AJNR

Segmental Agenesis of the Internal Carotid Artery Distal to the Posterior Communicating Artery Leading to the Definition of a New Embryologic Segment

Timo Krings and Pierre L. Lasjaunias

AJNR Am J Neuroradiol 2006, 27 (2) 246-247
http://www.ajnr.org/content/27/2/246.1

Collateral Circulation via the Ascending Pharyngeal Artery Arising from the Internal Carotid Artery

I read with interest the article by Chan et al.[1] They stated that there was a rare *congenital* anastomosis between the vertebral artery (VA) and internal carotid artery (ICA) with an absence of communication between the common carotid and cervical ICA. I, however, diagnose that there was an *acquired* occlusion of the ICA at its origin with a development of collateral circulation from the VA to the ICA via the ascending pharyngeal artery.

I am very interested in the diagnosis of the cerebral arterial variations.[2] It is well known that the ascending pharyngeal artery sometimes arises from the proximal ICA.[3] In the patient reported by Chan et al, the ascending pharyngeal artery was well visualized, but opacification of the ICA was faint and delayed. This suggests that there was not a direct anastomosis between the VA and ICA.

In patients with congenital absence of the ICA, the common carotid artery and the proximal external carotid artery are usually the same size. In the patient reported by Chan et al, the common carotid artery was definitely larger than the proximal external carotid artery. This fact suggests that there was an acquired occlusion of the ICA at its origin.

References

1. Chan PN, Yu SCH, Boet R, et al. **A rare congenital anastomosis between the vertebral artery and internal carotid artery**. *AJNR Am J Neuroradiol* 2003;24:1885–86
2. Uchino A, Sawada A, Takase Y, et al. **MR angiography of anomalous branches of the internal carotid artery.** *AJR Am J Roentgenol* 2003;181:1409–14
3. Teal JS, Rumbaugh CL, Segall HD, et al. **Anomalous branches of the internal carotid artery.** *Radiology* 1973;106:567–73

Akira Uchino
Department of Radiology
Saga Medical School
Saga, Japan

Reply:

We thank Dr Akira Uchino for his interest in our report and his thoughtful and reasoned letter. We have again examined our imaging findings and concluded that Dr Uchino's observations are probably correct:

1. Complete occlusion of the internal carotid artery at its origin. The location of internal carotid artery origin matches well with the site of common carotid artery on angiography.

2. There is no direct communication between vertebral artery and internal carotid artery. These vessels are connected by small collaterals, which likely arise from the ascending pharyngeal artery. This is in keeping with the slow flow in the internal carotid artery.

P.N. Chan and S.C.H. Yu
Prince of Wales Hospital
Department of Diagnostic Radiology and Organ Imaging
The Chinese University of Hong Kong
Shartin, NT, Hong Kong

Segmental Agenesis of the Internal Carotid Artery Distal to the Posterior Communicating Artery Leading to the Definition of a New Embryologic Segment

We read with interest Gailloud et al's "Segmental Agenesis of the Internal Carotid Artery Distal to the Posterior Communicating Artery Leading to the Definition of a New Embryologic Segment."[1] In the article, the authors describe a case of segmental agenesis of the internal carotid artery (ICA) distal to the origin of the posterior communicating artery (PcomA) that was well documented by angiography and surgical inspection secondary to an associated anterior cerebral artery aneurysm.

The ICA is constituted by a number of successive embryologically distinct segments, each of them located between embryonic arteries or their remnants. Each of these segments can be absent, representing as a focal agenesis. In the ICA developmental anatomy proposed by Lasjaunias and Santoyo-Vazquez,[2] the ICA ends with the bifurcation into a rostral branch (from which in adult life the ICA distal to the PcomA, the anterior choroidal [AchoA], the anterior cerebral [ACA], and the middle cerebral artery [MCA] are derived) and a caudal branch from which the PcomA, parts of the basilar artery (BA), and the posterior cerebral artery (PCA) are derived in later life. This concept is challenged by the authors taking their case of a segmental agenesis of the ICA distal to the PComA into account. They regard the caudal division of the ICA no longer as a terminal branch of the ICA but instead argue that the PcomA is simply another embryonic vessel bridging the anterior and posterior circulation, being the most cranial of the carotid-basilar anastomoses. From this perspective, a new segment (the eighth segment) of the ICA distal to the PComA has to be defined that ends with the bifurcation into MCA and ACA.

