Radiol Oncol 2000; 34(2): 123-36.

review

Computed tomographic angiography in intracranial vascular diseases

Zoran Miloševič

Clinical Radiology Instutute, University Medical Centre Ljubljana, Slovenia.

Background. *The development of spiral computed tomography (CT) introduced a more precise imaging of the vessels also with computed tomographic angiography (CTA). Because it is a minimaly invasive method, it was widely accepted by radiologists and clinicians. In early 90 ties CTA also accompanied conventional angiography and magnetic resonance angiography (MRA) in imaging of intracranial vascular diseases. CTA is used for the detection and evaluation of intracranial aneurysms, vascular malformations, stenoocclusive diseases of intracranial arteries and pathological changes of venous sinuses. Comparing to conventional angiography as the »gold standard«, CTA has high specificity, sensibility and diagnostic accuracy concerning detections of intracranial aneurysms. Regarding vascular malformations, CTA is used for diagnostics and pre and postoperative evaluation of it. CTA can show good results in imaging of venous angiomas, and so invasive conventional angiography can be avoided in this pathology. Stenoses and occlusions of arteries can be diagnosed and evaluated in patients with cerebral vasospasm, patients with acute stroke, and patients with chronical arterial stenoses and occlusions. CTA is useful for the demonstration of occlusive and stenosing changes of intracranial venous sinuses.*

Conclusion. *With CTA it is possible to generate threedimensional reconstructed images which give a more accurate determination of anatomical relations in intracranial vascular diseases. The main disadventage of CTA in comparison to intraarterial angiography is the lower spatial resolution of CTA, but is constantly improving with the developement of better scanners and workstations, so that there are great possibilities for further developement and wider use of CTA in the diagnosis of intracranial vascular diseases.*

Key words: tomography, x-ray computed; cerebral artery diseases-diagnosis; cerebral aneurysm-diagnosis; angiography

Received 15 May 2000

Accepted 31 May 2000

Correspondence to: Zoran Miloševič, M.D., Clinical Radiology Instutute, University Medical Centre Ljubljana, Zaloška 2, SI-1525 Ljubljana, Slovenia. Phone: +386 61 143 1530; Fax: +386 61 133 1044; E-mail: zoran.milosevic@moj.net

Introduction

Since its introduction in clinical practice in early 1970s, computed tomography has gone through a lot of important refinements and became more accurate and much faster from its beginnings till today. A progresive reduction in scan times and improved spatial and contrast resolution made CT imaging a workhorse for many years. In early 1990s computed tomography has been revolutionazed by technical advantages of spiral CT. Spiral CT scanning involves continous data aqusition throughout the volume of interest by simultaneous moving of the patient through the gantry while X-ray sources rotate.[1,2] As this proces is contiuonos rather than stepwise as in conventional CT scaning, the examination time is reduced. Besides advantages like increased patients throughput and reduction of motion artifacts, the spiral CT also offers additional properties which are not possible with conventional step by step CT scanning. Because of short acquisition time, scanning can be timed with the peak opacification of arterial or venous phase, after the peripheral intravenous application of contrast media. The resultant images (raw data) are processed with various computed rendering techniques, such as multiplanar reformatting (MPR), shaded surface display (SSD), maximum intensity projection (MIP) and volume rendering technique (VRT) to generate two or three-dimensional images of the vessels. As a result, CT angiography is performed less invasively, faster and at a lower cost than conventional intraarterial angiography.[3,4]

In neuroradiology, the diagnostics of cerebrovascular diseases represents one of its major fields of activity. It has progressed a lot during the last decade with the advent of MR imaging and spiral computed tomographic tehnology. CTA was increasingly used for the detection and evaluation of intracranial aneurysms, intracranial vascular malformations, intracranial vascular stenoses and occlusions and pathological changes on intracranial venous sinuses.

The purpose of this paper is to review the value of CTA in detection and evaluation of vascular intracranial diseases.

Intracranial aneurysms

Aneurysms are circumscribed dilatations of arteries that communicate directly with the vessel lumen. They may be saccular (berry) or fusiform.[5]

Intracranial saccular aneurysms

Saccular aneurysms are found in 1 % to 5,6 %.of population.[5,6] 15 % to 20 % of patients have multiple aneurysms.[7] Saccular aneurysms are an important part of vascular patology, because subarachnoid haemorhage (SAH) is in 80 %-90 % caused by a rupture of a saccular aneurysm. In 15 % SAH may be caused by the arteriovenous malformation, and 5 % by diverse causes.[8] SAH resulting from a ruptured aneurysm of intracranial arteries carries a poor prognosis and the mortality in untreated patients may be as high as 45 %.[8]

For many years the intraarterial cerebral angiography has been a technique of choice for the demonstration of the intracranial aneurysms, but it is invasive, expensive and has 1 % of complications, while 0,5 % of them develop permanent neurological deficits.[9,10] Therefore non-invasive MRA and minimaly invasive CTA have been increasingly used over the past few years.[11,12] Although MRA is capable of showing an accurate anatomy of intracranial vessels and vascular patology, there are some difficulties in detection and demonstraton of aneurysms with turbulent or slow blood flow.[13-17] MRA is contraindicated in patients with feromagnetic clips, pacemakers or life-support devices.[18]

CTA is insensitive to turbulent blood flow artifacts and in contrary to MRA, it can be performed in patients with ferromagnetic implants.[19-21] In patients with SAH CTA demands little additional time and is easily performed immediately after the conventional CT.[22] CTA is highly accurate, sensitive and specific as compared to DSA (gold standard).[20,23-27]

Because of its minimal invasiveness, the indications for CTA in diagnostics of cerebral aneurysms are broader than for DSA and can be divided into six groups:

