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## **Perfusion CT Scans**

Rüdiger von Kummer

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I read the article by Reichenbach et al (1) with interest and wonder why the CT scans in Figure 2B and C were considered as normal, although the lentiform nucleus is hypoattenuated (*right* in B and *left* in C) compared with the contralateral side. I further wonder how the authors could describe the sensitivity of perfusion CT for perfusion deficits without providing a reference method.

The same group of authors has since published a similar study, including 17 patients of the study presented in the *AJNR* article and adding five additional patients (2). Figure 2C of the *AJNR* article appears in Figure A in the *Archives of Neurology* article, and Figure 3 of the *AJNR* article is identical to Figure B in the *Archives of Neurology* article.

Comparing the patient data, one can see that there are similarities but also striking discrepancies regarding the data of individual patients. Because of the discrepancies regarding sex and age, I cannot identify the counterparts of patients 3, 7, and 11 in the *Archives of Neurology* article. The authors should explain why so many patients differed in both studies regarding time to CT, initial perfusion deficit, treatment, and volume of infarct on follow-up CT scans.

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## References

1. Reichenbach JR, Rother J, Jonetz-Mentzel L, et al. **Acute stroke evaluated by time-to-peak mapping during initial and early follow-up perfusion CT studies.** *AJNR Am J Neuroradiol* 1999;20:1842-1850
2. Rother J, Jonetz-Mentzel L, Fiala A, et al. **Hemodynamic assessment of acute stroke using dynamic single-slice computed tomographic perfusion imaging.** *Arch Neurol* 2000;57:1161-1166

## Reply:

We appreciate Dr. von Kummer's interest and meticulous efforts in the data reevaluation concerning our article (1). In our opinion, the presumed discrepancies noted by Dr. von Kummer are mainly due to misinterpretation of the presented data.

Dr. von Kummer argues that hypoattenuation of the lentiform nucleus was visible on the native CT scans in Figure 2B and C. This disagreement emphasizes that the detection of early ischemic signs on acute CT scans is subject to high interobserver disagreement. As Dr. von Kummer has shown in his study, the interobserver agreement of six neuroradiologists in assessing early ischemic signs varies between 49% and 71% (2). This is in line with our own inter-rater reliability of 67%.

Dr. von Kummer argues that the sensitivity of perfusion CT is difficult to assess because of the missing

reference method. We used follow-up CT or MR imaging as a reference, assuming that an infarction revealed by a follow-up scan or image is associated with an initial perfusion deficit. The term *sensitivity rate* describes the frequency of abnormal findings on the time-to-peak maps, which was 93% (14 abnormal CT perfusion maps of 15 participants with hemispherical territorial infarct).

Regarding the second article mentioned by Dr. von Kummer (3), we point out that the aims of the two works were different. Whereas the *AJNR* article focused mainly on the feasibility of using time-to-peak mapping during initial and early follow-up CT, the *Archives of Neurology* article aimed to correlate multiple parameters, such as final hemispheric lesion area and total infarct volume calculated from follow-up CT scans, with the severity of neurologic symptoms at stroke onset (National Institutes of Health Stroke Scale score) and therapeutic efficacy.

Fourteen patients presented in the *AJNR* article were included in the study population (n = 22) presented in *Archives of Neurology*, as is noted on page 1162 of that article. Figure 2C of the *AJNR* article contains the initial CT scan and the initial time-to-peak map, whereas Figure A of the *Archives of Neurology* article shows, in addition, the follow-up CT scan after 24 hr and the corresponding follow-up CT parameter map.

Dr. von Kummer noticed some inconsistencies in the tables of the two studies that are explained as follows. Many of these discrepancies are due to rounding errors, such as in the case of age (68 years versus 67 years for patient 8 [patient 3]) or in the case of hemispheric lesion area on initial time-to-peak map (*AJNR*) versus NAP<sub>0</sub> (*Archives of Neurology*) values (33.5 versus 33.6 for patient 10 [patient 8], with 33.55 being the correct calculated value; 28.5 versus 28.7 for patient 18 [patient 5], with 28.67 being the calculated value). Some discrepancies are due to typing errors, such as the hemispheric lesion area time-to-peak follow-up values for patient 2 (29.8 versus 19.8), with 19.8 being the correct value.

The noted inconsistency of the hemispheric lesion area time-to-peak follow-up value (2.5) versus the nAP<sub>1</sub> value (0.4) in the case of patient 19 (patient 6) is due to hemorrhage. In the *AJNR* article, the area of hemorrhage was included in the evaluation of hemispheric lesion area on the time-to-peak map, whereas in the *Archives of Neurology* article, this area was excluded. There were also some differences in the quoted time delay between symptom onset and initial CT scan in both articles that reflect the difficulty to assess reliable estimations of symptom onset from patients and/or relatives by two different investigators (radiologist for *AJNR* and neurologist for *Archives of Neurology*).

Dr. von Kummer has also noted inconsistencies

concerning the value of hemispheric lesion area on the follow-up scan (*AJNR*) and the values for total infarct volume (*Archives of Neurology*). This comparison, however, is not valid as is made easily evident by carefully reading the Methods section. Total infarct volume was calculated from the follow-up CT study to address the question of how reliable a single-section approach (perfusion CT) mirrors the total infarct volume.

A direct comparison between the values listed in the column *HLA* on follow-up CT in Table 2 (*AJNR*) and the column *HLA<sub>F</sub>* in the table presented in *Archives of Neurology* is therefore not possible. With respect to putative discrepancies concerning the treatment, the therapeutic options presented in the *AJNR* article are less detailed, categorizing therapy into “thrombolysis,” “conservative” (subcutaneous low weight heparin, aspirin, ticlopidine, or clopidogrel), and “heparinization” (meaning IV heparin administration aiming to double the partial thromboplastin time). Thus, therapy in patients 4 and 6 in the *AJNR* article was classified as *conservative* but in the *Archives of Neurology* article was classified as “heparin” and “ticlopidine.”

Supposed errors regarding “Clinical diagnosis” in *AJNR* (Table 1) and “Doppler sonography” in *Archives of Neurology* are obviously due to the different information presented: territory of the infarction in *AJNR* and vessel status in *Archives of Neurology*.

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2. von Kummer R, Holle R, Gizyska U, et al. **Interobserver agreement in assessing early CT signs of middle cerebral artery infarction.** *AJNR Am J Neuroradiol* 1996;17:1743–1748
3. Rother J, Jonetz-Mentzel L, Fiala A, et al. **Hemodynamic assessment of acute stroke using dynamic single-slice computed tomographic perfusion imaging.** *Arch Neurol* 2000;57:1161–1166