We, on the other hand, argue that the ICA terminates with the bifurcation into a caudal and rostral branch and that, therefore, no eighth segment of the ICA can be present as such. What, on first sight seems to be just a problem of nomenclature (ie, why can't we simply call the "rostral branch of the ICA" the "eighth segment of the ICA") is, when phylogeny and embryology are taken into account, a misnomer that creates a misunderstanding.

The MCA is a recent phylogenetic acquisition and must be considered as a collateral branch of the ACA.[3] The ACA and AchoA are phylogenetically old vessels, forerunners of which are present in fish. The MCA appears as late as in reptiles, though not as a single trunk but instead as a series of small anastomosed vessels arising from the olfactory artery as the forerunner of the ACA. It continues to evolve in mammals and primates to finally become the single stem that we know. In most species, the caudal branch supplies the posterior fossa.

When considering the embryology of the human ICA development, further contradictions against the hypothesis that the ICA ends with the bifurcation into MCA and ACA can be found. The embryonic period is characterized by the shaping of the rostral extremity of the neural tube and, with it, the simultaneous shaping of the arterial tree. At the end of the fifth week (the prechoroidal stage), the ICA ends with a rostral and a caudal division. The caudal branch reaches the cephalic end of the ipsilateral ventral neural artery to constitute the PcomA, the so-called P1 segment and the upper half of the BA. This leads to a regression of pre-existing transient carotid-basilar anastomoses—ie, the trigeminal and hypoglossal arteries.[4] The caudal division, therefore, supplies the diencephalon, the mes-, and metencephalon. The rostral division, on the other hand, may be called the telencephalic branch. It subdivides further into the ACA and AchoA that both encircle the neck of the telencephalic vessel and anastomose with each other to form a ring. Lateral branches of the AchoA will later become the telencephalic portion of the PCA (ie, the so-called P2, P3, and P4 segments). Similarly, lateral branches of the pericerebral network of the hemispheres supplied by the ACA will become, at the end of the choroidal stage (ie, during the seventh and eighth week) the future MCA. Because the MCA, therefore, has to be regarded from a morphogenetic point of view as a branch of the ACA, there cannot be a bifurcation of the ICA into MCA and ACA, because, if it was a bifurcation, both vessels should appear at the same time in phylogeny and embryology.

What is called the ICA bifurcation in the manuscript is, therefore, the termination of the rostral division of the ICA, whereas the termination of the ICA is in fact located at the level of the PcomA regardless of its size. No additional segment of the ICA distal to this point, named as such, can be envisaged.

Timo Krings and Pierre L. Lasjaunias
Service de Neuroradiologie Vasculaire Diagnostique et Thérapeutique
Centre Hospitalier Universitaire de Bicêtre
Le Kremlin-Bicêtre, France

References

1. Gailloud P, Clatterbuck RE, Fasel JHD, et al. **Segmental agenesis of the internal carotid artery distal to the posterior communicating artery leading to the definition of a new embryologic segment.** *AJNR Am J Neuroradiol* 2004;25:1189–93
2. Lasjaunias P, Santoyo-Vazquez A. **Segmental agenesis of the internal carotid artery: angiographic aspects with embryological discussion**. *Anat Clin* 1984;6:133–41
3. Lasjaunias P, Berenstein A, TerBrugge KG. *Surgical neuroangiography.* Part 1. *Clinical vascular anatomy and variations.* 2d ed. Berlin: Springer-Verlag; 2001
4. Padget DH. **The development of the cranial arteries in the human embryo**. *Contrib Embryol* 1948;32:205–62

Reply:

We are grateful to Krings and Lasjaunias for their comment. It gives us an opportunity to re-emphasize a point central to our recent publication describing a case of segmental agenesis of a portion of the internal carotid artery (ICA) located distal to the origin of the posterior communicating artery (PcomA).[1] In our report, the absence of the involved segment was unequivocally documented by angiography and direct surgical observation.

