Coils and Cash: What Coil Vendors Don't Want You to Know

H.J. Cloft

AJNR Am J Neuroradiol 2009, 30 (7) 1276-1277
doi: https://doi.org/10.3174/ajnr.A1638
http://www.ajnr.org/content/30/7/1276

EDITORIAL

Coils and Cash: What Coil Vendors Don't Want You to Know

My problem lies in reconciling my gross habits with my net income.

Errol Flynn

Until the introduction of coils by Micrus (San Jose, Calif) to the market in 2000, Boston Scientific (Natick, Mass) had a detachable coil monopoly. Since then, Cordis (Miami Lakes, Fla), MicroVention (Alisa Viejo, Calif), ev3 (Irvine, Calif), and Cook (Bloomington, Ind) have entered the market. Despite a large increase in competition in the marketplace, detachable coil prices have continued to rise. Perhaps the single largest contributor to coil cost escalation is that physicians are generally not sensitive to device pricing because their salary and resources have not been directly related to hospital costs. Physicians thus have tended to choose coils with little regard to cost. We now have coils on the market ranging in list price from $500 to $3000.

Many diseases can be treated with a single device, such as an arterial stenosis, which is treated with a single stent. However, cerebral aneurysms are unusual in that we generally need several expensive coils to treat a single aneurysm, and adding a stent for cerebral aneurysm treatment, which costs $5200 to $5300 each, makes the coils look cheap. In a recent study, it was shown that the hospital costs for coiling an aneurysm at a single center in the United States were *one-third higher* than the costs for clipping, largely because several coils cost much more than a single clip.[1] Stents under development to treat aneurysms by flow diversion are likely to take the costs even higher. This cost escalation is not sustainable in a new era of containment of medical care costs. In the current recession, many hospitals are suffering financially, with some even closing.[2] We physicians are going to be under increasing pressure to decrease costs. We need to help get control of device costs ourselves, because if we cannot regulate our own spending, health care administrators will probably ultimately step in and regulate it for us. Our salaries and resources may not remain disconnected from device expenses forever.

Physicians generally want what is best for the patient, which may in large part explain why we so willingly adopt new devices on the basis of only theoretic benefit and without regard to cost. Medicolegal paranoia also adds to the willingness of physicians to participate in this cost escalation. When "biologically active" coils were introduced to the market, many physicians were worried that they might be accused of malpractice for not using the "latest technology." This medicolegal concern was widespread, even though there was no proof of benefit with these coils, and, in fact, these coils were brought to market as being "substantially equivalent" to platinum coils. Now that the "biologically active" coil hype cycle has run its course,[3] we neurointerventionalists should face up to the fact that we spent millions of health care dollars and got no proved benefit in return.

Physicians will also buy new coils because they are afraid of being left behind if they do not embrace the latest technology. This is an understandable perspective, but rarely do any of the new coils represent a major technical advance. With time, there has been so much proliferation of subtle variations of coils that I no longer have time to listen to all of the hype and then try each one. With rare exception, my inclination now is to assume that each new coil is another "me too" product and then just wait to hear from an unbiased source if it actually seems to be of any particular added value.

Many neurointerventionalists are uninformed regarding the cost and reimbursement of medical devices. There seems to be a widespread misconception that when we use a coil, the hospital then charges for that coil and then the hospital gets reimbursed for that coil. In reality, that is not what happens for many, and perhaps most, of our patients. What often happens is that the third-party payers pay a limited amount for care of a patient with a specific problem. In 2008, the national average payment by Medicare for the entire hospitalization for the uncomplicated endovascular treatment of an unruptured aneurysm was $12,599. Just the access materials (guide catheter, microcatheter, microguidewire, etc) cost at least $1000. If you use 12 coils at $1000 each or if you use a stent for $5200 and 7 coils for $1000 each, then you have spent more than the entire Medicare reimbursement on devices, and every other expense related to the hospitalization is a financial loss to the institution. It is easy to see that even more expensive coils would rapidly escalate the cost. Every time you coil an aneurysm, the vendor gets paid well for the devices used, but the physician and the hospital do not have any such guarantee of getting paid a fair price and may actually end up losing money after they have paid their bills.

Third-party payers have only so much money that they are willing to pay out for the treatment of each patient's cerebral aneurysm. This means that physicians and hospitals are directly competing with the medical-device industry for health care dollars that have been committed to treatment of these patients.

Neurointerventionalists and device-industry representatives love to talk about a partnership with each other. Occasionally, this partnership has something to do with research or device development. Much more often, the partnership translates into some arrangement in which the physician buys more coils. The typical neurointerventionalist spends much more time dining with and speaking with vendor representatives than with hospital representatives, and this works against the interest of hospitals. In the long run, it almost certainly works against the interest of physicians as well because we need constructive relationships with our hospitals if we are to prosper. If you are paying retail list prices for your coils, then I dare say that the only explanations that I can think of are that you are being played for a chump or you have a conflict of interest.

Physicians have not yet taken full advantage of the highly competitive market for endovascular devices, in which each vendor is struggling for market share. To a large extent, aneurysm coils are a commodity, and we physicians could let vendors know that we are paying attention to prices. Even if you are loyal to a particular vendor because you think the product is better, you should be able to use your loyalty in this competitive market as a price negotiating advantage. I have started refocusing the discussion with vendors. I am telling each of

them that I do not want to spend my valuable time talking about the theoretic advantages of introducing yet another $1300+ coil to my practice, but instead I want to talk about *value*, which is a ratio of proven quality over cost. We can get proved quality from several vendors; so the question now is, which vendors can give us lower costs?

