

MR Sialography?

Sialography has become an infrequent procedure in current radiologic practice. Indeed only two sialograms have been requested in our department over the last five years! Sialography has been largely replaced by CT and MR, which provide information regarding intrinsic salivary gland disease including neoplasms, cysts, and inflammatory processes. Routine imaging with CT and MR, however, does not generally yield much information about ductal integrity. Because saliva has very high signal intensity on fat suppressed, fast spin echo T2-weighted images, it provides a perfect intrinsic contrast material for the visualization of the salivary ducts using 2-D or 3-D sequences. In this issue of the *American Journal of Neuroradiology*, Tonami et al (page 1199) report their experience with fast spin echo MR imaging to perform MR sialography. The term "MR sialography," however, is somewhat of a misnomer in this context. MR does not demonstrate delayed filling or emptying without an injection of contrast material, and the occasional therapeutic success of stone release during plain film sialography will not occur. Nevertheless, the potential for MR to demonstrate the ductal anatomy of the parotid and, hopefully, the submandibular duct is obvious. This may be useful in preoperative planning or in the evaluation of suspected ductal strictures.

Prior to the CT era, sialography was frequently used to evaluate suspected intrinsic abnormalities of the salivary glands and ducts, including calculi, strictures, and autoimmune diseases. CT and MR have largely replaced sialography for the evaluation of the gland, and with 1-mm helical scanning, even tiny calculi may be observed within the ducts of the major salivary glands. At our institution, a focused helical CT is the first examination performed for the detection of calculi. In some practices, sialography is still occasionally used in the evaluation of the patient with suspected ductal stricture or findings suggestive of Sjögren's syndrome. Sjögren's syndrome is a disorder of cell-mediated and humoral immunity. Patients present with dry eyes, dry mouth and chronic arthritis—symptoms associated with parotid gland enlargement. Other autoimmune diseases associated with

Sjögren's syndrome are rheumatoid arthritis, systemic lupus erythematosus, scleroderma, polymyositis, and periarteritis nodosa. The earliest symptoms arise from periductal lymphocytic infiltration of the lacrimal and salivary glands, resulting in hyperplasia of the ductal lining cells, and ultimately ductal narrowing, dilatation, and delayed emptying of contrast media. Sialography is used occasionally to confirm these findings that typify Sjögren's syndrome. The accuracy of sialography for the diagnosis of this syndrome has been shown to be excellent, but ultrasound also has an acceptable accuracy, is noninvasive, and has thus been suggested as a preferable first-line tool in this clinical situation (1). At our institution, the occasional patient presenting with ambiguous symptoms is usually referred for lip biopsy, which may demonstrate infiltrates of plasma cells and lymphocytes in the minor salivary glands, confirming the diagnosis of Sjögren's syndrome, rather than for sialography.

Tonami et al state that they carry out "MR sialography" in patients in whom clinical symptoms are suspicious or for evaluation of disease progression. They do not state what role the imaging features play in the decision-making process, nor do they demonstrate the ability of MR to monitor the progression of disease. It, therefore, remains unclear what the actual role "MR sialography" will play in the evaluation of patients with Sjögren's syndrome. Clearly this technique can be used in lieu of X-ray sialography to illustrate a variety of conditions as well as the relationship of the duct to intraparotid tumor deposits and the degree of ductal strictures in patients with chronic parotitis of unknown origin. As MR becomes faster, and high resolution imaging with phase array surface coils becomes a wider diagnostic application for these conditions, we may indeed find that the indication for sialography will be further limited.

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References

1. Yoshiura K, Yuasa K, Tabata O, et al. **Reliability of ultrasonography and sialography in the diagnosis of Sjögren's syndrome.** *Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology and Endodontics.* 1997;83(3):400-407

Understanding the Natural History of Saccular Aneurysms

An assessment of the chance of rupture of an asymptomatic saccular aneurysm is based almost entirely on a statistical analysis of the natural history of "similar" lesions. Although a number of factors combine to influence the magnitude of this risk (age, sex, number of aneurysms, history of smoking, etc.) the

one factor usually deemed of greatest impact is size, with larger aneurysms judged to be more at risk of rupture than smaller ones. The article by Tenjin et al appearing in this issue of the *American Journal of Neuroradiology* (page 1303) is noteworthy because it provides insight into one method that might be help-

ful in formulating techniques that would allow a more objective and individual estimation of this risk. Research directed at the use of several rapidly evolving techniques in angiography, MR and ultrasound aimed at the improved imaging of the hemodynamic stresses that affect aneurysms, may better define the relationship between these stresses and the vascular remodeling seen in aneurysm wall degeneration or healing. This research offers a rich opportunity for neuroradiologists to advance their understanding and treatment of this common and too often lethal disease.