It is often assumed that the embryonic ICA originally bifurcates into a rostral branch and a caudal branch. Following this view, principally based on the outstanding work of Padget,[2] the rostral branch is the precursor of the anterior cerebral artery (ACA) and its secondary branches, including the middle cerebral artery (MCA) and the anterior choroidal artery (AchoA), while the caudal branch corresponds to the posterior cerebral artery (PCA), including a proximal portion that later becomes known as the PcomA. In their theory on the segmental development of the ICA, Lasjaunias and Santoyo-Vazquez[3] use this assumption to set the distal limit of the embryonic ICA segments at the PcomA, the adult equivalent of the embryonic caudal division. According to this perspective, as they state in their letter, no segment can be defined distal to the PcomA, because it would then belong to the rostral branch of the fetal ICA, not to the ICA per se.

More recent investigations, however, offer a convincing alternate developmental scenario for the distal ICA. Van Overbeeke et al[4] looked at the relative role of the PcomA and the proximal PCA (P1 segment) in the formation of the circle of Willis in human fetuses and infants from 12 to 60 weeks of age. These authors defined 3 patterns of blood supply to the PCA territory: (1) an adult configuration, in which the P1 segment is the dominant source of blood supply, (2) a transitional configuration, in which the P1 segment and the PcomA are equivalent in size, and (3), a fetal or embryonic configuration, in which the PcomA is the dominant source of blood. Note that the name given to the 3 configurations is derived from the developmental pattern expected from Padget's conception of the distal ICA development—ie, a progressive transfer of the blood supply for the PCA territory from the ICA (fetal or embryonic configuration) to the basilar artery (BA; adult configuration), with an intermediate state of equivalent contribution (transitional configuration). The actual observations made by Van Overbeeke et al showed, however, a different pattern of development, with a largely dominant transitional configuration during the early fetal stages, which then progressively regresses in favor of either the adult or fetal/embryonic configurations. These findings strongly suggest that, in fact, the so-called caudal branch of the fetal ICA behaves as a carotid-basilar anatomosis, not as a dominant branch later annexed by the posterior circulation. In a superb publication dealing with the development of the PCA in the rat, Moffat[5] had already shown that the future territory of the PCA was, in the embryo, vascularized by the AchoA and not by the PcomA as initially believed, and later transferred to the cephalic end of the ipsilateral longitudinal neural artery (the future BA). This finding again suggests that the PcomA is, indeed, but the most cranial of the carotid-basilar anastomoses. If the concept of the ICA bifurcation into cranial and caudal branches is rejected, the assumption that the ICA terminates at the PcomA level becomes arbitrary, as does the rebuttal of a new embryonic segment based solely on that assumption.

We agree with Krings and Lasjaunias that the new segment documented in our publication is not located between the PcomA and the fetal ICA termination. Most likely, the distal end of this new segment is the AchoA, which is, as mentioned, a phylogenetically older vessel that plays a prominent role in the early development of the PCA. Unfortunately, the AchoA was visible neither angiographically nor during surgical exploration in our patient.

In summary, we have documented a segmental agenesis involving a previously unrecognized segment located distal to the origin of the PcomA.[1] This anatomic and angiographic fact does not fit in Lasjaunias and Santoyo-Vazquez theory of segmental development of the ICA as presently stated, but we think that it can be used, when combined with modern insight of the distal ICA development, to complement this theory.

Philippe Gailloud and Kieran J Murphy
Division of Interventional Neuroradiology
Johns Hopkins Hospital
Baltimore, Md

References

1. Gailloud P, Clatterbuck RE, Fasel JH, et al. **Segmental agenesis of the internal carotid artery distal to the posterior communicating artery leading to the definition of a new embryologic segment.** *AJNR Am J Neuroradiol* 2004;25:1189–93
2. Padget DH. **The development of the cranial arteries in the human embryo.** *Contr Embryol* 1948;212:205–61
3. Lasjaunias P, Santoyo-Vazquez A. **Segmental agenesis of the internal carotid artery: angiographic aspects with embryological discussion.** *Anat Clin* 1984;6:133–41
4. Van Overbeeke JJ, Hillen B, Tulleken CA. **A comparative study of the circle of Willis in fetal and adult life: the configuration of the posterior bifurcation of the posterior communicating artery.** *J Anat* 1991;176:45–54
5. Moffat DB. **The development of the posterior cerebral artery.** *J Anat* 1961;95:485–94