1. *Patients with acute SAH.* The patients are usually criticaly ill and clinically unstable. On time diagnosis of the etiology of bleedeng is essential for planning an early surgery or other intervention. Intraarterial conventional angiography performed within the first 6 hours after initial bleeding is associated with an increased rebleedeng rate.[28] CTA is very suitable in the acute stage after SAH because it does not require intraarterial catheterization, scaning time is only 50 seconds and it can be performed on the same scanner immediatly after the demonstration of SAH by conventional CT scan.[23-25,29]
2. *Patients with proved history of SAH, but with negative or indeterminate first angiography.* In a number of patients, no underlying cause of SAH is identified despite a complete neuroradiological investigation. In the literature this proportion varies between 3,8 % [30] and 46 % [31] with accepted mean of 15 %.[32] The etiology of angiogram-negative SAH remains elusive. Numerous theories have been proposed. One theory postulated that SAH may be due to leakage from the lenticulostriate and thalamoperforating vessels.[33] Another theory suggested a venous or capillary source for the patients with perimesencephalic SAH.[34] The most popular theory attributes bleeding to an aneurysm that undergoes thrombosis or is destroyed at the time of haemorrhage.[35] CTA performed about 3 weeks later, can demonstrate partially or completely recanalized aneurysm.
3. *Patients with a suggestive but uncertain history of SAH.* In these patients, instead of invasive DSA as first imaging modality, CTA or MRA can often make the diagnosis.[36,37] For example, we found an aneurysm at the bifurcation of the left middle cerebral artery in a 37 years old woman who suffered from a sudden strong headache two weeks before and did not seek medical help at that time (Figure 1).
4. *Patients without SAH, but with suspicious clinical signs of an intracranial aneurysm or an aneurysm-like lesion on conventional CT images or MR images.* In this group we have an example of a 68 years old woman suffering from paresis of the right third nerve, where we detected a large right internal carotid artery aneurysm (Figure 2).
5. *Screening in »high risk population«.* The existence of families with a history of intracranial aneurysms is well recognized. In large epidemiologic studies, the prevalence of familial intracranial aneurysms is higher than in general population (7% to 9%).[38-44] Even in the situation of sporadic case , relatives of the patients are often worried that they may also harbor an aneurysm. In screening we use CTA as an additional or alternative method to MRA in patients with a family history of aneurysmal disease and patients with predisposing hereditary disease, such as autosomal polycystic renal disease.
6. *Follow up of treated or non-treated aneurysms.* Once the aneurysm is detected and clipped, a question may arise as to the proper placement of the clip. One potential problem is to only partially clip the neck of the aneurysm and allow continued filling of the aneurysm. Another is a possible occlusion of vital arteries after the improper placement. The postoperative evaluation has traditionaly been done with a conventional angiography.[45] In a few cases we have had used CTA in order to clarify such

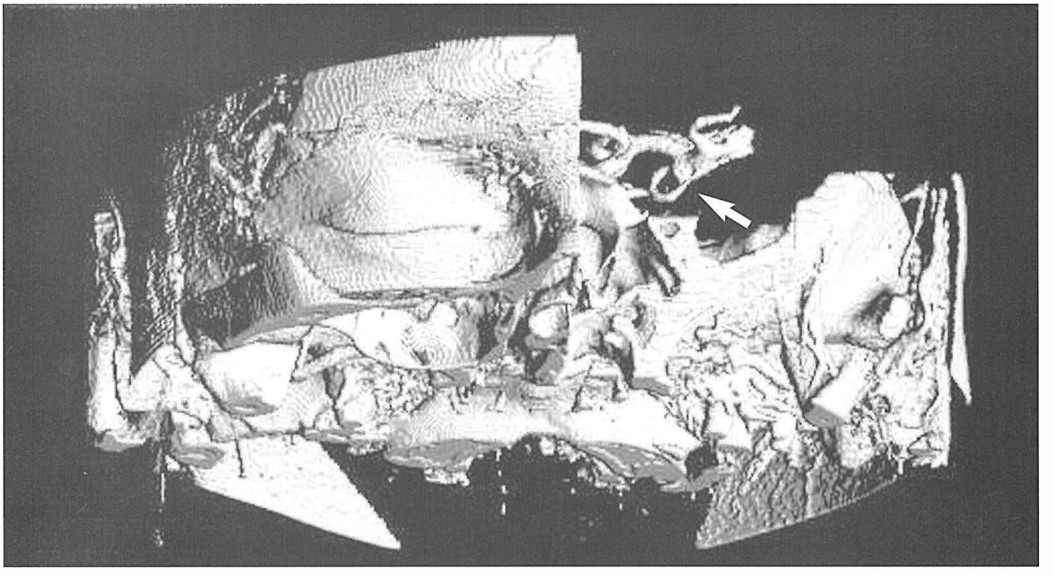

Figure 1. CT angiogram, volume rendering technique, anteroposterior view, demonstrates an aneurysm at the bifurcation of the middle cerebral artery and its relationship to the middle cerebral artery branches (arrow).

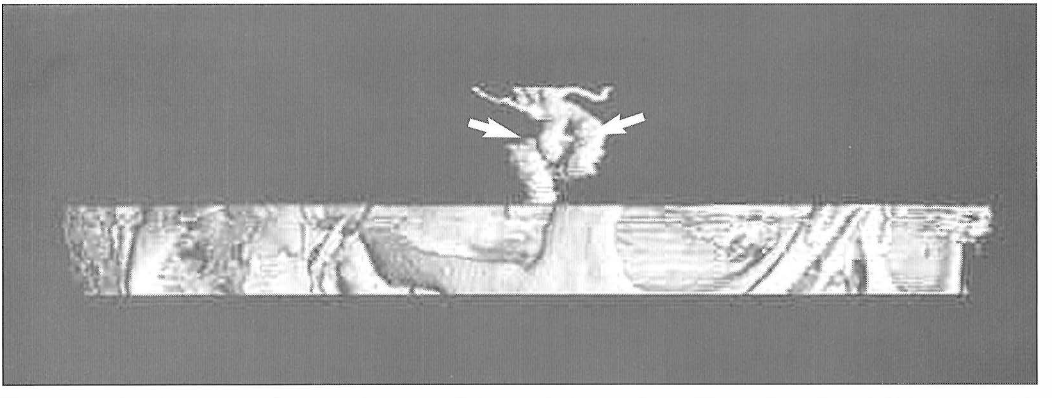

Figure 2. CT angiogram, volume rendering technique, right anterior oblique view, demonstrates a longish aneurysm coming out from the right internal carotid artery and spreading backwards and downwards in the area of the third nerve (arrows).

dilemmas. Depending on the size and orientation of the clip, a starshaped artifact in the immediate vicinity of the clip is seen. In most cases we have been able to demonstrate both clip and eventualy residual aneurism as well as patency of vessels despite this artifact (Figure 3). Inspite of this, CTA in its present form cannot replace DSA in all situations of the evaluation of the aneurysm clip placement.[46]

Finally, since CTA and DSA are in most cases complementary examinations, their combination often provides more data in the preoperative evaluation of intracranial saccular aneurysms than obtained with each of them separately. CTA could be considered useful tehnique in the preoperative evaluation due to their three-dimensional representation of outer and inner vessel surfaces. The so called endovascular view of both the neck and

Figure 3. CT angiogram, volume rendering technique, superior view, performed after basilar artery aneurysm cliping, demonstrates the clip (arrows), patent basilar artery and its branches (arrowheads) and no residual aneurysm.

