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Cerebral Hyperperfusion Syndrome After Angioplasty

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1. Introduction

Cerebral hyperperfusion syndrome (CHS) was first described by Sundt et al. (1981) as a clinical syndrome following carotid endarterectomy (CEA) characterized by headache, neurological deficit, and epileptic seizures that is not caused by cerebral ischemia.

This chapter deals with this uncommon but not exceptional complication of endovascular treatment of the arteries that supply the brain. We use the term carotid artery stenting (CAS) to refer to stenting of the internal carotid artery (ICA) because most publications are centered on this artery. Moreover, we include angioplasty without stent placement in the term CAS to facilitate reading comprehension because the relation between endovascular treatment and CHS is related to revascularization itself rather than to stent placement per se. Given the high rate of ischemic brain disease in relation to carotid stenosis and the high prevalence of asymptomatic carotid stenosis, numerous publications discuss CHS in relation to CEA: the incidence in these series ranges from 0.3% to 2.2%. However, CAS has continually evolved in recent years to the point where, after more than 40 years' experience, it is considered an alternative to CEA. Furthermore, the development of new materials for stents, filters for distal protection, dual antiplatelet treatment, and the learning curve are minimizing the short- and long-term adverse effects of CAS.

Documented complications of CAS include cerebral embolism, hemodynamic compromise, vessel dissection, and early restenosis and occlusion, as well as the hyperperfusion syndrome we deal with in this chapter. Moreover, the spectacular increase in endovascular treatment has revealed that hyperperfusion syndrome can also occur after revascularization of other arteries, such as the vertebral arteries, the subclavian arteries, or even those located within the brain, mainly the middle cerebral artery (MCA).

In this chapter we will begin by discussing the pathophysiology, clinical presentation, and incidence of CHS in the different published series. We will then discuss the risk factors, diagnostic methods, and strategies for prevention and treatment. We will also discuss a

condition that shares the same pathophysiology as CHS, contrast-induced encephalopathy, in which contrast agents crossing the blood-brain barrier have a toxic effect on the brain parenchyma, resulting in signs and symptoms similar to those of CHS. Given the larger number of publications about hyperperfusion after CEA and the obvious similarities in aspects like the pathophysiology and risk factors, we refer to CEA on numerous occasions in this chapter.

2. Pathophysiology

First, we must differentiate between the concept of hyperperfusion and CHS. In general, hyperperfusion is considered to occur when cerebral blood flow (CBF) in the revascularized territory increases by 100% or more with respect to the baseline values. In series by Ogasawara (2007) and Fukuda (2007), 16.7% to 28.6% of the patients with an increase in CBF 100% developed CHS. Moreover, a few cases of CHS in which CBF had increased less than 100% have been reported (Karapanayiotides et al, 2005; Henderson et al, 2001). Thus, other factors must be involved in CHS (Hosoda et al, 2003; Kaku et al, 2004; Ogasawara et al 2003; Suga et al, 2007; Yoshimoto et al, 1997).

All authors agree that it is very likely that there has to be damage to cerebral autoregulation, in other words, impaired cerebral vasoreactivity (CVR), for CHS to occur (Keunen et al, 2001).

Cerebral hemodynamics and CVR are individualized in each patient. This could be explained by the different extent of collateral circulation available and by the autoregulatory mechanisms of the cerebral circulation. The presence of sufficient collateral circulation has a key role in the preservation of CVR, and thus protects against CHS.

Similarly, other risk factors for CHS are low pulsatility index, severe ipsilateral and contralateral carotid disease, and an incomplete circle of Willis (Jansen et al, 1994; Reigel et al, 1987; Sbarigia et al 1993).

CVR makes it possible to keep blood pressure (BP) between acceptable limits (60 mmHg - 160 mmHg) through arteriolar vasodilatation or vasoconstriction in response to changes in carbon dioxide. This response is most pronounced in smaller arteries (diameter 0.5–1.0 mm), whereas arteries with a diameter of 2.5 mm or more like the ICA show no substantial change.

Regulation involves a myogenic and a neurogenic component. In myogenic autoregulation, increased intravascular pressure results in vasoconstriction of small arterioles at high systemic BP, but when BP exceeds the limit of myogenic autoregulation, the remaining autoregulation in small arteries is dependent on sympathetic autonomic innervation. As a result of sparse sympathetic innervation, the vertebrobasilar system is less protected than other regions of the brain, which explains why this system is more affected in entities like hypertensive encephalopathy. Impaired CVR results in failure of the arterial system to respond to a sudden increase in CBF and is usually due to severe vascular stenosis together with insufficient collateral blood flow. When these two factors coexist, cerebral perfusion is maintained by the maximum dilation of the arterioles. This prolonged vasodilation makes the vessels unable to respond with vasoconstriction when blood flow is increased, and especially when it is increased suddenly (Ascher et al, 2003; Jansen et al, 1994; Reigel et al, 1987; Tang et al, 2008 Sbarigia et al, 1993).

At the end of the 1990s, some surgical reports already suggested that patients with preoperative hemodynamic failure were at definite risk for CHS (Baker et al, 1998; Cikrit et al 1997; Yoshimoto et al, 1997) and that the presence of a critical stenosis in the ICA

increased the risk of intracranial hemorrhage (ICH) (Jansen et al, 1994; Macfarlane et al, 1991; Ouriel et al, 1999; Sbarigia et al, 1993). Preoperative significant reduction in flow velocity compared with baseline values is indicative of hypoperfusion and is associated with postoperative hyperperfusion (Keunen et al, 2001).

Sudden revascularization brought about by angioplasty leads to dysfunction of the blood-brain barrier after the failure of arteriolar vasoconstriction. This results in transudation of fluid into the pericapillary astrocytes and interstitium, giving rise to vasogenic edema. This hydrostatic edema predominantly affects the vertebrobasilar circulation territory in both CHS and hypertensive encephalopathy, possibly as a result of regional variation in cerebral sympathetic innervation.

The most extreme form of this syndrome is bleeding, either ICH, which results in high morbidity and mortality, or subarachnoid hemorrhage (SAH), which has a better prognosis. The pathophysiology of the hemorrhage that results from revascularization might be different from that of CHS described by Sundt, et al (1981). Some authors (Karapanayiotides et al, 2005) prefer to call this entity "reperfusion syndrome" to emphasize the damage to tissues caused by simple reperfusion. Several investigators have analyzed the characteristics of this ICH when it appears in the first few hours and without prodromes, attributing it to the rupture of deep penetrating arteries as a result of the sudden normalization of the pressure of cerebral perfusion after angioplasty, similar to what occurs in hemorrhage due to hypertension (Buhk et al, 2006; Coutts et al, 2003).

Many cases of SAH after CAS have been reported (Abou-Chebl et al, 2004; Coutts et al, 2003; Hartmann et al, 2004; Ho et al, 2000; McCabe et al, 1999; Meyers et al, 2000; Morrish et al, 2000; Nikolsky et al, 2002; Pilz et al, 2006; Qureshi et al, 2002); these have a better prognosis than ICH.

It is logical to assume that CBF increases substantially after CAS in a severely stenosed carotid artery. However, studies show that the increase in CBF is actually related to impaired CVR. In a study by Hosoda et al (1998) CBF significantly increased on the first postoperative day in subjects with reduced preoperative CVR but not in those with normal preoperative CVR. Similarly, in a study of 23 patients, Ko et al (2005) were unable to demonstrate a relation between the degree of stenosis and the increase in CBF. In short, the degree of stenosis cannot be considered a key risk factor for CHS, although some series have taken it into account.

Ascher et al (2003) studied 455 patients undergoing CEA and found no relation between CHS and the severity of ipsilateral or contralateral carotid stenosis, arterial hypertension, or perioperative perfusion pressure. However, mean ICA volume flow and peak systolic velocity measured at the onset of symptoms in the 9 CHS cases were higher than in the remaining 446 cases.

In most cases of symptomatic carotid stenoses due to a hemodynamic mechanism CVR is also deficient, so it is logical to think that they will be more susceptible to developing CHS after revascularization (Brantley et al, 2009). However, in a study of 333 patients undergoing CAS, Karkos et al (2010) found no significant differences between symptomatic and asymptomatic patients.

Fukuda et al (2007) carried out an interesting study of CBF and cerebral blood volume (CBV) in 15 patients without contralateral carotid stenosis undergoing CEA. They observed a correlation between increased CBV and increased CBF after CEA on single-photon emission computed tomography (SPECT) and magnetic resonance imaging (MRI), with signs of hyperperfusion in seven patients (47%). Two of these seven patients developed

CHS, whereas none of the eight patients with normal CBV developed CHS. In this study, elevated preoperative CBV was the only significant independent predictor of post-CEA hyperperfusion.

The endothelial damage caused mainly by chronic hypertension in the small arteries may also be related to cerebral autoregulation (Skydell et al, 1987). In fact, some authors relate a history of stroke with a greater risk of CHS (Chamorro et al, 2000; McCabe et al, 1999).