Proper Masking to Show the True Activation

A recent article by Strigel et al addressed a very important issue facing clinical functional MR imaging (fMRI): how confident of an activation map can one be, in light of the different susceptibility issues in clinical fMRI?[1] The authors touch upon only the tip of the iceberg as they present an ad hoc method for demonstrating confidence in the activation map by calculating a signal intensity mask (SIM).[1] The main problem with this approach is that it is independent of the blood oxygen-level dependent (BOLD) signal intensity change and would incorrectly create the same mask if the change were 0.5% or 5%. The criterion used to generate the threshold is described as "thresholded to eliminate signal intensity from regions outside the brain." This is problematic because the tissues outside the brain are for the most part

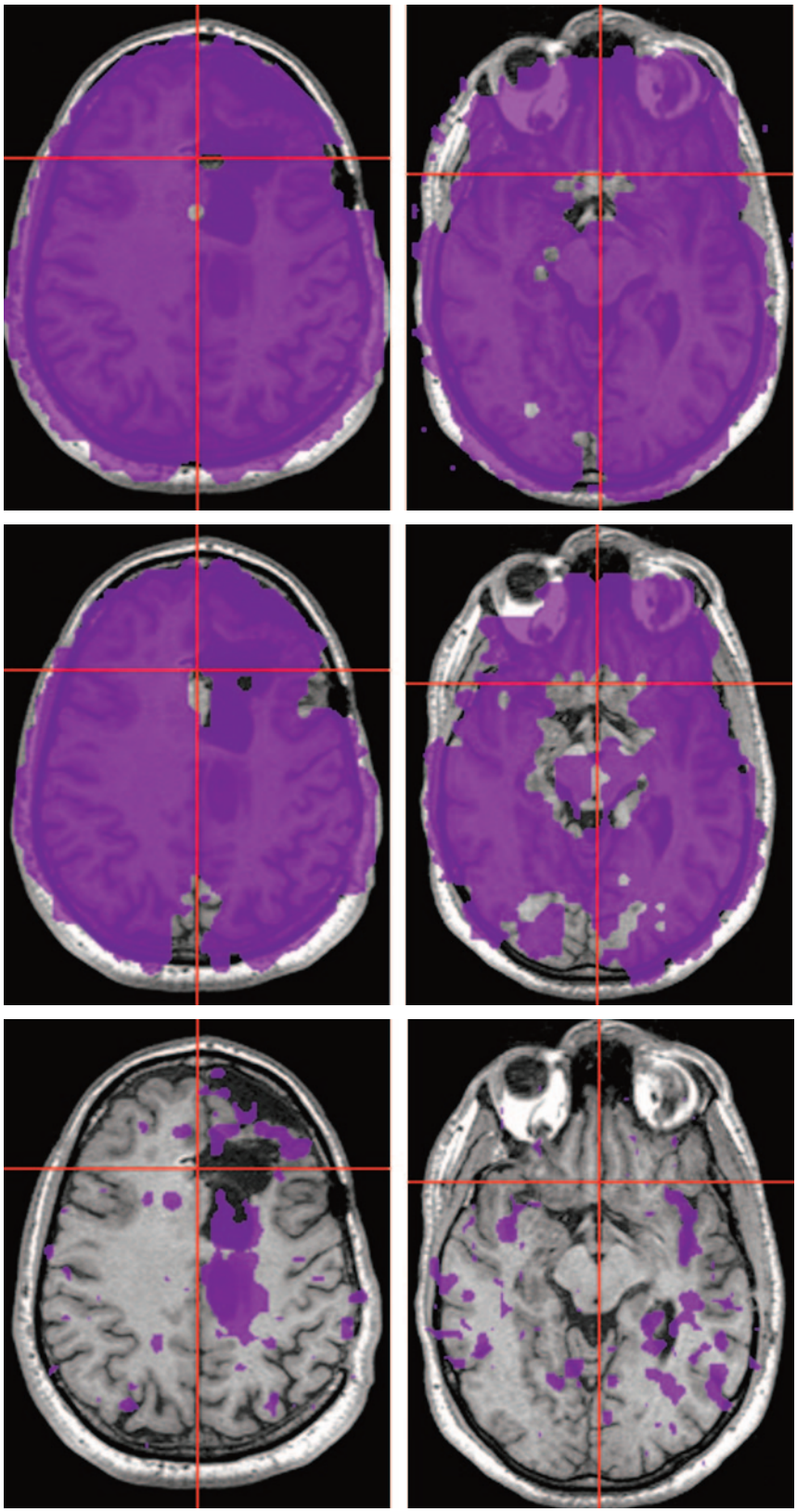

Fig 1. The color overlays represent regions that have sufficient levels of confidence to interpret the brain activation map. The 3 different rows represent different methods and conditions. The first row shows the SIM method based solely on the static image intensity. The second and third rows are based on a statistical model, BOLD signal intensity change and the temporal SNR. The second row indicates where it is possible to detect a 1% or greater BOLD signal intensity change. The third row represents where a 0.5% or greater BOLD signal intensity change can be detected. Smaller BOLD changes are likely to take place in clinical patients because of abnormal physiology and poor performance because of the presence of a lesion. It is clear that the temporal stability of these data are not sufficient to detect small BOLD changes.

skull, scalp, and muscle. Muscle has similar relaxation parameters as the brain, so yielding a threshold near brain intensity. With multichannel array coils becoming more mainstream, the images have significantly higher signal intensity near the surface coils, making the SIM threshold artificially high, and may even cause voids in the center of the image. The SIM threshold will vary for each subject according to his or her anatomy and positioning in the coil. The SIM threshold will change if the service engineer makes an adjustment or upgrades the scanner software, altering the image intensity scale. The use of an intensity-based threshold, as suggested, may give a false sense of confidence.

The authors describe the method as "the initial EPIs" (echo-planar images) were used to generate the SIM.[1] This points to an even more fundamental problem. Functional imaging is based on detecting small signal intensity changes over time. Having a high signal intensity–to-noise ratio (SNR) in a single image is not sufficient to detect small signal intensity changes over time. The stability of the signal intensity over time is more important than the absolute level of the signal intensity. One needs to use temporal SNR, the signal intensity to noise calculated over the entire time course, as the basis of an activation map threshold.