Partnering with our hospitals, we should be able to push vendors to give us the products that we want at competitive pricing. We need to make it clear to vendors that controlling device expenses is a priority and will be a central theme of our partnership with them in the future. If we do not, I suspect that we will eventually find that hospitals can no longer afford to take care of our patients.

References

1. Hoh BL, Chi YY, Dermott MA, et al. **The effect of coiling versus clipping of ruptured and unruptured cerebral aneurysms on length of stay, hospital cost, hospital reimbursement, and surgeon reimbursement at the University of Florida**. *Neurosurgery* 2009;64:614–19
2. Girion L, Medina M. **Hospitals feel ill effects of recession.** *Los Angeles Times.* January 14, 2009. Available at: http://articles.latimes.com/2009/jan/14/business/fi-hospitals14. Accessed March 10, 2009
3. Cloft HJ. **What is all of the hype about?** *AJNR Am J Neuroradiol* 2008;29:1604

H.J. Cloft
Senior Editor

DOI 10.3174/ajnr.A1638

EDITORIAL

Tissue-Specific MR Imaging in Multiple Sclerosis

Conventional MR imaging techniques (cMRI), such as T2-weighted sequences and gadolinium-enhanced T1-weighted sequences, which are highly sensitive for detecting multiple sclerosis (MS) plaques, have become established as the most important paraclinical tool for diagnosing MS, as well as for understanding the natural history of the disease and monitoring the efficacy of experimental treatments. In fact, cMRI metrics have become common primary end points in phase II immunomodulatory drug therapy trials.[1] However, a possible role of cMRI metrics as surrogate end points in phase III trials has been disclaimed because of the poor correlation between cMRI metrics and the clinical disease course, particularly disability progression, which is driven by the neurodegenerative component of the disease.[2]

Explanations for this clinical-radiologic discrepancy include inappropriate clinical rating, neglect of spinal cord involvement, underestimation of damage to the normal-appearing brain tissue (both white and gray matter), and compensation by cortical adaptation.[3] However, one of the major contributors to this paradox is the lack of pathologic specificity of T2-weighted imaging, which provides only a dichotomous type of information, that is, it simply discriminates between MS focal lesions and normal-appearing white matter but not between the type and degree of the underlying pathologic substrates (edema, inflammation, demyelination, remyelination, reactive gliosis, and axonal loss)[4] that contribute differently to the development of permanent disability.

In the last 15 years, a huge effort has been made by the MR imaging research community to overcome the limited pathologic specificity of cMRI by developing new MR imaging techniques that selectively measure the more destructive aspects of MS pathology and monitor the reparative mechanisms, such as T1 hypointense lesions, quantitative analysis of global and regional brain volume, magnetization transfer MR imaging, diffusion-weighted MR imaging, and proton MR spectroscopy. These techniques appear to be more sensitive biomarkers for measuring the pathologic processes underlying the progression of clinical disability (demyelination and axonal loss).[5]

The first MR imaging–based measure proposed as a specific marker of focal MS lesions with severe tissue destruction was T1 hypointense lesions.[6] However, these so-called T1 "black holes" may have a different pathologic substrate depending, in part, on the lesion age. Hypointensity is present in ≤80% of recently formed lesions and likely represents marked edema, with or without myelin destruction or axonal loss. In most cases, acute ("wet") black holes become isointense or slightly hypointense within a few months, as inflammatory activity abates, edema resolves, and reparative mechanisms such as remyelination become active, resulting in partial axonal preservation. Less than 40% evolve into persisting or chronic black holes,[7,8] which correlate pathologically with permanent demyelination and severe axonal loss. Several immunomodulatory drugs (glatiramer acetate, interferon beta, and natalizumab) reduce the progression of acute gadolinium-enhancing lesions into persistent or chronic black holes, supporting a certain neuroprotective effect of these treatments by disrupting the advancement of tissue destruction.[9-11]

However this MR imaging–based measure has some important drawbacks that limit its use as a true marker of severe irreversible tissue damage. One of the most important limitations is the fact that the definition of what constitutes a black hole is arbitrary, highly dependent on the MR imaging technique used, and based on visual inspection. Therefore, it remains a challenge to accurately discriminate between slightly/moderately and strongly hypointense T1 lesions, which reflect different degrees of remyelination and axonal loss. This pathologic heterogeneity has been demonstrated by postmortem studies[12] and by in vivo MR spectroscopy and magnetization transfer imaging studies, which have shown that tissue damage is extremely variable in individual black holes.[13,14] Consequently, patients with similar black hole lesion volume may have different degrees of disability depending on the nature of the histopathologic substrate.

For this reason, new ways of measuring black holes have been recently developed, such as the T1 hypointensity ratio[15] and the one proposed in this issue of *American Journal of Neuroradiology* by Riva et al.[16] These authors assessed the ability of a new MR imaging technique, which they call "tissue-specific imaging" to selectively identify black holes with the longest T1 values, which likely reflect lesions with severe demyelination and axonal loss. The results of this study are promising and provide data indicating that this technique could be a sensitive method for detecting and quantifying hypointense T1 lesions with more advanced tissue destruction. Nevertheless, additional studies are required before new MR imaging–based measures can be considered markers of disease severity and progression in MS or surrogate markers of remyelination and