Another important but not essential factor associated with CHS is high blood pressure. High blood pressure is the only factor we can treat, so it has become the principal target for prevention and treatment. Indeed, the pathophysiology of CHS is similar to that of hypertensive encephalopathy in which the blood-brain barrier ruptures as a consequence of severe hypertension. Furthermore, histologic changes like fibrinoid necrosis and petechial hemorrhage also occur in both hypertensive encephalopathy and CHS (Bernstein et al, 1984; Mansoor et al, 1996; Schwartz 2002; Vaughan & Delanty, 2000).

The mechanisms by which BP increases after carotid revascularization are poorly understood. The baroreceptor reflex might break down after receptor denervation after CEA or CAS, and hypertension accompanying this feature might increase cerebral perfusion which is more evident after bilateral carotid surgery (Ahn et al, 1989; Bove et al, 1979; Timmers et al, 2004) and is reported in 19% to 64% after CEA.

The stimulation of these baroreceptors in the carotid bifurcation during angioplasty can cause transient bradycardia and hypotension that can be followed by rebound hypertension. Other phenomena proposed to explain the high blood pressure include increased norepinephrine levels probably related to cerebral edema and increased intracranial pressure, the release of vasoactive neuropeptides, the use of anesthetic drugs, and perioperative stress (Bajardi et al, 1989; Benzel & Hoppens, 1991; Macfarlane et al, 1991; Towne JB & Bernhard, 1980; Skydell et al, 1987; Skudlarick & Mooring, 1982;).

Another possible mediator of impaired autoregulation in CHS is nitric oxide, which causes vasodilatation and can increase the permeability of cerebral vessels. Increased nitric oxide levels during clamping of the ICA and increased oxygen-derived free radicals produced during the restoration of cerebral perfusion are involved in endothelial dysfunction and deterioration of autoregulatory mechanisms after CEA (Suga et al, 2007). Several authors (Ogasawara et al, 2004; Saito et al, 2007) have reported that the degree of reactive oxygen species production after ischemia and reperfusion during CEA depends on the intensity of cerebral ischemia during ICA clamping.

Reactive oxygen species can play a role in the pathogenesis of post-CEA hyperperfusion, leading to widespread endothelial damage in the ipsilateral cerebral arteries and thereby increasing the risk of ICH in the early postoperative period. Furthermore, administering a free-radical scavenger can prevent CHS, providing additional support for this mechanism (Ogasawara et al, 2004).

Finally, an axon-like trigeminovascular reflex has been implicated in the pathophysiology of CHS (Macfarlane et al, 1991). The release of vasoactive neuropeptides from perivascular sensory nerves via axon reflex-like mechanisms has a significant bearing upon a number of hyperperfusion syndromes.

3. Clinical presentation

The typical clinical presentation of CHS combines symptoms due to ICH and those due to brain damage caused by vasogenic edema. The most common symptoms caused by ICH are

headache, confusion, altered levels of consciousness, and sometimes vomiting. On the other hand, the edema usually manifests as a neurological deficit on the side of the untreated carotid artery, often associated with epileptic activity (seizures, usually starting as partial seizures). Arterial hypertension is the norm in patients that develop symptoms of CHS; however, it is important to remember that bradycardia and hypotension often occur initially after angioplasty due to stimulation of the baroreceptor reflex.

When a patient has symptoms of neurological deficit after angioplasty, the first diagnosis considered is embolic stroke from carotid plaque broken off during the procedure. Thus, CHS can mimic a stroke or transient ischemic attack (TIA), so it is important to take into account symptoms like headache, seizures, and altered mental status that can suggest CHS. Nevertheless, acute neurological deficit accompanied by headache or even seizures is obviously compatible with ICH, which can be ruled out only by neuroimaging.

Neurological deficit due to vasogenic edema is usually transitory, given the absence of ischemic infarction (Bernstein et al, 1984; Piepgras et al 1988; Reigel et al, 1987; Sundt et al, 1981; Solomon et al, 1986). Although the neurological symptoms can vary, the most common are visual or motor deficits and aphasia. Other, rarer, symptoms include psychotic alterations or mild cognitive deficit (Ogasawara et al, 2005).

Seizures are generally partial at first and sometimes become generalized later, although generalized seizures can also occur initially (Ho et al, 2000); in fact, even status epilepticus has been reported up to two weeks after the procedure (Kaku et al, 2004). One third of patients with CHS after CEA have seizures without hemiparesis, another third have hemiparesis without seizures, and another third have both (Bouri et al, 2011).

Curiously, the onset of symptoms after CEA and CAS differs. Symptoms usually do not appear until three to six days after CEA. In contrast, symptoms usually appear within a few hours of CAS. Ogasawara et al (2007) report that the incidence of CHS peaks six days after CEA and 12 hours after CAS. After reviewing 36 studies, Bouri et al (2011) concluded CHS peaks five days after CEA and the latest case occurred after 28 days.

The same is true of ICH, which appears 10.7 ± 9.9 days after CEA and 1.7 ± 2.1 days after CAS, peaking in the first 12 hours. Tan et al (2004) studied the appearance and onset of complications after CAS in 201 patients; they report 10 cases with TIA (4.9%), 5 of which occurred more than 48 hours after the procedure, and 8 strokes (3.9%), 5 of which occurred between 2 and 19 days after the procedure. Curiously, however, these authors found no cases of CHS.

The headache in CHS is usually moderate to severe and throbbing, similar to a migraine headache (Coutts et al, 2003), and it usually affects the same side as the artery treated.

Headache may be the only manifestation of CHS (Connolly 2000; Ouriel et al, 1999; Sbarigia et al, 1993), so occasionally it has been considered a diagnostic criterion. After CEA, headaches are reported in 20% of patients without CHS, in 59% of those with CHS, and in 84% of those with ICH (Bouri et al, 2011).

Postprocedural hypertension is a critical, though not essential, finding associated with CHS (Solomon et al, 1986; Schroeder et al, 1987; Ouriel et al 1999). Bouri et al review (2011) found that the mean systolic BP of CHS cases was 189 mmHg at presentation, and the proportion of patients with severe hypertension was significantly higher in patients who developed CHS after CEA than in those who did not.

Hypotension occurs immediately after CAS in 19% to 51% of patients. It is usually transient and rarely symptomatic, although it lasts longer than 24 hours in nearly 5% of patients. Bradycardia is also common, with an incidence of 3% to 37% in patients administered

prophylactic atropine and of 20% to 60% in series with no use of prophylactic atropine. Increased age, symptomatic lesions, presence of ulceration and calcification, and carotid bulb lesions are significant predictors of bradycardia during CAS (Cayne et al, 2005; Lin et al, 2007; Pappada et al, 2006 & Taha et al, 2008).

Another complication with more dramatic consequences is ICH, which affects less than 1% of patients after CEA and between 0.36% and 4.5% after CAS. Generally, ICH has a poor prognosis, with a 37% to 80% mortality rate and a 20% to 30% risk of poor recovery in survivors after CEA (Piepgras et al, 1988; Connolly 2000) and similar consequences after CAS.

4. Diagnosis

The diagnosis of CHS is based on the initial suspicion arising from the characteristic triad of headache, focal neurological deficit, and seizure after arterial revascularization. The differential diagnosis should include stroke and TIA. Seizures and altered consciousness favor the diagnosis of CHS. After the initial clinical suspicion, neuroimaging plays a crucial role because in addition to ruling out ischemic and hemorrhagic lesions it can reveal characteristic signs of hyperperfusion.

Given the widespread availability of CT, any acute neurological event after revascularization is usually studied with this technique. CT is most useful for ruling out hemorrhagic processes. Given that the initial symptoms of CHS can mimic stroke or TIA, CT can give us clues that argue against an ischemic stroke, because CT findings are usually normal after a TIA and are often normal within hours after a stroke. Diffusion MRI is the technique of choice to rule out acute ischemic stroke; MRI has shown that there are a greater number of embolic lesions up to 48 hours after CAS, although nearly all are asymptomatic (Rapp et al, 2007).

We will comment on two important aspects of neuroimaging studies. First, we will discuss their usefulness in the diagnosis of CHS, as apart from demonstrating typical findings like vasogenic edema (Case 1) they also enable CBF to be quantified (increases in CBF > 100% with respect to baseline values have been related to greater risk of developing CHS). Second, we will discuss the usefulness of these techniques in the evaluation of CVR, the key pathophysiological factor in CHS.

4.1 Diagnosing cerebral hyperperfusion

The imaging techniques that can demonstrate hyperperfusion are single-photon emission computed tomography (SPECT), positron emission tomography (PET), transcranial Doppler (TCD), CT and MRI. According to Penn et al (1995), xenon-enhanced CT is the best method for demonstrating hyperperfusion. Nevertheless, SPECT and TCD are the most common methods in the literature, followed by CT and MRI.