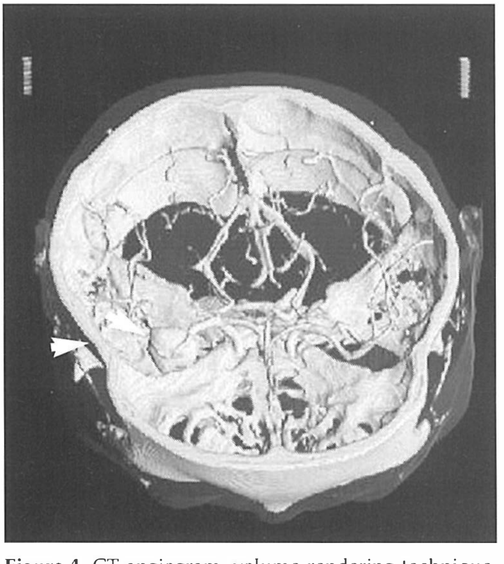

Figure 4. CT angiogram, volume rendering technique, anterosuperior view, demonstrates a large right middle cerebral artery aneurysm. The relationship of this aneurysm to the inner table of the skull is well shown (arrows).

sack of the aneurysm can demonstrate the relationship between the aneurysm and arterial branches.[47,48] CTA also allows the display of adjacent bone structures (Figure 4) and allow surgeons to plan a craniotomy with the best approach to the neck of the aneurysm.[26]

Intracranial fusiform aneurysms

Fusiform aneurysms are dilated and elongated atherosclerotic vessels. They commonly affect the supraclinoid segment of internal carotid artery and vertebrobasilar arteries. Mural thrombus is common. Hemorrhage is rare.[5] Surgical therapy is not possible in the majority of cases. CTA is able to clearly demonstrate this type of aneurysm and so to avoid DSA (Figure 5).

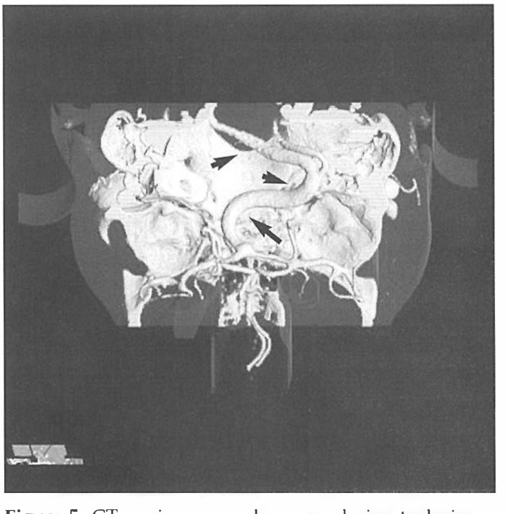

Figure 5. CT angiogram, volume rendering technique, posterosuperior view, demonstrates fusiformly dilated and elongated right vertebral artery and basilar artery (arrows). On the basis of this examination, it was decided that the patient was not an operative candidate. DSA was avioded in this case.

Intracranial vascular malformations

Intracranial vascular malformations are a diverse group of congenital lesions of blood vessels. These lesions are usually classified as arteriovenous malformations (AVM), venous angiomas, cavernous angiomas and capilary teleangiectasias.[5]

Intracranial arteriovenous malformations

Patologically, the AVMs show clusters of abnormal arteries and veins. The vessel walls are typically thickened and contain elastin and smooth muscle. AVMs are subdivided into pial AVMs, dural AVMs and dural arteriovenous fistulas.

Pial AVMs consist of an plexus of arterial feeders, nidus and dilated draining veins. Because there is no intervening capillars, blood shunts directly from arteries to veins. These vessels are patological and are prone to rupture. The risk of hemorrhage is 2 % to 4 % per year. For each episode of hemorrhage there is a 29 % chance of death and 23 % chance of long term morbidity.[49] The therapy of AVMs can be surgical, radiosurgical or endovascular.

A pre-therapeutical neuroradiological evaluation requires a diversity of anatomical and hemodynamic information. From the morphological point of view, neuroradiological studies identify feeding arteries and draining veins and evaluate angioarchitecture of the nidus. From hemodynamic point of view, flow velocities in the different vascular compartments shoud be evaluated. Conventional angiography still represents the gold standard for evaluating feeding arteries, draining veins and the angioarhitecture of the nidus. It is also mandatory for hemodinamic evaluation of AVMs. The experience in the last few years showed that MRA and CTA can be useful in the diagnostics of AVMs.[50-52]

CTA can have important role in the following situations:

1. *Detection of AVM.* CTA can be useful in a diagnosis, excluding or confirming the presence of AVM in a sugestive clinical context.
2. *Pre-therapeutical evaluation of AVM.* In conjunction with conventional angiography, conventional MR and CT images, CTA can be used to obtain three-dimensional images of AVM. Reconstructed three-dimensional CTA images can be viewed from any perspective which can be used for an exact localisation of feeding arteries, nidus and draining veins (Figure 6).

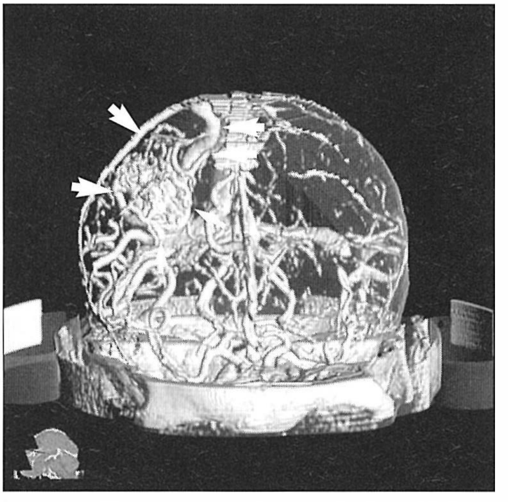

Figure 6. CT angiogram, volume rendering technique, anteroposterior view, shows pial arteriovenous malformation of the right hemisphere. Two main feeding arteries (arrows), nidus (arrowheads) and draining vein (open arrow) are well demonstrated.

3. *Post-therapeutical evaluation of AVM.* The analysis of AVM reduction after the treatment can be performed with CTA images. This technique offers a suitable method for a minimally invasive and reproducible follow up.

In dural AVMs and dural arteriovenous fistulas neither CTA nor conventional MR or MRA can substitute or complement a conventional angiography in a diagnosis and pre-treatment evaluation.[27,53,54]

Venous angiomas

Venous angiomas are congenital anomalies of the intracranial venous drainage. They represent anatomic variants resulting from the arrested embryological venous development causing the persistence of primitive medulary veins.[55] They are described as a local network of small, medulary veins, resembling so called caput medusae, which converge centrally into a large transmedullary vein that courses toward the cortical surface or the deep venous system.[56,57] Venous angiomas are most common incidental vascular malformations detected radiologically and at autopsy.[58] They are also designed »developmental venous anomalies« to emphasize their frequency and their benign nature. Usually they cause no symptoms but may rarely be associated with headache, seizure, or focal neurologic deficit [58,59] and even more rarely with acute symtomatic hemorrhage.[60] In most cases a surgical therapy is not necessary or possible.[58-60]

Today the technique of choice in the neuroradiological diagnosis of venous angiomas is MRA and conventional MRI with the administration of gadolinium. A stellate appearence on MR is said to be patognomonic of venous angiomas.[61] Only in rare circumstances, when the diagnosis is not certain, a conventional angiography needs to be performed. With the conventional angiography, a venous phase abnormality characterized by multiple dilated medulary veins converging on a larger trancortical vein, giving a »caput medusa« appearance is diagnostic.[58-61]

Instead the conventional angiography, CTA can also be used.[62] CTA demonstrates findings characteristic for venous angiomas: small vascular structures in deep white matter converging to a more dilated trancortical draining vein (Figure 7). The use of CTA in the diagnosis of venous angiomas shows good preliminary results and could obviate the need for the conventional angiography in most cases.

