Starting Strength

The Valsalva and Stroke:
Time for Everyone to Take a Deep Breath

by

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Introduction

Have you heard? *They* are telling us that lifting weights under Valsalva isn't just unsafe, it may soon be *illegal*. Perhaps not by statute, but in terms of the standard of coaching practice and tort liability. Not long ago, one of the denizens of the *Starting Strength* forum sent me an article from an online legal journal published specifically for the fitness community[1]. It describes a malpractice case brought against a trainer and facility for failing to warn a client against the "dangers of Valsalva." The case settled after expert testimony for the defense was undermined by *pivotal new data* showing that hemorrhagic stroke caused by weightlifting under Valsalva "is not rare at all."

The message is clear: warning clients against Valsalva – indeed, actively intervening to prevent Valsalva – is the new professional standard for coaches. Allowing your clients to lift under Valsalva is legally indefensible. And *actually coaching them to do so?* You're just asking for a trail of corpses leading right to the courthouse steps.

There's more. The American Heart Association Scientific Statement on Resistance Exercise discourages lifting under Valsalva[2], although they cite no good clinical evidence of an increased risk of stroke. Exercise science papers are often careful to mention that subjects were not permitted to perform Valsalva[3]. A number of physiologic studies in humans and animals claim to show that lifting under Valsalva predisposes to cerebral hemorrhage[4,5]. And there are case reports of individuals blowing an O-ring in their heads while lifting weights – presumably under Valsalva.

Athletes who engage in serious, programmatic, heavy resistance training will do so under Valsalva – whether they want to or not, as we shall see. And a *very* small number of them do, in fact, suffer hemorrhagic strokes. But is this a cause-effect relationship? Is there either a physiologic or evidentiary basis for claiming that the Valsalva is unsafe under a load? *Are you going to die?*

The answer to the last question is definitely *yes*…although probably not today. The answers to the other questions are rather murkier. Let's try for some clarity, or at least some full-frontal nerdity.

The Valsava Maneuver: Background

Valsalva refers to a Dead Italian Dude named Antonio Maria Valsalva (1666-1723). He was a brilliant physician, surgeon and anatomist. He championed humanitarian reforms in the treatment

of the mentally ill, he helped pioneer anatomic pathology, and he wore one badass wig. His work is remembered in a half-dozen eponyms: the Valsalva antrum of the ear, the aortic sinus of Valsalva, Valsalva's muscle, Valsalva's ligament, tineae Valsalva, and, of course, the Valsalva maneuver. He is also honored eponymously in the ***Valsalva device***, a unit incorporated into space suits so astronauts working outside the spacecraft can pop their ears without taking off their helmets (which would defeat the purpose). This great physician-scientist reportedly died of a stroke in Bologna at the age of 57. It is not clear whether Valsalva stroked under Valsalva, although it seems a good bet that he was not in a squat rack or a spacesuit at the time.

Valsalva's principle interest was *otology*. He was passionate about the ear, and he gave us the first modern description of the Eustachian tube[6]. He was obsessed with the relationship between the ear and the cranial vault. The Valsalva maneuver, which at the time of this writing stands accused of *causing* cerebral pathology, was first described by Valsalva as a way to *treat* cerebral pathology. The idea was that exhaling against a closed glottis would cause "salubrious air" to rise against the meninges (the membranes that enclose the brain) and force pathological intracranial material (pus, blood, gunk, goobers, *schmutz*) through "new foramina" linking the intracranial vault to the ear.