CT in CHS typically reveals ipsilateral sulcal effacement and cerebral edema immediately following the onset of symptoms; these findings are considered indirect signs of hyperperfusion. CT findings early after the onset of symptoms can be completely normal, even when SPECT shows hyperperfusion.

Without doubt, T2-weighted and FLAIR MRI sequences are more precise in demonstrating areas of cerebral edema, and diffusion-weighted MRI makes it possible to rule out hyperacute ischemic lesions.

However, normal findings on MRI do not exclude the presence of CHS. Both MRI and CT enable angiographic maps to be constructed to rule out arterial occlusions and perfusion

maps can show local hyperemia. Karapanayiotides et al (2005) reported no abnormalities on diffusion-weighted MRI in patients with CHS after CEA, ruling out acute ischemia; however, perfusion sequences revealed differences in CBF between the hemispheres.

Hypoperfusion before revascularization and especially hyperperfusion (increase in CBF > 100% with respect to baseline values) after revascularization are conditions that are closely related with CHS. TCD is the method most often used to detect these conditions because it enables variations in CBF to be calculated in real time. TCD has many advantages and multiple indications in cerebral vascular disease (Alexandrov et al, 2010). TCD monitoring can provide direct and real-time information on MCA flow indicative of preoperative cerebral hypoperfusion, CVR, postoperative hyperperfusion, and emboli after CEA and CAS. Moreover, TCD is widely available, noninvasive, and reproducible. It is important to do a baseline study to enable flow velocities before and after revascularization to be compared (Dalman et al, 1999; Jansen et al, 1994).

Asher et al (2003) studied 455 patients undergoing CEA and reported a significant increase in mean ICA flow volume in all patients with CHS during the symptomatic period; moreover, after flow velocities return to normal, the symptoms of hyperperfusion disappear.

Diverse publications about patients undergoing CAS emphasize the role of TCD in detecting hemodynamic changes that make it possible to select patients with greater risk of developing CHS. For example, in one interesting study published recently, Kablak et al (2010) monitored both MCAs before and after CAS, finding a relation between ICH in 3 patients and an increase in peak systolic velocities in both MCAs after CAS. Fujimoto et al (2004) examined the changes in the MCA mean flow velocity measured by TCD before and 4 days after CEA. They reported a significant correlation between changes in mean flow velocity and changes in regional CBF; mean flow velocity increased more than 50% in all cases of CHS.

Some studies have used both TCD and SPECT to assess patients before and after revascularization. Recently, Iwata et al (2011) used these two techniques to study 64 patients and found 9 patients who fulfilled the clinical criteria for CHS. These authors relate CHS with decreased CVR and changes in MCA flow velocity after angioplasty.

Perfusion CT has also contributed to our understanding of CHS. Tseng et al (2009) used CT to study 55 patients with symptomatic stenoses >70% of the ICA, analyzing absolute values of CBV, mean transit time (MTT), and CBF. Three (5%) of 55 patients had CHS after CAS. The only significant factor related to the occurrence of CHS was MTT. An MTT cutoff of 3 seconds distinguished between the occurrence and absence of CHS. MTT prolongation is proportional to the degree of stenosis and decrease in blood flow (Maeda et al, 1999; Lythgoe et al, 2000; Soenne et al, 2003). Findings of decreased CBF together with MTT prolongation and a slight increase in CBV indicate that blood vessels are dilated, thus confirming that the autoregulation mechanism is impaired.

Several authors have examined the role of CT and MRI in demonstrating hyperperfusion (Adhiyaman & Alexander 2007; Imai et al 2005; Sundt et al, 1981). Multislice dynamic susceptibility contrast MRI or perfusion-weighted MRI can also be used in the preoperative assessment of CBF (Fukuda et al, 2007; Wiart et al 2000). Perfusion sequences, however, are not quantitative and can only help in the absence of contralateral ICA stenosis.

PET has also provided valuable information about CHS. Matsubara et al (2009) used PET to study patients before and after angioplasty. They found that the vascular reserve tended to improve gradually after CAS, while CBF, cerebral perfusion pressure, and cerebral

metabolic rate of oxygen increased rapidly and peaked soon after CAS. These results suggest that a large discrepancy between rapidly increased CBF, perfusion pressure, and a small increase in vascular reserve in the acute stage after CAS could cause CHS.

Cerebral oxygen saturation can serve as an indirect measure of CBF. Clinically, regional cerebral oxygen saturation can be monitored using transcranial near-infrared spectroscopy, which enables noninvasive continuous real-time detection of changes in the ratio of oxyhemoglobin to deoxyhemoglobin in the frontal cortex, an indirect measure of cerebral oxygenation. Recently, a strong linear correlation was reported between increased transcranial regional cerebral oxygen saturation and increased CBF after CEA (Ogasawara et al, 2003). When compared with SPECT, the sensitivity and specificity of transcranial regional cerebral oxygen saturation for the detection of hyperperfusion were 100%. Transcranial near-infrared spectroscopy can demonstrate decreased cerebral oxygenation resulting from ICA clamping (Beese et al, 1998; Duncan et al, 1995; Kirkpatrick et al, 1995; Samra et al, 1996) and can predict post-CEA CHS. Matsumoto et al (2009) used transcranial near-infrared spectroscopy to study 64 patients undergoing CAS, two of whom developed CHS (diagnosed by increased CBF at SPECT the day after treatment). An increase in regional oxygen saturation > 24% three minutes after revascularization was associated with the development of CHS (with impaired CVR). In contrast, in patients without CHS, the normal upper limit of the change in regional oxygen saturation three minutes after revascularization was 10 %.

Oxygen saturation should be monitored for a prudential time because bradycardia and hypotension often occur with CAS and can occasionally lead to low initial values. As occurs in many studies, the small number of patients with CHS in this study does not allow clear conclusions to be drawn; nevertheless, given that transcranial near-infrared spectroscopy is noninvasive and easy to perform, it should be considered for monitoring patients at risk for CHS.

Alternative methods have been applied to identify risk factors for postoperative hyperperfusion, but their utility is not yet clearly established. Electroencephalography is used for neurological monitoring during CEA, but it is of low predictive value for CHS (Reigel et al, 1987). Nicholas et al (1993) reported that a postoperative increase in ocular blood flow greater than 204% measured by ocular pneumoplethysmography is associated with a high risk for CHS.