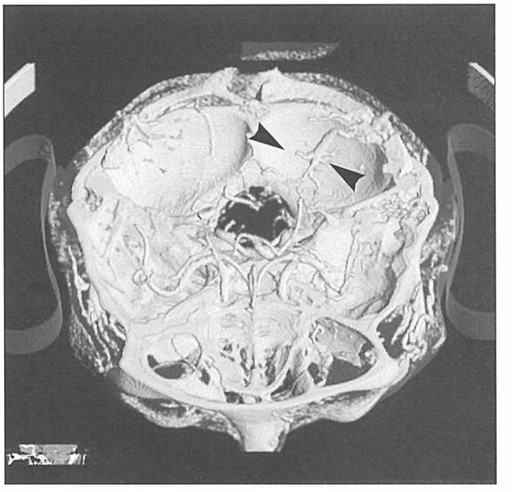

Figure 7. CT angiogram, volume rendering technique, anterosuperior view, shows venous angioma in left cerebelar hemisphere (arrows). The lesion has characteristic »caput medusa« configuration.

Cavernous angiomas and capillary telangiectasias

In cavernous angiomas and capillary talengiectasias, both conventional and non or minimaly invasive angiographic techniques fail to revel the majority of lesions, whereas a conventional MRI still remains the technique of choice.[5,27]

Intracranial vascular stenoses and occlusions

The intracranial stenotic and the occlusive vascular patology can be divided in cerebral vasospasm in patients with SAH, acute arterial occlusion in patients with acute ishemic stroke and chronic stenoticocclusive diaseases of arteries.

Cerebral vasospasm

The most debilating complication of acute SAH is the cerebral vasospasm, acounting for 14 % of deaths or severe disability in patients with SAH.[63] The onset of spasm occurs 4 to 11 days after hemorrhage in approximately

30 % of patients.[64] A current therapy includes the hypervolemic and pharmacologic therapy and its efficacy is well documented.[64]

A conventional angiography is one diagnostic method for this complication, but the risk of performing this procedure in the critically ill patient can limit its application. Transcranial Doppler sonography is noninvasive and rapidly performed, but does not provide anatomic information and is limited to a small acoustic window.[65,66] MR angiography is restricted in the evaluation of these patients due to a reduced intracranial blood flow.

CTA offers the potential for rapid, minimally invasive method of diagnosing and monitoring this complication (Figure 8).[67]

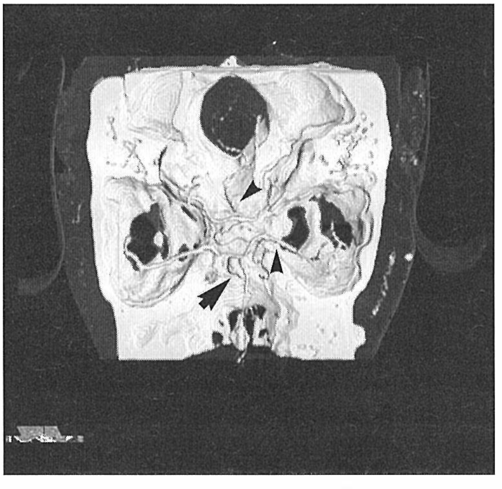

Figure 8. CT angiogram, volume rendering technique, superior view, shows vasospasm of intracranial arteries (arrowheads) after subarachnoidal haemorhage and an anterior communicating aneurysm (arrow).

Acute ischemic stroke.

Strokes are a major public health problem. Stroke is the third most common cause of death since one third of the patients die and another third are rendered permanently disabled.[68] In Slovenia the incidence of stroke is 190,5/100000 people. Mortality in the first 30 days is 21 %.[69] Ischemic infarction of brain tissue, because of the acute arterial occlusion, is the major causative factor. The majority of infarctions are caused by thrombembolism from underlying atherosclerotic disease.[70,71] The majority of stroke patients are treated conservatively.[72] Systemic intravenous or local intraarterial thrombolysis has recently shown the promise of improving the patient's outcome.[73,74] However, thrombosis must be identified and treated promptly for optimal results. Because trombolytic drugs produce intracranial hemorrhage in 6 % to 20 % of cases, the potential for salvaging the ischemic brain must be defined.[74,75] The reversibility of ischemic process not only depends on the time after ictus, but is primarily a function of the degree of persistent collateral flow to the affected tissues. Brain without sufficient collateral flow will die within minutes, whereas tissue with good collateral flow will remain viable. In the latter circumstances thrombolytic terapy can be effective.

Patients with acute stroke are examined with unenhanced CT of the brain to exclude intracranial hemorrhage or other rare causes for stroke. CT is also useful in assessing early signs of cerebral ishemia, such as parenhimal hypodensity and focal brain swelling.[76,77] But conventional CT does not show the extent of disturbed cerebral perfusion, which is determined by the site of occlusion and collateral blood supply and is not capable of showing the volume of viable tissue at risk from the low perfusion, which is the target of thrombolytic treatment.[78] Recently, MRA and MR imaging with hemodynamic and diffusion weighted pulse sequences are increasingly used in patients with acute stroke. Diffusion and perfusion images are highly sensitive to early infarction and an extent of infarcted brain tissue.[79,80]

In acutely ill stroke patients CTA is more practical and faster than MR imaging and can be performed immediately after the conventional unenhanced CT of the brain. Because of these considerations, few authors studied whether CTA is capable of showing the site of arterial occlusion, estimating collateral circulation, and determining the extent of severe parenhimal perfuson deficit. Preliminary results of these studies showed that CTA is safe in cases of acute stroke and can add an important diagnostic information to those obtained by the conventional CT and may provide a rational basis for optimal treatments of patients with an acute stroke.[78,81,82]

Chronic stenoocclusive diseases

Chronic stenoocclusive diseases of intracranial arteries are most commonly caused by atherosclerosis,[5,83] less often by nonatheromatous causes, like fibromuscular dysplasia, vasculitides and idiopathic progressive arteriopathy (moyamoya).[5,84] Stenoocclusive diseases of intracranial arteries impeares the blood supply of the brain and increase the possibility of an ischaemic stroke. Early diagnosis and treatment of these pathologies has an important role in the stroke prevention.