> *I will explain the expurgation of praeternatural cranial matters: he who has inflated his mouth and nose allows air to reach as far as the dura mater... if with occluded mouth and nostrils air is compressed inwardly, this action will extrude sanies from the middle ear, a remedial exercise, to be repeated,* [for] *extrusion of praeter-natural cerebral matter either via the wound, via the nostrils, via the mouth, or via the auditory meatus... **with great benefit**...*
>
> *De aure humana tractatus* —Antonio Maria Valsalva, 1704

Valsalva's "new foramina" appear to have been figments of his fevered Mediterranean imagination, and in fact the cranial vault is not normally in communication with the ear canal (for which you should be grateful). Jellinek has surveyed Valsalva's writings[7] and concluded that his sole interest in the maneuver was its supposed demonstration of these non-existent tunnels between the brain pan and the ear. But this great man might rest easier knowing the maneuver that bears his name was subsequently found to have important implications for brain physiology after all.

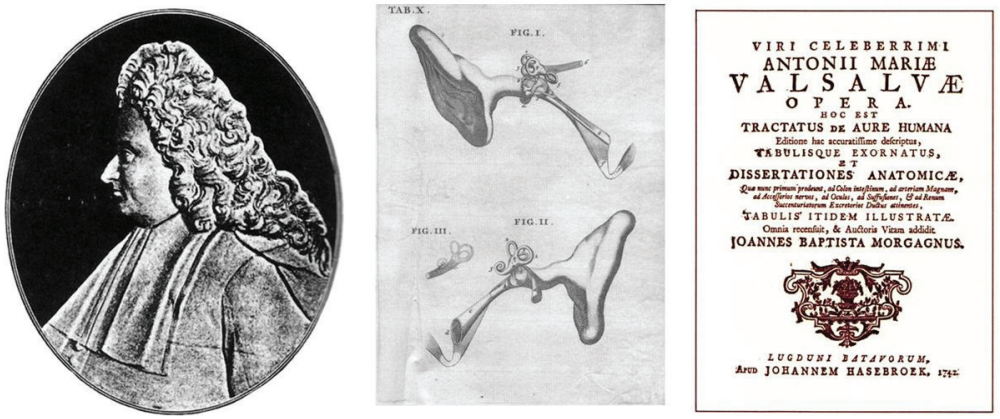

Figure 1. Obligatory Pictures of Old Stuff for Historical Reference. *Left*, Antonio Valsalva and his wig. *Middle,* Valsalva's anatomy of the ear, demonstrating the Eustachian tube. *Right*, First edition of the manuscript in which Valsalva and his wig described the Valsalva maneuver for the expurgation of schmutz from the brain. Images reproduced under Creative Commons license or under doctrine of Public Domain.

Physiology of the Valsalva Maneuver

In the three centuries since Valsalva's death, the physiologic consequences of holding breath against a closed glottis have led to the use of the maneuver in basic research and clinical medicine. In my own practice, I have asked patients to "take a deep breath and bear down" on hundreds if not thousands of occasions – during deliveries (where it has been *pro forma* for centuries), during vascular procedures (to fill the jugular or subclavian with blood and make it easier and safer to insert or remove a central venous catheter)[8], or in the setting of supraventricular tachycardia, to restore sinus rhythm (where it occasionally works, but not nearly as often as we would like)[9]. To date, no patient has ever stroked in front of me during a medical Valsalva. But I'm getting ahead of myself.

The consequences of the Valsalva with immediate relevance for us are its effects on ***thoracoabdominal cavitary pressure***, its effects on ***hemodynamics***, and its effects on ***intracranial pressure***.

That the Valsalva causes a steep increase in thoracic and abdominal cavitary pressures in support of the spine is not an issue of contention[10]. This of course is the principle reason for its use in structural barbell lifts. Holding a large breath against a closed glottis creates a "balloon" of relatively incompressible gas in the thorax, and, via the diaphragm, a corresponding pressure increase in the abdomen. These pressures support the spine and resist vertebral shear forces[11,12,13] in a way that probably protects against orthopedic injury. Although no randomized trial of this hypothesis exists as far as I know, this putative, protective and desirable effect of Valsalva does not seem to be at issue.

The hemodynamic effects of Valsalva deserve rather more detailed attention, and indeed they remain an area of ongoing study. Table 1 summarizes the effects of ***four phases of the Valsalva*** on hemodynamic parameters[14,15].