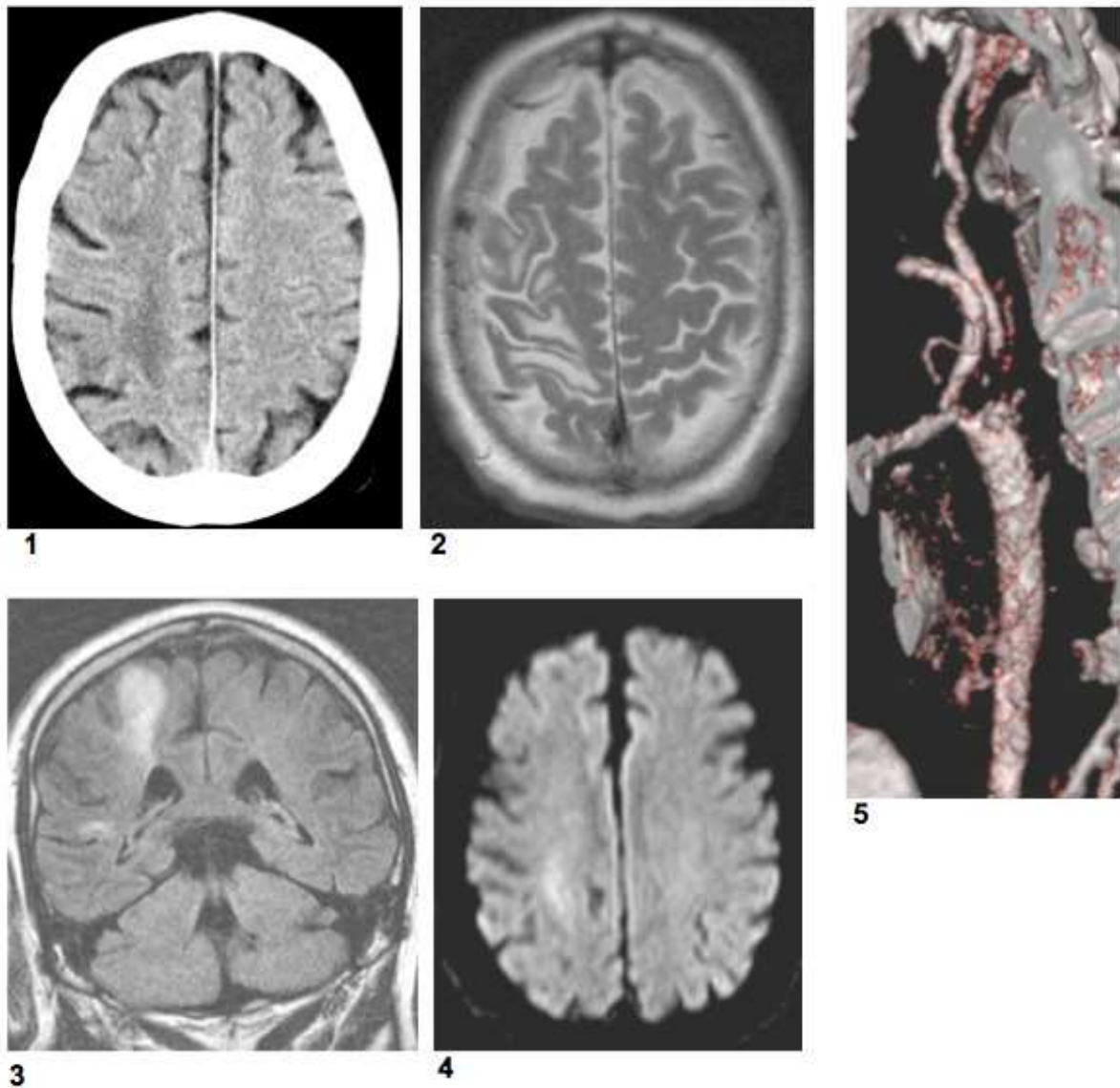
4.2 Diagnosing hemodynamic reserve

One strategy that is key to preventing CHS is the study of CVR, which is usually done by TCD and SPECT.

SPECT is sensitive for recognizing CHS, differentiating between ischemia and hyperperfusion, and identifying patients at risk for hyperperfusion after CEA (Hosoda et al, 2001; Naylor et al, 2003; Sfyroeras et al, 2006). Several studies using SPECT have demonstrated that decreased CVR using acetazolamide is a significant predictor of post-CEA hyperperfusion (Ogasawara et al, 2003; Yoshimoto et al, 1997).

Fewer studies have focused on patients undergoing CAS. Kaku et al (2004) published one of the first studies about predicting CHS with nuclear medicine techniques in patients undergoing CAS.

They measured resting CBF and CVR to acetazolamide to evaluate CVR, using split-dose [¹²³I] iodoamphetamine SPECT before and 7 days after CAS in 30 patients with critical carotid stenosis. The 3 patients with hyperperfusion all had impaired CVR and asymmetrical carotid



Case 1

A 71-year-old man with symptomatic pseudo-occlusion of the right ICA had a seizure with Todd's paralysis six days after CEA. Neuroimaging showed vasogenic edema (1- CT, 2- Axial T2-weighted MRI, 3-Coronal FLAIR MRI, 4- Axial diffusion-weighted MRI, 5- CT angiography)

CBF. These authors determined that pretreatment resting CBF value, degree of carotid stenosis, and interval from the onset of ischemic symptoms were not significant risk factors. However, the high cost and limited availability of SPECT preclude its clinical use.

TCD has numerous advantages in diagnosing hemodynamic reserve: it is noninvasive, relatively simple, cheap, and reproducible, and it is risk free when the breath-hold and hyperventilation method is used. TCD enables CVR to be calculated using stimuli like hypocapnia (induced by breath holding or by inhalation of CO₂) or acetazolamide.

The response to these stimuli reflects the cerebral autoregulation capacity and thus makes it possible to determine which patients have a high risk of developing CHS (Sfyroeras 2006,

2009). However, TCD has some drawbacks. The absence of a cranial window makes TCD impossible in 15% of patients, mainly elderly women. Moreover, TCD is operator-dependent and the results also depend on anatomic variants, the degree of collateralization, and contralateral ICA occlusion or stenosis.

Table 1 shows the formulas to calculate the CVR using breath-holding and CO₂ inhalation or acetazolamide. In the breath-hold method, patients are asked to hold their breath for at least 30 seconds during continuous MCA flow velocity monitoring; normal values are 1.2 +/- 0.6% / sec. In the hyperventilation/ breath-holding method, patients are asked to hyperventilate for 40 seconds followed by a breath-holding phase of at least 30 seconds. Flow velocity values under maximal hyperventilation and hypoventilation are compared; a relative difference greater than 15% argues against relevant impairment of CVR.

Breath-holding index (BHI)	
BHI =	$\frac{V \text{ apnea} - V \text{ baseline}}{V \text{ baseline} \times T \text{ apnea}} \times 100$

CO ₂ inhalation test/acetazolamide test	
CVR =	$\frac{\text{CO}_2/\text{acetazolamide} - V \text{ baseline}}{V \text{ baseline}} \times 100\%$

Table 1.

Chang et al (2009) used functional MRI to assess baseline CVR and changes in CBF after CAS. Although this small series of 14 patients had no cases of CHS, this study revealed that after CAS early CBF changes on the lesion side are more prominent in patients with impaired CVR. Therefore, baseline CVR might predict early CBF increase after CAS. New MRI techniques like dynamic susceptibility contrast MRI or perfusion-weighted MRI can determine CVR (Wiart et al, 2000).

5. Incidence and risk factors

This section reviews the incidence of CHS after CAS in the most relevant series included in PubMed from 2003 to April 2011. We focus on three aspects of CHS: extracranial CAS, angioplasty of intracranial arteries (including the ICA) with or without stenting, and cerebral hemorrhage, the most-feared complication of this treatment.

5.1 Extracranial carotid angioplasty

Most articles about CHS refer to CAS or CEA of the ICA because occlusive disease is more prevalent in these arteries than elsewhere. Bouri et al (2011) reviewed 36 studies of patients undergoing CEA and found 1% incidence of CHS and a 0.5% incidence of ICH.

In many CAS series, patients referred for endovascular treatment comprise a high-risk cohort of suboptimal candidates for conventional surgical management. This might partially

explain the greater number of complications, including CHS, in patients treated with CAS. Furthermore, the endovascular procedure is performed with stricter antithrombotic management, with anticoagulation and dual antiplatelet treatment that might lead to a higher rate of hemorrhagic events, although not all authors agree with this hypothesis (Abou-Chebl et al, 2004; Meyers et al, 2000). Table 2 lists risk factors for CHS, broken down into modifiable and non-modifiable factors.

Although procedural and midterm complication rates of CAS in elderly patients are acceptable, high age seems to be a possible risk factor for CHS (Kadkhodayan et al, 2007). Other risk factors often mentioned in the literature are severe (>90%) ipsilateral stenosis, impaired collateral flow secondary to advanced occlusive disease in other extracranial cerebral vessels or an incomplete circle of Willis, perioperative and postoperative hypertension, and the use of antiplatelet agents or other anticoagulants (Chamorro et al, 2000; Reigel et al, 1987; Sfyroeras et al 2008; Zahn et al, 2007).

Abou et al (2004) report a series of 450 patients undergoing CAS where 5 (1.1%) developed CHS, 3 of them developed ICH (0.67%), and 2 of them (0.44%) died. All the patients that developed CHS had stenoses >90%, contralateral stenoses >80%, and longstanding preprocedural hypertension. The authors calculate that in patients with these three conditions, the risk of developing CHS was 16%. Only 5.8% of the patients that did not develop CHS met these three criteria. The low incidence of CHS in this series might be due to the fact that CHS was not diagnosed in cases with headache and vomiting. Two of the cases of ICH appeared a few days after CAS and only one occurred immediately after the procedure.

Ogasawara et al (2007) published a series of 4494 patients revascularized with CEA or CAS. Of the 1596 patients treated with CEA, 30 (1.9%) developed CHS and 6 of these developed ICH (0.4% of the total). Of the 2898 patients treated with CAS, 31 (1.1%) developed CHS and 21 (0.7% of the total) of these developed ICH. In the group of patients treated with CEA but not in those treated with CAS, poor BP control after revascularization correlated with CHS. CHS and ICH occurred significantly earlier after CAS than after CEA. The difference between the two procedures in terms of the timing of CHS onset may be explained as follows. First, the higher incidence of embolisms after CAS (Roh et al, 2005) might explain how a hemorrhagic transformation could occur after the resolution of the embolism in the tissue that was damaged; from a pathophysiological point of view, however, this would represent hemorrhagic infarction due to reperfusion rather than CHS. Second, the higher incidence of bradycardia and hypotension after the stimulation of the carotid baroreceptors during CAS (Mendelsohn et al, 1998; McKeivitt et al, 2003; Qureshi et al, 1999) can favor cerebral ischemia and CHS after severe rebound hypertension (Abou-Chebl et al, 2007). In an earlier publication (Ogasawara et al, 2003), these authors suggested that SPECT findings of hyperperfusion continuing at least three days after revascularization predisposes to CHS.

In an excellent review of 9 studies of CAS comprising a total of 4446 patients, Moulakakis et al (2009) found the incidences of CHS and ICH were 1.16% (range, 0.44% - 11.7%) and 0.74% (range, 0.36% -4.5%), respectively. Table 3 shows the incidence of CHS and of ICH in the largest series published before 2010, including series of patients undergoing angioplasty of intracranial arteries.

