0$ is the original number of patients, $\lambda$ is a constant describing the fractional cure rate per dose and D is the number of doses administered.

For Silver's series (*4*) (Fig. 1), 50% of patients ($H/H_0$) were still hyperthyroid after one dose, so that $\lambda = 0.693$, i.e., about 31% of patients were always uncured after every dose of this size (80 μCi/g). It is therefore not surprising that Leslie et al. (*1*) had the same failure rate after both dosings.

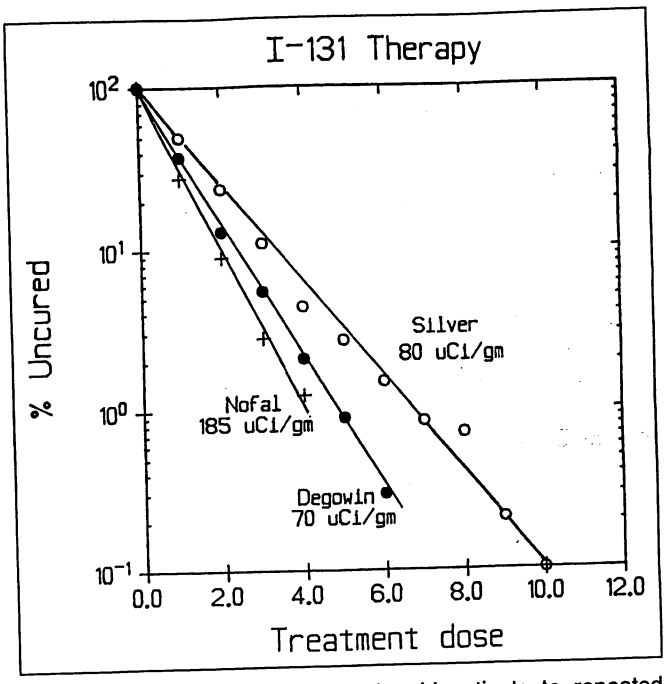


**FIGURE 1.** Response of hyperthyroid patients to repeated treatment with sodium iodide $^{131}I$ in same tissue concentrations. Vertical axis = percentage uncured (i.e., still hyperthyroid); horizontal axis = number of doses administered.

It appears that $\lambda$ is greatest for the highest $^{131}I$ concentration (Fig. 1). To get a better feel for this relationship, I have plotted the mean success rate for each of 12 series in which a constant $^{131}I$ concentration was used (Fig. 2). The mean success rate is the same as the success rate for each dose. There is a strong linear relationship, $y = 0.0018x + 0.48$, with a correlation coefficient (r)

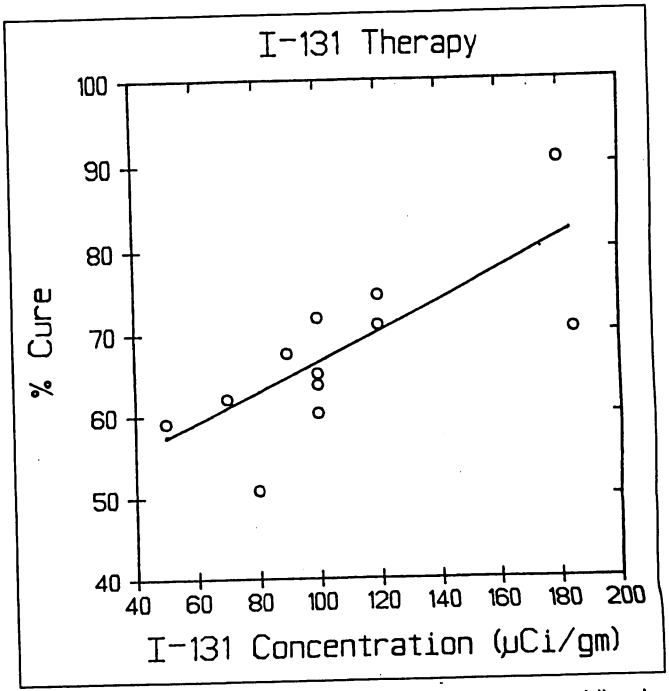


**FIGURE 2.** Percentage of patients cured of hyperthyroidism by treatment with sodium iodide $^{131}I$ at a given tissue concentration, in 12 published series. Vertical axis = percent of patients cured; horizontal axis = $^{131}I$ tissue concentration.