In ***Phase I***, we take a deep breath and hold it against a closed glottis. This produces an immediate increase in thoracic pressure and a slight increase in left ventricular stroke volume, cardiac output and blood pressure. Because cardiac output is relatively stable or slightly increased, there is little initial change in heart rate.

In ***Phase II***, the "strain" continues. Decreased filling of the heart leads to a fall in stroke volume and cardiac output. The resultant drop in blood pressure triggers compensatory increases in heart rate and systemic vascular resistance, causing the blood pressure to rise again.

When the pressure is released in ***Phase III***, the aorta and great vessels suddenly expand, and cardiac transmural pressures fall. This results in a further decrease in cardiac output and blood pressure. This phase is brief, as within a few heartbeats blood has filled the heart and preload has recovered.

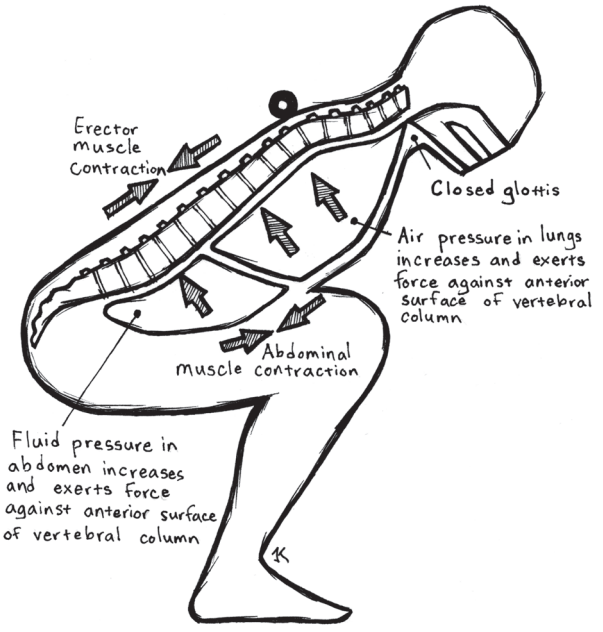

Figure 2. Synergistic effect of Valsalva and spinal erector isometric contraction in the promotion of spinal stability under a load that generates a vertebral shear stress. Reproduced with permission from *Starting Strength: Basic Barbell Training*, 3d Ed 2011, The Aasgaard Company.

PHASE	DESCRIPTION	ITP-IAP	BP	HR	SV	ICP	CPP
I	Initial rise in pressure; "onset of strain."	Increase	Increase	Decreased or Unchanged	Increase	Increase	Decrease?
II	5-20 sec; "continuation of strain."	Increase	Decrease	Increased	Decrease	Increase	Decrease?
III	Release of pressure and cardiovascular "refill."	Decrease	Decrease	Remains increased	Decrease, Increase	Decrease	Increase?
IV	Recovery	Recovery	"Overshoot," Recovery	Recovery	Recovery	Recovery	Recovery

Table 1. Classical physiological effects of a sustained (30-35 sec) Valsalva. ITP-IAP=intrathoracic-intraabdominal pressure; BP=blood pressure; HR=heart rate; SV=stroke volume; ICP=intracranial pressure; CPP = cerebral perfusion pressure.

During ***Phase IV***, or recovery, we observe a sudden rapid rise in blood pressure, as the restored preload primes the heart for a surge in stroke volume. Increased cardiac output and vascular resistance jack up the blood pressure – the frequently-described "overshoot." These hemodynamic responses are represented in Figure 3.

Such is the *classical* description of a Valsalva maneuver lasting about 20-30 seconds. The situation with exercise is more complicated, and more poorly described. Valsalva under a load tends to be rather more brief, and the hemodynamic demands of the movement are superimposed on a truncated version of the maneuver [4]. When a lifter is performing any but the most protracted squat, *there may be no Phase II*, because the rep just doesn't last that long.