In nonatherosclerotic stenoocclusive diseases a conventional angiography still plays a primary role, due to mainly changes on arteries of the second and the third order.[84] Spatial resolution of MRA and CTA is too low for precise imaging of these arteries, which measure less than 1 mm in diameter.[85]

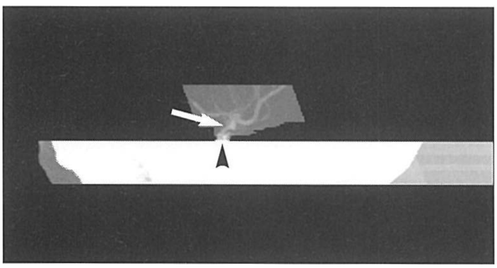

Figure 9. CT angiogram, maximum intensity projection, left anterior oblique view, demonstrates atherosclerotic stenosis of supraclinoid segment of left internal carotid artery (arrow) and extensive calcifications in this atherosclerotic lesion (arrowheads). Because of calcifications, angioplasty is contraindicated in this case.

Because atherosclerosis affects mostly larger intracranial arteries, like internal carotid artery, middle cerebral artery, basilar and vertebral arteries, useful and reliable diagnostic method is MRA[86,87] and nowadays also CTA.[88] CTA most reliably demonstrates calcifications in atherosclerotic lesion, which can have an important impact concerning further therapy (Figure 9).

Venous sinus compression and trombosis

The external compression of venous sinuses can cause their narrowing or obstruction. It is most commonly caused by the tumor or bone fragment with the impression fracture. Sinus thrombosis is a partial or complete obstruction of sinus lumen due to intraluminal clot and usually affects superior sagital sinus, then transversal, sigmoid and cavernous sinus.[5] Thrombosis can spread into cortical veins, straight sinus and internal cerebral veins. The interruption of venous outflow can cause local or diffuse cerbral edema and cortical venous infarcions, which are often haemorrhagical.[89]

In the past, the prognosis in patients with venous thrombosis has been poor, with the mortality rate between 30% and 80%, but has been improved in the later years by the effective systemic heparin anticoagulation, fibrinolytic therapy and anti-edema therapy.[90,91] The availability of a successful treatment has increased the need for the prompt and accurate diagnosis.

Besides conventional angiography, conventional CT,[92] MR and MRA[91,93,94] have increased the ability to detect this condition. The conventional contrast enhanced CT has low sensibilitiy in the diagnosis of dural sinus thrombosis.[95] MRA, the present examination of the choice for evaluation of dural sinuses, is limited by motion artifacts and the patient's contraindications.[92] Recently developed CTA with another name CT venography offers grea-

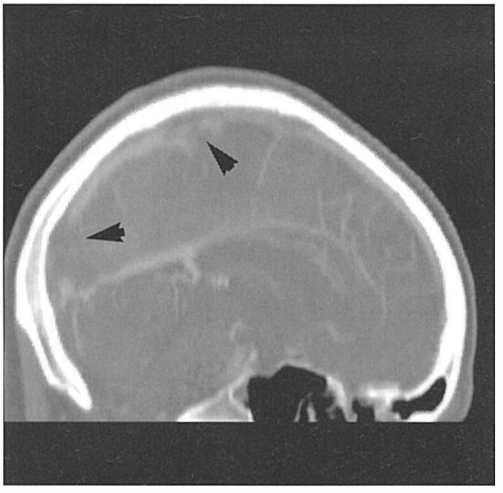

Figure 10. CT angiogram, multiplanar reconstruction, sagital reconstruction, shows absence of opacification in posterior and middle portion of superior sagittal sinus because of thrombosis (arrows).

ter sensitivity and specificity than a routine contrast-enhanced CT in the diagnosis of dural sinus thrombosis.[92,96] On CT venography, dural sinus thrombosis is seen as the absence of opacification of the affected dural sinus on the reconstructed images (Figure 10) and as a filling defect in the dural sinus on the source images.[92,96] Also, in cases of external venous compresion, CTA can reliably demonstrate venous sinuses and cortical veins, important for the preoperative planning (Figure 11).

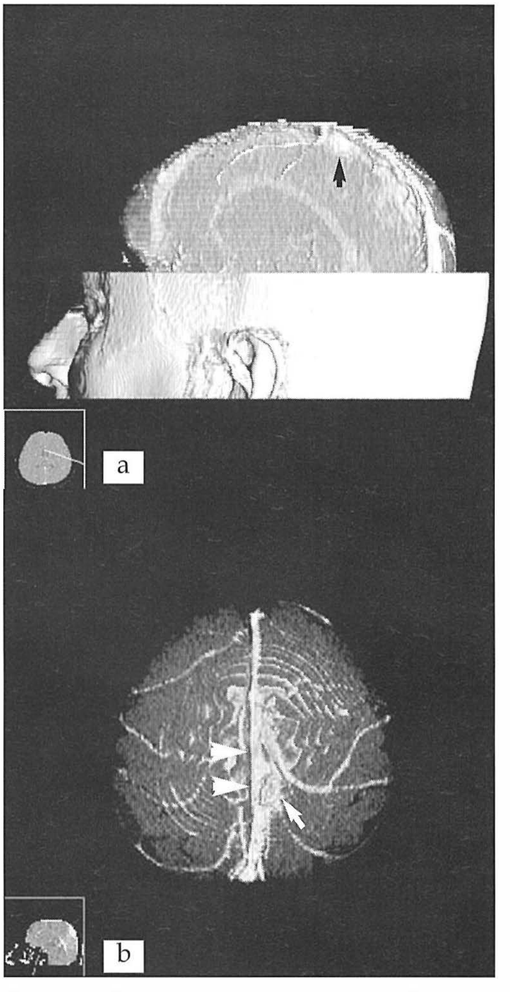

Figure 11. CT angiogram, volume rendering technique, left posterior oblique view (a) and inferior view (b) clearly demonstrates parasagittal meningeoma (arrows) and its relationship to the superior sagittal sinus (arrowhead) and cortical veins, which is important in preoperative planing.

Conclusion

CTA is the youngest angiografic imaging modality which has been quickly accepted especially for the detection and evaluation of intracranial saccular aneurysms, so far mostly diagnosed by the conventional intraarterial angiography.

An important part of CTA are post-processing techniques. Because threedimensional reconstructions of intracranial vessels offer anatomical imaging in the similar way as percepted with human vision, we can better understand the morphology of pathological proceses and its relations to surrounding structures.

Minimaly invasivenes of CTA represents an important advantage to conventional intraarterial angiography. The main disadvantage of CTA, has been and in some regard still is a lover spatial resolution compared to conventional angiography. Inspite of this, the balance between advantages and limitations still supports CTA in many clinical issuses.

A quick development of spiral CT scanners and image processing software enables further developement and improvement of CTA. A recent innovation of CT scanners with multiple detectors makes scanning of larger volumes with higher spatial resolution possible. Further improvement represent new software with volume rendering techniques and fast workstations, so that it is now possible to process larger quantity of data in much shorter time.