Aneurysm growth, unlike the progressive thinning found when a weak spot forms in the wall of a balloon or an inner tube, is a dynamic process that results from complex and incompletely understood interactions between hemodynamic forces and the degeneration and production of structural components that compose an aneurysm's wall. Because of physical limitations in the collagen present at the site where an aneurysm forms, it would be expected that growth much beyond a diameter of 8 mm would always result in rupture. In the majority of instances this does not occur. Whether a particular aneurysm grows and ruptures, grows and does not rupture, remains unchanged in size, or even shrinks, could be thought of as being dependent on the outcome of an engagement between forces that enhance healing by strengthening the wall and forces that inhibit healing by intensifying degeneration of the wall. Changes in a variety of hemodynamic factors (shear stress, pressure, and impingement force) as well as the presence of several humoral factors (inflammatory mediators and adhesion molecules) are potential signals of arterial injury. Sensing these signals, endothelium then has the capacity to regulate the activity of substances that either act to promote or to inhibit repair of an aneurysm wall. For example, a recent experimental study in humans has demonstrated the simultaneous presence of increased amounts of certain proteinases and proteinase inhibitors found in tissue of the walls of saccular aneurysms. This increase of a proteinase along with a simultaneous, associated increase in its inhibitor provides some evidence of a real-life correlate of the engagement between healing and degeneration. Likewise, studies have documented a decrease in the presence of fibronectin (one extracellular matrix factor that promotes wound healing) in aneurysm tissue of rats with hemodynamically induced intracranial aneurysms. When the hemodynamic stress was reduced, an increase in fibronectin in this tissue was shown, and was associated with corresponding histologic evidence of aneurysm healing. Well-documented observations of saccular aneurysm healing proximal to arteriovenous vascular malformations after treatment of the malformation provide a human analogy to these animal experiments. How then do

these still theoretical considerations regarding vascular remodeling and aneurysm growth relate to opportunities for neuroradiologic research?

Because of the capacity of available, evolving imaging techniques to depict vascular morphology accurately and to assess, and in some instances, quantify certain aspects of cerebral hemodynamics, neuroradiologists have a new opportunity to add to the understanding of the natural history of saccular aneurysms. As one example, the hypothesis that there is a correlation between the geometry of an aneurysm and its parent artery and the risk of aneurysm growth and rupture might be tested. Lateral aneurysms and terminal aneurysms with symmetrical branching are associated with sluggish flow whereas bifurcation aneurysms and terminal aneurysms with asymmetrical branching are characterized by hyperdynamic flow. These patterns of flow result in markedly different stresses on the aneurysms, and may generate distinct biologic responses. Hemodynamics are determined primarily by geometric relationships, and the use of either CT or MR angiography offers the opportunity to define and classify these geometric features non-invasively with far more accuracy and precision than has previously been the case. If the hypothesis is proven, aneurysms might then be classified according to geometry, and an individual profile for risk of rupture might be established.

These same imaging modalities have potentially even greater impact when used as study tools aimed at increasing the effectiveness of endovascular, therapeutic techniques for saccular aneurysms. Combinations of CT, MR imaging and angiography, and ultrasound techniques can, in some instances, measure both geometric relationships *and* such physiologic parameters as shear stress, pulse pressure and compliance prior to and after a given intervention. As these techniques evolve, improve and become more widely used, they will provide data that is valuable both in laboratory and clinical settings. Several simple, consistent, and reproducible animal models of saccular aneurysms offer helpful paradigms for understanding the mechanisms whereby hemodynamic stresses are relieved following treatment with mechanical devices. Other more complex experimental models offer the opportunity to study the influence these interventions have on hemodynamic stresses as well as their impact on biologic responses. This chance to advance device development from a historically hypothetical to an empirically-based approach is potentially revolutionary. In all of the neurosciences, few investigators have such powerful tools at their disposal as do neuroradiologists. We must not waste this good fortune.

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