In order to document the incidence of CHS after CAS and to determine possible predisposing factors, Sfyroeras et al (2009) studied 29 patients with CT, MRI, TCD including assessment of CVR, and SPECT before and after the procedure. A total of 5 patients developed adverse neurological events. Two of them developed CHS (6.9%); both had

exhausted CVR in the preoperative TCD examination. All studies that investigate CVR before treatment have found a relation between impaired CVR and the risk of CHS.

Brantley et al (2009) studied 482 patients, 7 (1.45%) of whom developed CHS after CAS. None had an ICH and all recovered within 6 to 24 hours. All had been classified as high risk for CEA, and CHS was more common in those with a previous TIA. The absence of ICH was probably related to the fact that 64% of the patients had asymptomatic stenoses. These authors found no significant relation between CHS and risk factors reported in other series like hypertension, high-grade ICA stenosis, and contralateral disease. The postprocedural BP in the CHS cohort tended to be higher than in the other patients, but this difference did not reach statistical significance.

Potential risk factors for CHS	
Modifiable	Not modifiable
High blood pressure	Diminished CVR
Excessive administration of antithrombotic drugs	Hypertensive microangiopathy
Simultaneous revascularization of multiple vessels	Recent minor stroke
Use of high doses of volatile halogenated hydrocarbon anesthetics	Age >70 years
Recent (<3 months) contralateral CEA	High grade carotid artery stenosis
	Incomplete circle of Willis
	Contralateral carotid occlusion
	Poor collateral flow
	Increase in regional cerebral oxygen saturation >24%
	Diabetes mellitus, Hypertension
	Increase in perfusion >100%
	Preoperative hypoperfusion

Table 2.

Grunwald et al (2009) report a series of 417 patients treated with CAS in whom BP was meticulously controlled during the first 24 hours; furthermore, MRI was performed before and after the procedure in 269 cases. The mean degree of carotid stenosis was 87%, and 65% of the patients were symptomatic. Of the 10 (2.4%) patients who developed CHS, seven had excessive small vessel disease with old territorial infarcts or freshly demarked lesions. Small vessel disease is considered a risk factor for CHS because it impairs the capacity of these arteries to contract. Curiously, none of these patients had severe hypertension. In three cases, ICH occurred within a few hours of CAS, and all of these had extensive microvascular changes and impaired collateral blood flow due to high-grade stenosis (>80%) of the contralateral ICA. However, 23% of the patients that did not develop CHS also had high-grade stenosis of the contralateral ICA. On MRI, all had increased signal intensity in the subarachnoid space on the same side as the stented ICA, which resolved within 3-5 days.

Curiously, this study was unable to demonstrate a relation between CHS and factors like postprocedural hypertension, advanced age, degree of ipsilateral stenosis, or contralateral disease.

Regarding prior stroke as a risk factor for CHS, many authors have found that diseases like diabetes mellitus or longstanding pre-existing hypertension in which microangiopathy affects the endothelium of small vessels predispose to hyperperfusion and CHS (Chamorro et al, 2000; McCabe et al, 1999; Naylor et al, 2003; van Mook et al, 2005).

Tietke et al (2010) analyzed the outcomes of 358 patients treated with CAS using small closed-cell stents without distal protection. The peri-interventional and 30-day mortality/stroke rate was 4.19% (15/358). These events included 3 deaths, 5 CHS (comprising one death by a secondary fatal ICH), one SAH and 7 ischaemic strokes. All but one of the patients with CHS had an initial stenosis of >90%; the remaining patient had an initial stenosis of 50% to 70% and was the only one with CHS without ICH. The patient who died was the only woman with CHS and she also had an occluded contralateral ICA. Most complications occurred in initial symptomatic patients (5.36%).

The risk of CHS related to the type of protection (proximal or distal) has not been thoroughly studied. Pieniazek et al. (2004) compared the complications in 135 patients undergoing CAS, 42 with proximal protection and 93 with distal protection, but only one case of CHS developed.

Bilateral carotid stenoses are generally treated in two separate stenting procedures to minimize hemodynamic impairment from stimulation of the carotid sinus baroreceptor reflex (severe bradycardia, hypotension) and the risk of CHS. As we explained in section 2 (pathophysiology), the baroreceptor reflex might break down after receptor denervation after CEA or CAS; this is more common after bilateral carotid surgery, and accompanying hypertension might increase the risk of CHS.

Author / Year	Patients	CHS (%)	ICH (%)
Meyers / 2000	140	7 (5%)	1 (0.7%)
Coutts / 2003	44	3 (6.8%)	2 (4.5%)
Abou / 2004	450	2 (0.44%)	3 (0.67%)
Kaku / 2004	30	1 (3.33%)	0%
Imai / 2005	17	2 (11.7%)	2 (11.7%)
du Mesnil de Rochemontn / 2006	50	1 (2%)	0%
Kablak-Ziembicka / 2006	92	2 (2.2%)	2 (2.2%)
Abou / 2007	836	8 (0.96%)	3 (0.36%)
Ogasawara / 2007	2989	31 (1.1%)	21 (0.7%)
Sfyroeras / 2008	29	2 (7%)	0%
Brantley / 2009	482	7 (1.5%)	0%
Grunwald / 2009	417	7 (1.7%)	3 (0.7%)
Tietke et al (2010)	358	4 (1.1%)	1 (0.27%)
Karkos et al (2010).	316	10 (3%)	0%

Table 3. Incidence of hyperperfusion syndrome, and intracranial hemorrhage after CAS in the reviewed series from 2003 to 2010

Few studies have addressed the subject of simultaneous bilateral CAS. Henry et al. (2005) reported a series of 17 patients who underwent simultaneous bilateral CAS and 40 patients who underwent bilateral CAS in a staged manner (among these 40 patients 10 underwent the second procedure 24 hours after the first, while the other 30 underwent the second procedure from 2 days to 2 months after the first). Two cases of CHS occurred, one each group, although the patient in the simultaneous treatment group who developed CHS died. Lee et al. (2006) found no CHS in a series of 27 patients who underwent bilateral CAS. Diehm et al. (2008) studied patients treated with bilateral CAS with at least one month between procedures and reported no significant differences in complications compared to patients treated with unilateral CAS.

An interesting study that deals with pseudo-occlusive carotids was published by Choi et al (2010). These authors analyze the outcome after CAS in 48 patients with nearly occlusive stenosis of the ICA. The procedural success rate was 98% and a good outcome at six months (modified Rankin scale ≤ 2) was achieved in 44 patients (92%). Four (8%) patients developed CHS.

Another interesting article was published by Karkos et al (2010). They studied the complications in the first 30 days in 333 angioplasties in 316 patients, 35% of whom had symptomatic carotid disease. Perioperative neurological events included stroke in 6 patients (1.8%), TIA in 15 (4.5%), and CHS in 10 (3.0%). The incidence of CHS did not differ between the group of patients with symptoms and those without. Bradycardia was noted in 48 patients (14%) and hypotension in 45 (13%), and two of these patients (0.6%) required admission to the intensive care unit for hemodynamic instability. Curiously, the only factors related to increased morbimortality were hyperlipidemia and current or previous smoking.

5.2 Angioplasty in intracranial arteries

As is to be expected, fewer studies have addressed CHS in relation to intracranial angioplasty because this procedure is newer than angioplasty in extracranial arteries. In this section, we will discuss the most interesting series and cases of patients treated with this technique. In 2000, Meyers et al reported the first SAH due to stenosis of the intracranial vertebral artery. In their series of 140 patients treated with CAS (including 10 intracranial carotids, 14 intracranial vertebral arteries, 4 basilar arteries, and 1 MCA), the incidence of CHS was 5% (7 of 140 patients, 5 carotids and 2 vertebral arteries), one with ICH and another with SAH. Importantly, six patients (85%) were symptomatic with crescendo TIAs before treatment, and these symptoms were probably related to impaired CVR. The first case of CHS with ICH after intracranial MCA angioplasty was reported by Liu et al in 2001. One of the first series of patients undergoing intracranial CAS was published by Terada et al (2006). These authors reported 106 procedures in 99 patients (57 patients had intracranial ICA stenosis, 23 had MCA stenosis, and 19 had vertebrobasilar stenosis). The ICA stenosis involved the petrous or cavernous in 47 cases (24 patients were treated with angioplasty and 23 with stenting). Four hemorrhagic complications occurred in 106 procedures. One patient had SAH and the other 3 cases had the following characteristics: severe stenosis with poor collateral flow, low perfusion with CVR damage on SPECT, appearance of ICH between 30 minutes and 16 hours after the procedure, and patient age greater than 70 years. The rate of ICH directly related to CAS was 3%. In two of three cases, CHS was strongly suspected from the SPECT findings. In the nonhemorrhagic group, hemodynamic compromise was found in 27 of 47 (57%) patients.