In conclusion, CTA, if combined with threedimensional techniques, has excellent possibilities to become a reliable and acceptable method for the evaluation of not only intracranial saccular aneurysms, but also of most intracranial vascular diseases.

References

1. Kalender WA, Sissler W,Vock P. Spiral volumetric CT with single breath-hold technique, continuous transport, and continuous scanner rotation. *Radiology* 1990; **176**: 181-3.
2. Vock P, Soucek M, Daepp M, Kalender WA. Lung: spiral volumetric CT with single-breath-hold technique. *Radiology* 1990; **176**: 864-7.
3. Rubin GD, Dake MD, Napel SA, McDonnell CH, Jeffrey RB. Abdominal spiral CT angiography: Initial clinical experience. *Radiology* 1993; **186**: 147-52.
4. Napel S, Marks MP, Rubin GD, Dake MD. McDonnel CH, Song SM, et al. CT angiography with spiral CT and maximum intensity projection. *Radiology* 1992; **185**: 607-10.
5. Osborn AG. *Diagnostic Neuroradiology*. St.Louis: Mosby; 1994.
6. Atkinson JL, Soundt TM Jr, Houser OW, Whisnant JP. Angiographic frequency of anterior circulation intracranial aneurysms. *J Neurosurg* 1989; **70**: 551-5.
7. Stone JL, Crowel RM, Gandy YN, Jafar JJ. Multiple intracranial aneurysms: Magnetic resonance imaging for determination of the site of rupture: Report of a case. *Neurosurgery* 1988; **23**: 97-100.
8. Hop JW, Rinkel GJE, Algra A, van Gijn J. Case fatality rates and functional outcome after subarachnoid hemorrhage: a sistematic review. *Stroke* 1997; **28**: 660-4.
9. Mani RL, Eisenberg RL, McDonald EJ Jr, Pollock JA, Mani JR. Complications of catheter cerebral arteriography: Analysis of 5000 procedures. I. Criteria and incidence. *Am J Roentgenol* 1978; **131**: 861-5.
10. Heiserman JE, Dean BL, Hodak JA, Floam RA, Bird CR, Drayer BP, et al. Neurologic complications of cerebral angiography. *Am J Neuroradiology* 1994; **15**: 1401-7.
11. Katz DA, Marks MP, Napel SA, Bracci PM, Roberts Sr. Circle of Willis: evaluation with CT angiography, MR angiography and conventional angiography. *Radiology* 1995; **195**: 445-9.
12. Harrison MJ, Johnson BA, Gardner GM, Welling BG. Preliminary results on management of unruptured intracranial aneurysms with MR angiography and CT angiography. *Neurosurgery* 1997; **40**: 947-57.
13. Ross JS, Masaryk TJ, Modic MT, Ruggieri PM, Haacke EM, Selman WR. Intracranial aneurysms: Evaluation by MR angiography. *Am J Neuroradiology* 1990; **11**: 449-56.
14. Huston j, Rufenacht DA, Ehman RL, Wiebers DO. Intracranial aneurysms and vascular malformations: Comparison of time of flight and phase contrast MR angiography. *Radiology* 1991; **181**: 721-30.
15. Korogi Y, Takahashi M, Mabuchi N, Miki H, Fujiwara S, Horikawa J, et al. Intracranial aneurysms: Diagnostic accuracy of three-dimensional, Furier transform, time of flight MR angiography. *Radiology* 1994; **193**: 181-6.
16. Stock KW, Radue EW, Jacob AL, Bao XS, Steinbrich W. Intracranial arteries: Prospective blinded comparative study of MR angiography and DSA in 50 patients. *Radiology* 1995; **195**: 451-6.
17. Ida M, Kurisu Y, Yamashita M. MR angiography of ruptured aneurysms in acute subarachnoid hemorrhage. *Am J Neuroradiology* 1997; **18**: 1025-32.
18. Shellock FG, Morisoli S, Kanal E. MR procedurs and biomedical implants, materials and devices: 1993 update. *Radiology* 1993; **189**: 587-99.
19. Anzalone N, Scomazzoni F, Strada L, Patay Z, Scotti G. Intracranial vascular malformations. *Eur Radiol* 1998; **8**: 685-90.
20. Alberico RA, Patel M, Casey S, Jacobs B, Maguire W, Decker R. Evaluation of Circle of Willis with three-dimensional CT angiography in patients with suspected intracranial aneurysms. *Am J Neuroradiol* 1995; **16**: 1571-8.

21. Ogawa T, Okudera T, Noguchi K. Cerebral aneurysms: evaluation with three-dimensional CT angiography. *Am J Neuroradiol* 1996; **17**: 447-54.

22. Aoki S, Sasaki Y, Machida T, Ohkubo T, Minami M, Sasaki Y. Cerebral aneurysms: Detection and delineation using 3-D-CT angiography. *Am J Neuroradiol* 1992; **13**: 115-20.

23. Miloševič Z. Acute subarachnoid haemorrhage: detection of aneurysms of intracranial arteries by computed tomographic angiography. *Radiol Oncol* 1999; **33**: 275-82.

24. Zouani A, Sahel M, Marro B, Clemenceau S, Dargent N, Bitar A, et al. Three-dimensional CT angiography in detection of cerebral aneurysms in acute subarachnoid hemorrhage. *Neurosurgery* 1997; **41**: 125-30.

25. Velthuis BK, Rinkel GJ, Ramos LM, Witkamp TD, van der Sprenkel JW, Vandertop WP, et al. Subarachnoid hemorrhage: aneurysm detection and preoperative evaluation with CT angiography. *Radiology* 1998; **208**: 423-30.

26. Korogi Y, Takahashi M, Imakita S, Abe T, Utsunomiya H, Ochi M, et al. Diagnostic accuracy of three-dimensional CT angiography in screening evaluation of intracranial aneurysms- *Int J Neuroradiol*, 1998; **4**: 373-9.

27. Preda L, Gaetani P, Rodriguez RB, Di Mggio EM, La fianza A, Dore R, et al. Spiral CT angiography and surgical correlations in evaluation of intracranial aneurysms. *Eur Radiol* 1998; **8**: 739-49.

28. Inagawa T. Ultra-early rebleeding within six hours after aneurysmal rupture. *Surg Neurol* 1994; **42**: 93-9.

29. Vieco PT, Shuman WP, Alsofrom GF, Gross CE. Detection of Circle of Willis aneurysms in patients with acute subarachnoid hemorrhage: A comparison of CT angiography and digital subtraction angiography. *AJR* 1995; **165**: 425-30.

30. Suzuki S, Kayama T, Ogawa A, Suzuki J. Subarachnoid haemorrhage of unknown case. *Neurosurgery* 1987; **21**: 310-3.

31. Levy LF. Subarachnoid haemorrhage without arteriographic vascular abnormality. *J Neurosurg* 1960; **17**: 252-8.

32. Kassel NF, Torner JC, Jane JA, Haley EC, Adams HP. The international cooperative study of the timing of aneurysm surgery, part 2. Surgical results. *J Neurosurg* 1990; **73**: 37-47.