[2] By using the entire time series data, the method of screening the activation map is now sensitive to susceptibility signal intensity loss, spike artifacts, scanner instabilities (radiofrequency, gradient, and B0), and movement artifacts. The latter is critical around susceptibility-induced signal intensity voids, where small movements could mimic large signal intensity changes.

In 2000, I proposed a method that described the temporal SNR map and a method to threshold it on the basis of the imaging parameters, the desired confidence levels, and a computer model.[2] In that report, the idea of a BOLD sensitivity map independent of field strength, coil used, or signal intensity level was introduced. On the basis of this method and an expected BOLD signal intensity change of 0.5% would require a minimum temporal SNR of 164 in an experiment with 80 volumes (10 on/10 off, repeated 4 times), a type I error of $\alpha = 0.05\%$ and a power level of $\beta = 0.95$. The required minimum SNR is the same for any subject, does not change based on scanner manufacturer, coil used, or field strength. The results are scalable to meet any type of fMRI protocol. For example, if one

changed the level of BOLD signal intensity expected to 1%, the required minimum SNR decreases by a factor of 2, to 82. In a separate publication, we also showed how the BOLD sensitivity maps could be used to determine if the actual measured BOLD signal intensity change was detectable in the amygdala.[3]

What is the practical implication for real fMRI data? In Fig 1, 2 different anatomic levels of a postsurgical fMRI patient study are shown. In the first row, the mask was generated by the SIM method[1] by setting the threshold so that the tissue surrounding the brain in the raw BOLD EPI data was suppressed; signal intensity was 240. In the second row, the mask was generated by the SNR-based method,[2] with the parameters described above and an expected BOLD signal intensity change of 1% (SNR $>$ 82). Note the large differences in the mask in the region where the sinus susceptibility artifact exists, as well as near the surgical site. The third row demonstrates a very different mask based on a 0.5% BOLD signal intensity change (SNR $>$ 164). The lower level of BOLD change may be expected in patients with disease. The lower 2 rows are based on SNR, statistical confidence, and BOLD signal intensity changes, whereas the first row is based on the SIM, a number that has very little meaning.[1]

I am encouraged that the authors are concerned about the impact of image quality, artifacts, and signal intensity voids on the interpretation of clinical fMRI and have done some excellent work to illuminate this problem. We should, however, proceed carefully when developing a method to demonstrate confidence in the activation maps. Using an arbitrary method may "mask" the clinical utility of BOLD imaging.