It is important to remember that hemorrhage caused by vessel injury is also a possible mechanism of hemorrhagic complications. For instance, in the patient with SAH in Terada et al (2006) studies, wall dissection, perforation of the vessel wall by the guidewire, or rupture of a tiny aneurysm located at the distal part of ICA were not completely ruled out. Rezende et al (2006) reported a case of CHS after stenting for intracranial vertebral stenosis. They point out the significant hemodynamic component due to the absence of the contralateral vertebral artery and collateral supply from the carotid territory.

More recent articles about intracranial angioplasty show more promising results. Guo et al (2010) implanted 53 self-expanding stents with a technical success rate of 98%. Complications included SAH (1.9%) and occlusion (3.8%), but there were no cases of CHS. Zhang et al (2008) reported the first case of ICH after CAS in both vertebral arteries with stenosis >90%. The flow velocity of both vertebral arteries measured by TCD increased more than 100% and high BP coincided with the abrupt onset of ICH three hours after the procedure.

In conclusion, the factors involved in the development of CHS after intracranial procedures seem similar to those involved in extracranial procedures, and the results of intracranial angioplasty are very promising.

5.3 Intracranial hemorrhage after angioplasty

ICH is the severest form of CHS and it has the worst prognosis (Case 2). The low incidence of ICH and the small number of patients in the various series reported precludes clear conclusions about the risk factors involved, although presumably they are the same as those involved in CHS. The first question is whether ICH is an extreme consequence of CHS or whether it has a distinct pathophysiology. Numerous mechanisms are possible: CHS, hemorrhagic diathesis caused by antiplatelet and anticoagulation therapy after stenting, hemorrhage around or in a recent infarction or other associated lesion (including hypertensive ICH), or rupture of an intracranial aneurysm.

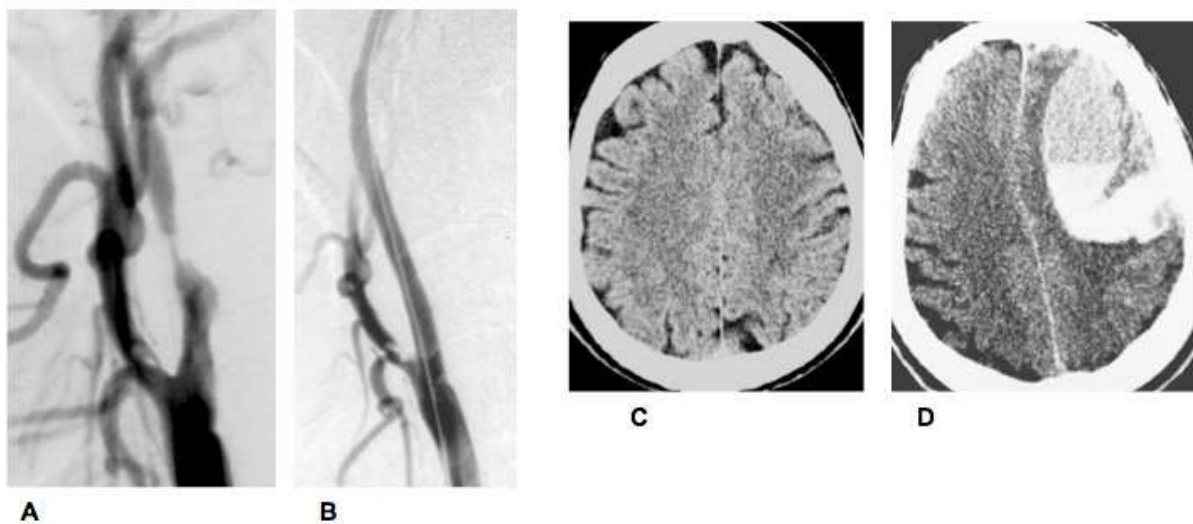
In an interesting article published in 2003, Coutts et al try to narrow the definition of CHS. After studying 129 patients treated with CEA and 44 treated by CAS, these authors postulate that three different syndromes can occur in relation to hyperperfusion: acute focal edema, acute hemorrhage, and delayed classic presentation described for Sundt et al (1981). One of their patients had ICH three hours after CAS in the absence of high BP or symptoms suggestive of hyperperfusion. Other authors like Buhk et al (2006) argue for the existence of two distinct syndromes: first, classic CHS, in which symptoms of ipsilateral, frontotemporal, or retro-orbital headache, neurological deficit, and sometimes seizures typically begin between the fifth and seventh days after revascularization, and second, a more dramatic clinical presentation with ICH considered as damage due to reperfusion (Imparato et al, 1984; Takolander & Bergqvist 1983). In many of the cases published, ICH occurred within a few hours of the procedure and predominantly affected the basal ganglia; furthermore, all the patients in these cases presented with a high-grade stenosis. Therefore, the pathophysiology of this type of ICH might differ from that of CHS, being closer to that of hypertensive ICH, in this case due to rupture of small perforating arteries in the basal ganglia after acute exposure to suddenly normalized perfusion pressure after angioplasty of a high grade stenosis.

Brantley et al (2009) reported a patient with a nearly occlusive ICA stenosis who developed a fatal ipsilateral ICH immediately after the intervention; ICH was due to hemorrhagic conversion of a prior stroke.

The incidence of ICH after CEA in the series published ranges between 0.2% and 0.7% (Piepgras et al, 1988; Pomposelli et al, 1988; Solomon et al, 1986; Wilson & Ammar, 2005), whereas the incidence of ICH after CAS is higher (Timaran et al, 2009), reaching 5% in some series.

Schooser et al (1997) reported the first case of ICH after CAS, a 59-year-old woman with severe stenosis of the left ICA who developed putaminal hemorrhage on the third day after the procedure. CT showed an ipsilateral border zone infarction.

McCabe et al (1999) reported the first fatal case of ICH after CAS, a man with severe stenosis who developed ICH within hours of CAS without any prodromes. Mori et al (1999) reported a similar case in which ICH affected the basal zones with ventricular and subarachnoid extension. Both cases had signs of microangiopathy, which is associated with increased risk of ICH (Chamorro et al, 2000; McCabe et al, 1999).



Case 2

- 1- Angiogram showing 95% stenosis of the left ICA in a patient with occlusion of the right ICA.
- 2- Angiogram after left CAS.
- 3- No lesions were discernible on the pre-treatment CT .
- 4- CT 24 hours later shows extensive hematoma in the left frontal lobe (Courtesy of Dr. Carlos Castaño).

Tan and Phatouros (2009) reviewed 170 patients treated with CAS, 4 (2.3%) of whom developed CHS, one of these with cerebral edema, one with petechial hemorrhage, and two with ICH, which was fatal in one case. All developed CHS within six hours of the procedure and all had stenoses of the internal carotid >95%. Both patients who developed ICH had been treated within three weeks after an ischemic event.

Morrish et al (2000) observed a 4.4% incidence of ICH after 104 CAS in 90 patients; the mean ICA stenosis was 95% in those who developed ICH. In two of the patients, who died, ICH involved the basal ganglia. In this series, the incidence of ICH may have been increased due to a high dose of heparin and the absence of distal protection, given that recent ischemia is a risk factor for ICH.

Matsuo et al (2000) reported two cases of ICH, one of which affected the basal ganglia the day after CAS. The fatal ICH reported by Abou et al (2004) appeared at the level of the basal ganglia one hour after CAS. Finally, the series of 161 patients reported by Koch et al (2002) included a single case of fatal ICH after CAS in a severely stenosed ICA. Kablak et al (2010) reported 3 (1.4%) cases ICH among 210 patients, one of whom had SAH. In their study, increased systolic velocity in both MCAs was a clear risk factor, and one of the three patients had occlusion or severe stenosis of the contralateral carotid.

In addition to impaired CVR, the most widely accepted risk factors are insufficient intracranial collateralization and signs of cerebral microangiopathy. We know that hypertensive encephalopathy does not consist only of periventricular demyelination but possibly also includes small areas of perivascular hemorrhage that can be associated with higher risk of developing ICH. It also seems that the severity of the stenosis plays an important role, as most patients in the literature have severe stenosis.