33. Alexander MSM, Dias PS, Uttley D. Spontaneous subarachnoid haemorrhage and negative cerebral panangiography. Review of 140 cases. *J Neurosurg* 1986; **64**: 537-42.

34. Rinkel GJE, Wijdicks EFM, Vermeulen M, Ramos LMP, Tanghe HLJ, Hasan D, et al. Nonaneurysmal perimesencephalic subarachnoid haemorrhage: CT and MR patterns that differ from aneurysmal rupture. *Am J Neurorad* 1991; **12**: 829-34.

35. Hayward RD. Subarachnoid haemorrhage of unknown aetiology. A clinical and radiological study of 51 cases. *J Neurol Neurosurg Psychiatry* 1977; **40**: 926-31.

36. Dorsch NWC, Young N, Kingston RJ, Compton JS. Early experience with spiral CT in diagnosis of intracranial aneurysms. *Neurosurgery* 1995; **36**: 230-38.

37. Anzalone N, Scomazzoni F, Strada L, Patay Z, Scotti G. Intracranial vascular malformations. *Eur Radiol* 1998; **8**: 685-90.

38. Ronkainen A, Hernesniemi J, Ryynanen M, Puranen M, Kuivaniemi H. A ten percent prevalence of asymtomatic familial intracranial aneurysms: Preliminary report on 110 magnetic resonance angiography studies in members of 21 Finnish familial intracranial aneurysms families. *Neurosurgery* 1994; **35**: 208-13.

39. Ronkainen A, Hernesniemi J, Tromp G. Special features of familial intracranial aneurysms: Report of 215 familial aneurysms. *Neurosurgery* 1995; **37**: 43-7.

40. Ronkainen A, Puranen MI, Hernesniemi JA, Vanninen RL, Partanen PLK, Saari JR, et al. Intracranial aneurysms: MR angiographic screening in 400 asymptomatic individuals with increased familial risk. *Radiology* 1995; **195**: 35-40.

41. Schievink WI, Schaid DJ, Michels VV, Piepgras DG. Familial aneurysmal subarachnoid hemorrhage: A community based-study. *J Neurosurg* 1995; **83**: 426-9.

42. Inagawa T, Hada H, Katoh Y. Unruptured intracranial aneurysms in eldery patients. *Surg Neurol* 1992; **38**: 364-70.

43. Juvela S, Porras M, Heiskanen O. Natural history of unruptured intracranial aneurysms: A long term follow up study. *J Neurosurg* 1993; **79**: 174-82.

44. Mizoi K, Yasimoto T, Nagamine Y, Kayama T, Koshu K. How to treat incidental cerebral aneurysms: A review of 139 consecutive cases. *Surg Neurol* 1995; **44**: 114-21.

45. Wolpert SM, Caplan LR. Current role of cerebral angiography in the diagnosis of cerebrovascular disease. *AJR* 1992; **159**: 191-7.

46. Vieco PT, Morin III. EE, Gross CE. CT angiography in the examination of patients with aneurysm clips. *Am J Neuroradiology* 1996; **17**: 455-7.

47. Marro B, Galanaud D, Valery CA, Zouaoui A, Biondi A, Casasco A, et al. Intracranial aneurysm: inner view and neck identification with CT virtual endoscopy. *J Comput Assist Tomogr* 1997; **21**: 587-9.

48. Dumas JL, Oabdennebi A, Belin C. Role of 3D display MR angiography in the study of intracranial aneurysms. *Intervent Neuroradiol* 1995; **1**: 59-64.

49. Brown RD Jr, Wiebers DO, Forbes G, O'Fallon WM, Piepgras DG, Marsh WR, et al. The natural history of unruptured intracranial arteriovenous malformations. *J Neurosurg* 1988; **68**: 352-7.

50. Kesava P, Baker E, Mehta M, Turski P. Staging of arteriovenous malformations using three-dimensional time-of-flight MR angiography and volume rendered displays of surface anatomy. *AJR* 1996; **167**: 605-9.

51. Tanaka H, Numaguchi Y, Konno S, Shrier DA, Shibata DK, Patel U. Initial experience with helical CT and 3D reconstruction in therapeutic planning of cerebral AVMs: Comparison with 3D time-of-flight MRA and digital subtraction angiography. *J Comput Assist Tomogr* 1997; **21**: 811-7.

52. Rieger J, Hosten N, Neumann K, Langer R, Molsen P, Lanksch WR, et al. Initial clinical experience with spiral CT and 3D arteriel reconstruction in intracranial aneurysms and arteriovenous malformations. *Neuroradiology* 1996; **38**: 245-51.

53. Chen YC, Tsuruda JS, Halbach VV. Suspected dural arteriovenous fistula: Results with screening MR angiography in seven patients. *Radiology* 1992; **183**: 265-71.

54. De Marco JK, Dillon WP, Halback VV, Tsuruda JS. Dural arteriovenous fistulas: Evaluation with MR imaging. *Radiology* 1990; **175**:193-9.

55. Lasjaunias P, Burrows P, Planet C. Developmental venous anomalies (DVA): the so called venous angioma. *Neurosurg Rev* 1986; **9**: 233-44.

56. Awad IA, Robinson JR Jr, Mohanty S, Estes ML. Mixed vascular malformations of the brain: Clinical and pathogenetic considerations. *Neurosurgery* 1993, **33**: 179-88.

57. Valavanis A, Wellauer J, Yasargil MG. The radiological diagnosis of cerebral venous angioma: Cerebral angiography and computed tomography. *Neuroradiology* 1983; **24**: 193-9.

58. Garner TB, Curling OD Jr, Kelly DL, Laster DW. The natural history of intracranial venous angiomas. *J Neurosurg* 1991; **75**: 715-22.

59. Truwit CL. Venous angioma of the brain: History, significance and imaging findings. *AJR* 1992; **159**: 1299-307.

60. Wilms G, Demaerel P, Marchal G, Baert AL, Plets C. Gadolinium enhanced MR imaging of cerebral venous angiomas with emphisasis on their drainage. *J Comput Assist Tomogr* 1991; **15**: 199-206.

61. Toro VE, Geyer CA, Sherman JL, Parisi JE, Brantley MJ. Cerebral venous angiomas: MR findings. *J Comput Assist Tomogr* 1988; **12**: 935-40.

62. Peebles TR, Vieco PT. Intracranial developmental venous anomalies: Diagnosis using CT angiography. *J Comput Assist Tomogr* 1997; **21**: 582-6.

63. Kassell NF, Sasaki T, Colohan ART, Nazar G. Cerebral vasospasm following aneurysmal subarachnoid hemorrhage. *Stroke* 1985; **16**: 562-81.

64. Piepgras DG. Clinical decision making in intracranial aneurysms and aneurysmal subarachniod hemorhhage-science and art. *Clin Neurosurg* 1992; **39**: 68-75.