Todd Parrish
Department of Radiology
Northwestern University Medical School
Chicago, Ill

References

1. Strigel RM, Moritz CH, Haughton VM, et al. **Evaluation of a signal intensity mask in the interpretation of functional MR imaging activation maps.** *AJNR Am J Neuroradiol* 2005;26:578–84
2. Parrish TB, Gitelman DR, LaBar KS, et al. **Impact of signal-to-noise on functional MRI.** *Magn Res Med* 2000;44:925–32
3. LaBar KS, Gitelman DR, Mesulam MM, et al. **Impact of signal to noise on functional MRI of the human amygdala.** *Neuroreport* 2001;12:3461–64

Reply:

We thank Dr. Parrish for his comments on the relationship of susceptibility and signal intensity–to-noise ratio (SNR) for confidence levels in clinical functional MR imaging (fMRI). We welcome the discussion of these issues and laud him for his comprehensive investigation of the effects of temporal SNR on blood oxygen-level dependent (BOLD) time course analyses.[1]

The statements and example of a signal intensity map (SIM) that Parrish includes in his letter, however, do not match our experience. In our study, each SIM threshold was individually matched to the patient's echo-planar imaging (EPI) data, thus eliminating the possibility for errors incurred by use of an arbitrary threshold applied across all datasets.[2] In our experience, as demonstrated by the examples for SIM formation in Figs 1–3 of our article, SIM is sensitive to regions of signal intensity loss produced by magnetic susceptibility effects when conventional echo-planar BOLD imaging is used. In all of our cases, EPI susceptibility effects in regions of frontal and basilar sinuses were delineated by the SIM. The intent of our report was to evaluate the SIM as an indication of susceptibility-induced artifact upon the interpretation of clinical fMRI mapping. These susceptibility-induced artifacts are substantially stable during the course of a fMRI time series acquisition. Therefore, within this limited assessment, the static SIM provides an adequate means for evaluation. A version of the SIM is relatively easy to produce on a clinical system and thus offers widespread utility to fMRI users.

Parrish et al[1] have applied the temporal nature of the fMRI acquisition to further evaluation of BOLD sensitivity. We appreciate the importance of their report and encourage fMRI users to become familiar with the significance of their findings. Temporal SNR measurements provide information about the BOLD signal intensity stability that is not contained within a static SIM, and indeed it is our practice to produce both types of signal intensity evaluation maps for our fMRI studies.

We regret any misunderstanding that might have led Dr. Parrish to question our report on the utility of a SIM. We are gratified by the forum for discussion of these issues, particularly when the opportunity leads toward increased awareness of limitations and capabilities for clinical fMRI.

Chad Moritz
Roberta Strigel
Howard Rowley
Victor Haughton
Department of Radiology
University of Wisconsin
Madison, Wisc

References

1. Parrish TB, Gitelman DR, LaBar KS, et al. **Impact of signal-to-noise on functional MRI.** *Magn Reson Med* 2000;44:925–32
2. Strigel RM, Moritz CH, Haughton VM, et al. **Evaluation of a signal intensity mask in the interpretation of functional MR imaging activation maps.** *AJNR Am J Neuroradiol* 2005;26:578–84

Erratum

Due to a translation error, Chung Hwan Baek's name was misspelled in the published list of authors for the article "Nodular Fasciitis in the Head and Neck: CT and MR Imaging Findings" in the November/December 2005 issue. The correct author list should be:

Sung Tae Kim, Hyung-Jin Kim, Sun-Won Park, Chung Hwan Baek, Hong Sik Byun, and Young Mo Kim. (*AJNR Am J Neuroradiol* 2005; 26:2617–23.)