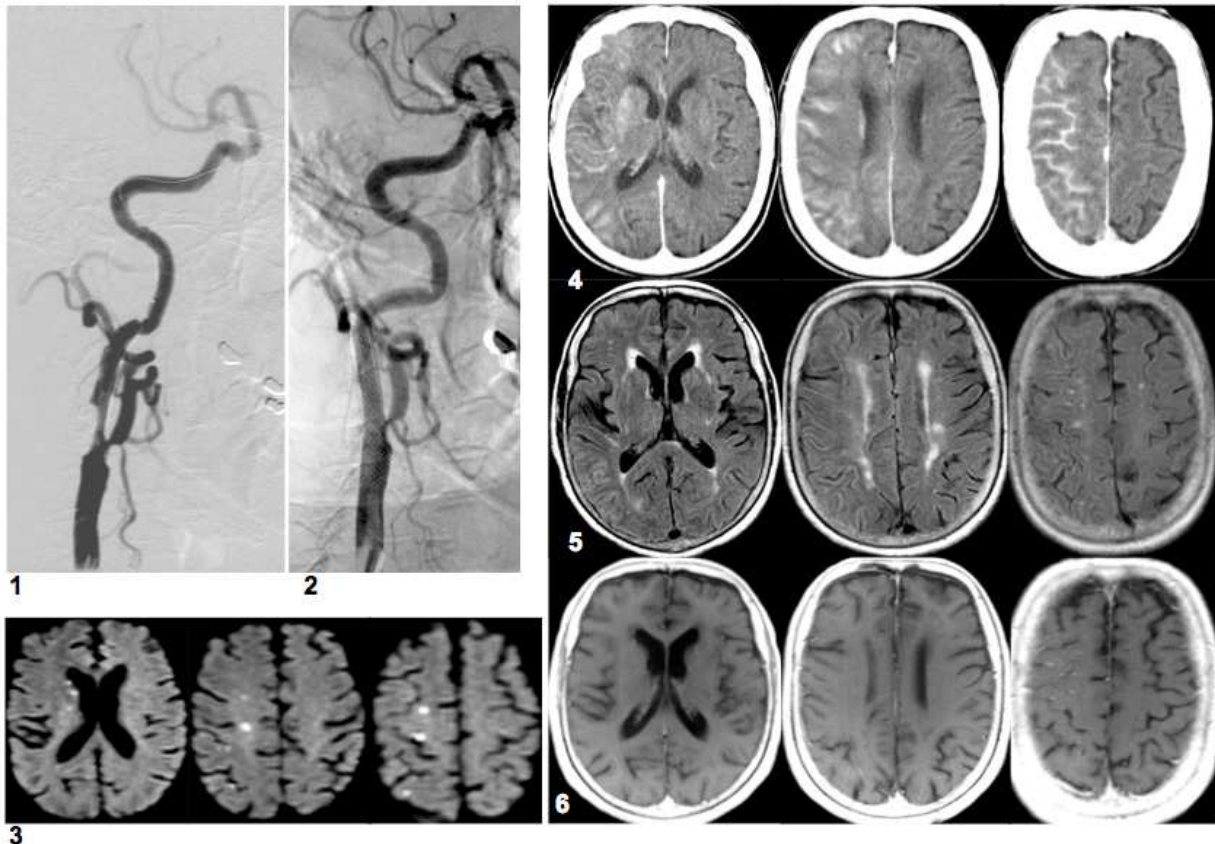
6. Contrast-induced encephalopathy

Neurotoxicity from contrast agents is a rare but well-known complication of diagnostic and therapeutic procedures that employ these agents.

Leptomeningeal enhancement is often reported after CAS due to the abrupt increase in blood flow even when this does not cause symptoms (Wilkinson et al, 2000). Nevertheless, some authors purport that this phenomenon represents the extravasation of contrast material toward the subarachnoid space; Bretschneider and Strotzer (2000) reported 11 cases, some of which were related to hypoxic brain damage. Ekel et al (1998) reported a case of contrast enhancement mimicking SAH, and Mamourian et al (2000) used an animal model to demonstrate that contrast material can cross into the cerebrospinal spinal fluid in sufficient concentration to alter the appearance of the subarachnoid space on MRI. Dargas et al (2001) reported a case of contrast-induced encephalopathy after CAS in an 82-year-old man with a TIA and 90% stenosis in the right carotid. Immediately after CAS, this patient presented confusion and left hemiparesis in the territory of the right carotid. CT showed marked cortical enhancement and edema of the right cerebral hemisphere. The patient improved rapidly and by day 2 was completely recovered; MRI found no cortical edema and normal sulci.

Canovas et al (2007) published a case of extravasation of contrast material immediately after the rupture of the balloon in a woman with a very calcified plaque (Case 3) in whom the pressure of the balloon reached 8 atmospheres. The pressure of the balloon probably magnified the hemodynamic effect, making the extravasation of the contrast material very aggressive and giving rise to a clinical picture identical to an embolic stroke of the MCA. As in other cases reported in the literature, this patient's condition improved and the imaging findings were normal after 48 h.

Contrast-induced encephalopathy should be differentiated from the classical CHS described Sundt et al (1981), although it probably has a similar pathophysiology. A high dose of contrast agent may result in acute breakdown of the blood-brain barrier, allowing the contrast material to enter the brain and resulting in the acute development of a dramatic clinical presentation. The higher osmolality of ioxaglate compared with blood may in turn produce fluid extravasation and cerebral edema. The prognosis is usually excellent, as is evidenced by other recently published cases occurring after endovascular procedures (Guimaraens et al, 2010, Fang et al, 2009; Paúl et al, 2009).



Case 3

- 1- Angiogram before left CAS showing 70% stenosis of the left ICA
- 2- Angiogram after left CAS
- 3- Axial diffusion-weighted MRi showing ipsilateral silent ischemic lesions
- 4- CT with extravasation of the contrast
- 5- Axial FLAIR MRi
- 6- Axial T1-weighted MRi

7. Prevention and treatment

It is crucial to identify patients with risk factors for developing hyperperfusion so that preventive measure can be taken during and after revascularization. In the previous section, we discussed the factors most commonly considered to increase this risk, and in this section we discuss the most interesting preventive strategies.

There is a consensus that the most important risk factors are severely impaired CVR and deficient collaterality (severe ipsilateral stenosis, impaired collateral flow, occlusive disease in other extracranial cerebral vessels, and incomplete circle of Willis). Other proposed factors include advanced age, perioperative and postoperative hypertension, and the use of antiplatelet agents or other anticoagulants. Thus, we should concentrate our efforts on the factors in which we can intervene. Regarding preventive measures before the procedure, we will discuss the assessment of CVR as the primary measure and we will also examine the usefulness of assessing the supra-aortic trunks and the circle of Willis. Regarding preventive measures during and after the procedure, we will focus on detecting cerebral

hyperperfusion and thus on the importance of strict, prolonged BP control and appropriate antithrombotic management.

As we discussed in the Diagnosis section, various options are available for assessing CVR. Probably the most widely available option is TCD, which has many advantages and enables us to measure cerebral flow at rest and under certain stimuli (breath-holding, inhalation of CO₂, intravenous acetazolamide administration). The simplest and most noninvasive TCD test is breath-holding with or without hyperventilation (see the Diagnosis section). Therefore, the first preventive measure that is recommended before revascularization is CVR assessment using TCD (Sfyroeras 2006, 2009).

It would also be advisable to do a thorough MRI study of the supra-aortic trunks and of the circle of Willis as well as a study of the cerebral parenchyma using FLAIR, T2-weighted, and diffusion sequences to detect hyperacute lesions and small-vessel disease, which are also related to increased risk of CHS. As mentioned in the Diagnosis section, CVR can also be assessed by SPECT, CT, and MRI, although these approaches are more expensive and less widely available.

Again, TCD is very useful for monitoring cerebral flow during revascularization procedures. In patients undergoing CEA, TCD can detect increases in MCA flow velocity greater than 100% during the intervention, thus alerting to a situation of risk. Likewise, TCD monitoring during CAS and probably in the hours after the procedure can help select high risk patients (Dalman et al, 1999; Fujimoto et al, 2004; Kablak et al, 2010; Jansen et al, 1994; Iwata et al, 2011; Sfyroeras et al, 2009).

Strict control of hypertension is one of the preventive measures that has received the most attention. Most Investigators recommend strict control of BP in the postoperative period to prevent ICH after CEA (Ahn et al, 1989; Bernstein et al, 1984; Bove et al, 1979; Buhk et al, 2006; Hosoda et al, 2001; Ko et al, 2005; Roh et al, 2005; Safian et al, 2006; Tang et al, 2008) and after CAS, as we will see below.

It has been suggested that even BP in the normal range may be deleterious in patients at high risk for CHS (Piepgras et al, 1988; Ouriel et al, 1999; Jorgensen & Schroeder, 1993).

Regarding strict control of BP, Abou-Chebl et al (2007) published an interesting study that analyzed the presence of CHS and ICH in 836 patients treated with CAS. These authors maintained BP < 140/90 mmHg in patients with lower risk and BP < 120/80 mm Hg in patients with a treated stenosis \geq 90%, contralateral stenosis \geq 80%, and hypertension (i.e., risk factors for CHS). They conclude that comprehensive management of arterial hypertension can lower the incidence of ICH and CHS in high-risk patients following CAS, without additional complications or prolonged hospitalization. The strict control of BP must be maintained until CVR is restored, and this interval varies among patients. Thus, the use of TCD to assess the recovery of CVR can probably help guide antihypertensive therapy (Buhk 2006).