65. Tsuchiya T, Yasaka M, Yamaguchi T, Kimura K, Omae T. Imaging of basal cerebral arteries and measurement of blood velocity in adults by using transcranial real-time color flow Doppler sonography. *Am J Neurorad* 1991; **12**: 497-502.

66. Klingelhofer JD, Sander D, Holzgraefe M, Bischoff C, Conrad B. Cerebral vasospasm evaluated by transcranial Doppler ultrasonography at different intracranial pressures. *J Neurosurg* 1991; **75**: 752-8.

67. Ochi RP, Vieco PT, Gross CE. CT angiography of cerebral vasospasm with conventional angiographic comparison. *Am J Neurorad* 1997; **18**: 265-9.

68. Hurst RW. Carotid angioplasty *Radiology* 1996; **201**: 613-6.

69. Žvan B. Epidemiology of stroke in Republic of Slovenia. *Acta Clin Croat* 1998; **37 (Suppl 1)**: 95-7.

70. De Bakey M. Carotid endarterectomy revisited. *J Endovasc Surg* 1996; **3**: 3-5.

71. Grosset DG, Georgiadis D, Abdullah I, Bone I, Lees KR. Doppler emboli signals vary according to stroke subtype. *Stroke* 1994; **25**: 382-4.

72. Wildenhain SL, Jungreis CA, Barr J, Methis J, Wechsler L, Horton JA. CT and intracranial intraarterial thrombolysis for acute stroke. *Am J Neurorad* 1995; **15**: 487-92.

73. Lanzieri CF, Tarr RW, Landis D, Selman WR, Lewin JS, Adler LP, et al. Cost effectivnes of emergency intraarterial intracerebral thrombolysis: a pilot study. *Am J Neurorad* 1995; **16**: 1987-93.

74. The Natonal Institute of Neurological Disorders and Stroke rt-PA Stroke Study Group. Tissue plasminogen activator for acute ischemic stroke. *N Engl J Med* 1995; **333**: 1581-7.

75. Hacke W, Kaste M, Fieschi C, Toni D, Lesaffre E, von Kummer R,et al. Intravenous thrombolysis with recombinant plasminogen activator for acute hemispheric stroke: the European Cooperative Acute Stroke Study (ECASS). *JAMA* 1995; **274**: 1017-25.

76. Tomura N, Uemura K, Fujita H, Higano S, Shishido F. Early CT finding in cerebral infarction. *Radiology* 1998; **168**: 463-7.

77. von Kummer R, Nolte PN, Schnittger H, Thron A, Ringeistein EB. Detectability of hemispheric ischemic infarction by computed tomography within 6 hours after stroke. *Neuroradiology* 1996; **38**: 31-3.

78. Knauth M, von Kummer R, Jansen O, Hahnel S, Dorfer A, Sartor K. Potential of CT angiography in acute ischemic stroke. *Am J Neuroradiol* 1997; **18**: 1001-10.

79. Sorensen AG, Buonanno FS, Gonzalez RG, Schwamm LH, Lev MH, Huang-Hellinger FR, et al. Hyperacute stroke: evaluation with combined multisection diffusion-weighted and hemodynamically weighted echo-planar MR imaging. *Radiology* 1998; **199**: 391-401.

80. Ueda T, Yuh WTC, Maley JE, Quets JP, Hahn PY, Magnotta VA. Outcome of acute ischemic lesions evaluated by diffusion and perfusion MR imaging. *Am J Neuroradiol* 1999; **20**: 983-9.

81. Shrier DA, Tanaka H, Numaguchi Y, Konno S, Patel U, Shibata D. CT angiography in the evaluation of acute ischemic stroke. *Am J Neuroradiol* 1997; **18**: 1911-20.

82. Kucinski T, Koch C, Grzyska U, Freitag HJ, Kromer H, Zeumer H. The predictive value of early CT and angiography for fatal hemispheric swelling in acute stroke. *Am J Neuroradiol* 1998; **19**: 839-46.

83. Consigny PM. Advances in clinical medicine: pathogenesis of atherosclerosis. *AJR* 1995: **164**: 553-8.

84. Harris KG, Yuh WTC. Intracranial vasculitis. *Neuroimaging Clin North Am* 1994; **4**: 773-97.

85. Stock KW, Radue EW, Jacob AL, Bao XS, Steinbrich W. Intracranial arteries: prospective blinded comparative study of MR angiography and DSA in 50 patients. *Radiology* 1995; **195**: 451-6.

86. Dagirmanjian A, Ross JS, Obuchowski N, Lewin JS, Tkach JA Ruggier PM, et al. High resolution, magnetization transfer saturation, variable flip angle, time of flight MRA in the detection of intracranial vascular stenoses. *J Comput Assist Tomogr* 1995; **19**: 700-6.

87. Korogi Y, Takahashi M, Nakagawa T, Mabuchi N, Watabe T Shiokawa Y, et al. Intracranial vascular stenosis and occlusion: MR angiographic findings. *Am J Neuroradiol* 1997; **18**: 135-43.

88. Skutta B, Furst G, Eilers J, Ferbert A, Kuhn FP. Intracranial stenoocclusive disease: Double-detector helical CT angiography versus digital subtraction angiography. *Am J Neuroradiol* 1999; **20**: 791-9.

89. Zimmerman RD, Ernst RJ. Neuroimaging of cerebral veins: Thrombosis. *Neuroimaging Clin North Am* 1992; **2**: 463-85.

90. Johnson BA, Fram EK. Cerebral venous occlusive disease *Neuroimaging Clin North Am* 1992; **2**: 769-83.

91. Medlock MD, Olivero WC, Hanigan WC, Wright RM, Winek SJ. Children with cerebral venous thrombosis diagnosed with magnetic resonance imaging and magnetic resonance angiography. *Neurosurgery* 1992; **31**: 870-76.

92. Casey SO, Alberico RA, Patel M, Jimenez JM, Ozsvath RR, Maguire WM, et al. Cerebral CT venography. *Radiology* 1996; **198**: 163-70.

93. Buonanno FS, Mody DM, Ball MR, Laster DW. Computed cranial tomographic findings in cerebral sinovenous occlusion. *J Comput Assist Tomogr* 1978; **2**: 281-90.

94. Vogl TJ, Bergman C, Villringer A, Einhaupl K, Lissner J, Felix R. Dural sinus thrombosis: value of venous MR angiography for diagnosis and follow up. *AJR* 1994; **162**: 1191-8.

95. Mattle HP, Wentz KU, Edelman RR, Wallner B, Finn JP, Barnes P, et al. Cerebral venography with MR. *Radiology* 1991; **178**: 453-8.

96. Ozsvath RR, Casey SO, Lustrin ES, Alberico RA, Hassankhani A, Patel M. Cerebral venography: comparison of CT and MR projection venography. *AJR* 1997; **169**: 1699-707.