Bando et al (2001) reported a stroke patient with a 90% stenosis of the intracranial left vertebral artery treated with CAS. Immediately after the procedure, hyperperfusion was detected by SPECT and TCD. The patient recovered from CHS quickly after a week's antihypertensive therapy.

Brus-Ramer et al (2010) published an interesting case of a patient treated with CAS who developed signs of hyperperfusion detected by TCD and depicted on angiography as hyperintense punctate foci potentially representing small dilations in the vascular territory of stented arteries. Lowering BP by 40% probably prevented CHS; thus, in high risk patients, aggressive BP management during and after CAS can prevent potentially serious sequelae.

Another aspect that remains to be determined is the most appropriate type of drugs for these patients. In this context, it seems logical that drugs that have no direct effects on CBF and those that give some degree of cerebral vasoconstriction could be beneficial. Drugs like nitroprusside and calcium antagonists that increase CBF should be avoided. The β 1-adrenergic antagonists (beta-blockers) reduce BP with little effect on intracranial pressure within the autoregulatory range, although they can exacerbate the bradycardia that can occur after CAS.

The mixed alpha-adrenergic antagonist and β -adrenergic antagonist labetalol, which has no direct effects on CBF and decreases the cerebral perfusion pressure and mean arterial pressure by about 30% compared with baseline, has successfully been used in CHS after CEA (Halliday et al, 2004). The alpha 2-adrenergic agonist clonidine, which is commonly used after CEA (associated with raised cranial and plasma catecholamine concentrations), has the advantage of decreasing CBF.

General anesthesia is often unnecessary for CAS. However, when general anesthesia is required, it is important to use anesthetics that do not increase CBF. Studies of CBF during surgery have shown that high doses of volatile halogenated hydrocarbon anesthetics may lead to the development of CHS (Skydell et al, 1987). Isoflurane is the volatile anesthetic of choice in neurosurgery because it results in less pronounced vasodilation than other halogenated anesthetics at equipotent doses. The effects of isoflurane on cerebral metabolic rate and autoregulation are dose dependent, with impairment of CVR at high doses. Propofol has been used in patients with CHS, it normalizes CBF, probably because of its effects on cerebral metabolism (Kaisti et al, 2003).

Safety concerns have been raised about the effects of anticoagulants and antiplatelet agents and the risk of ICH following CEA, but no causal link has been found (Ouriel et al, 1999; Penn et al, 1995). Likewise, no association between these drugs and ICH has been found in patients undergoing CAS (Abou et al, 2003), although some studies have reported higher incidences of ICH, probably related to higher than usual doses of anticoagulants (Meyers et al, 2000 ; Morrish et al, 2000).

Levy et al (2002) propose an interesting preventive strategy consisting of performing angioplasty in two phases, with posterior stent collocation. These authors published a series of 8 cases of intracranial vertebral stenosis with good outcomes despite one case of arterial dissection that required stenting. Yoshimura et al (2009) also used two-step endovascular treatment in high risk patients with impaired CVR. These authors first performed angioplasty with a small balloon (3 mm), and once hyperperfusion improved on SPECT about one month later they performed a second, definitive angioplasty with stent placement. None of the 9 patients treated with the two-step approach had problems related with hyperperfusion (one required stenting for a dissected artery), whereas 5 of the 9 patients in the control group had hyperperfusion and one had status epilepticus related to CHS.

Additional efforts to reduce the risk of ICH may include limiting the duration of balloon inflation and employing emboli-prevention devices, as these practices have been related to ischemia with posterior development of ICH (Jansen et al, 1994; Sakaki et al, 1992; Sundt et al 1981).

An important, somewhat controversial factor is the optimal interval between stroke and revascularization. We know that an extensive ischemic lesion represents a greater risk of damage due to reperfusion. Furthermore, classically a six-week interval was recommended to avoid treatment complications. However, studies like the NASCET show that the benefits

of carotid revascularization are greatest in the first two weeks after the event, and the subgroup of patients with less risk for early revascularization are those with small ischemic lesions and mild neurologic impairment (Keldahl et al, 2010). A recent (<3 months) contralateral CEA is an additional potential risk factor for CHS and should also be considered in the timing of surgery (Ascher et al, 2003). Very few studies have addressed the use of CAS in hyperacute strokes, but those that have report good safety outcome (Miyamoto et al, 2008; Setacci et al 2010).

Some authors (Henry et al, 2005; Lee et al, 2006) claim that treatment of both carotid arteries is feasible in carefully selected patients, either in the same procedure or in two procedures separated by an interval of one day; these authors report safety and complication rates comparable to those of large published series in high-risk patients. Nevertheless, careful monitoring of the patient, blood pressure, and heart rate is mandatory to avoid complications related to CHS.

Owing to the presence of free radicals during reperfusion and their relation to post-ischemic hyperperfusion, substances like edaravone have been investigated. Edaravone inhibits lipid peroxidation and vascular endothelial cell injury, improving edema cerebral and tissue injury. Pretreatment with edaravone decreased the incidence of hyperperfusion after CEA as measured by SPECT (Ogasawara et al, 2004).

Once CHS occurs, aggressive measures to lower BP are imperative. As there are no data from randomized trials comparing the optimal perioperative management protocol for patients with CHS due to the rarity of this complication, we must focus on controlling BP, reducing cerebral edema, and, according to some authors, temporarily withdraw antithrombotic therapy. Treatments for cerebral edema include adequate sedation, hyperventilation, and administration of mannitol or hypertonic saline. Evidently, there are no data to support these treatments in CHS. Corticosteroids and barbiturates have also been used in CHS.

There are no available data recommending prophylactic use of anticonvulsant therapy in patients undergoing carotid revascularization; however, in the presence of seizures, treatment with anticonvulsants is indicated.

In conclusion, assessing CVR before treatment and monitoring CBF velocities during and after the procedure can help select the patients who need strict control of BP to prevent CHS. The optimal BP remains to be determined, but BP should be lowered to below the baseline after luminal gain with stenting to prevent secondary injury. Despite the lack of a precise BP target, lowering systolic BP to at least 20% to 30% below baseline values seems critical, particularly in patients with critical stenosis and above all in patients with impaired CVR. Labetalol and clonidine seem to be the most appropriate drugs for BP control in this context.

To determine when discharge is safe after CAS, patients should be divided into two groups. One group includes asymptomatic, hemodynamically stable patients with low comorbidity who could be discharged after 6 h of observation, according to some authors. These patients should be treated using a hemostatic closure device for the arterial puncture. The other group includes older patients with associated comorbidity, mainly those with altered renal function, those that require anticoagulation, and those with altered BP or bradycardia. Finally, it is recommendable to warn the family about symptoms that call for re-evaluation, especially seizures, neurological deficit, and headaches associated with hypertension.

8. Abbreviations

CHS: Cerebral Hyperperfusion Syndrome
 CBF: Cerebral Blood Flow
 CBV: Cerebral blood volume
 CT: Cranial tomography
 CVR: Cerebral Vasoreactivity
 HTA: hypertension
 ICA: Internal Carotid Artery
 ICH: Intracranial Hemorrhage
 MMT: mean transit time
 MCA: Middle Cerebral Artery
 MRi: magnetic resonance imaging,
 PET: Positron Emission Tomography
 TCD: transcranial doppler
 TIA: Transient Ischemic Attack
 SAH: Subarachnoid hemorrhage
 SPECT: Single-photon emission computed tomography

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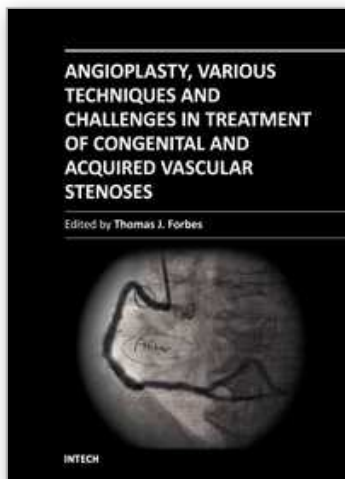
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The field of performing transcatheter interventions to treat vascular lesions has exploded over the past 20 years. Not only has the technology changed, especially in the arena of balloon/stent devices, but the techniques of approaching complex lesions has evolved over the past decade. Lesions that no one would have imagined treating back in the 1990's are now being done routinely in the catheterization suite. This book provides an update on the current techniques and devices used to treat a wide variety of lesions. Though, at first, the outward appearance of the topics appears to be varied, they are all related by the common thread of treating vascular lesions. We hope, by publishing this book, to accomplish two things: First, to offer insight from experts in their field to treat, both medically and procedurally, complex vascular lesions that we frequently encounter. Secondly, we hope to promote increased communication between areas of medicine that frequently don't communicate, between adult interventional cardiologists, pediatric interventional cardiologists, interventional radiologists, and neurosurgeons. Much can be learned from our respective colleagues in these areas which can further our own world of interventions.

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