Under a load, the blood pressure *rises* during Valsalva. Narloch et al have speculated that this is due to augmentation of venous return by the pump action of the muscles. This speculation is reasonable, but it is just that – speculation. Nevertheless, it seems clear that resisting a load under a Valsalva generates a *much* more dramatic increase in blood pressure than performing the same exercise

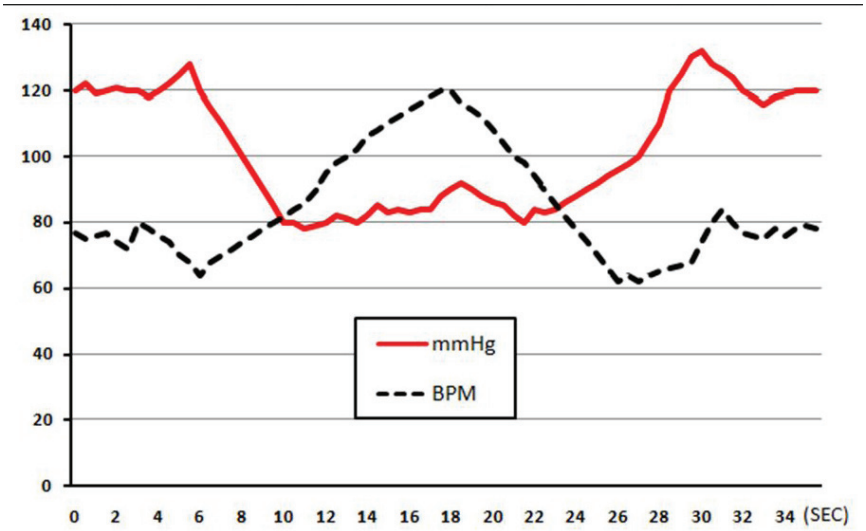

Figure 3. Changes in systolic blood pressure (mmHg) and pulse (BPM) during a classic sustained Valsalva maneuver. Data adapted from various sources by the author.

without Valsalva. Systolic blood pressures in excess of 300 mmHg are not uncommon in the literature[10, 16]. It is important to note, however, that *exercise without Valsalva also precipitates very marked increases in blood pressure*, although not as dramatically[17].

In summary, then, lifting weights causes your blood pressure to shoot up. If you lift weights under Valsalva, this hemodynamic response may be quite exaggerated, and your blood pressure can get very, very high indeed. For our present purposes, that's important, because it is this increase in blood pressure that is invoked as the precipitant for intracranial hemorrhage in the setting of resistance training:

- You lift weights under Valsalva.
- Your blood pressure goes up.
- You blow an O-ring.
- *You stroke*.

That's what we are asked to believe. But before we swallow, maybe we should take a closer look at how strokes happen, and the role of intracranial pressure.

Stroke me, Stroke me

A ***stroke*** is a brain injury arising from a cerebrovascular catastrophe (often called a "cerebrovascular accident"). Stroke comes in two major flavors: ischemic and hemorrhagic. An ***ischemic stroke*** occurs when the blood supply to a brain region is cut off. This is by far the most common variety of stroke, but it is not where our present interest lies.

Hemorrhagic strokes comprise about 15-20% of all cerebrovascular accidents, and occur when the rupture of a blood vessel results in bleeding into the cranial vault[18]. A "bleed" can be one of several varieties. Traumatic bleeds (epidurals and subdurals) are not the focus of this discussion (but try not to drop the barbell on your head). Intraparenchymal hemorrhages are bleeding directly into the brain tissue, frequently in the setting of severe uncontrolled hypertension. They are rarely described in the context of resistance training.

Intracranial hemorrhage (ICH) in the setting of resistance training and other strenuous activities is almost always of the variety known as ***subarachnoid hemorrhage*** (SAH). This term refers to the location of the bleeding, between two layers of the ***meninges***, the membranes that

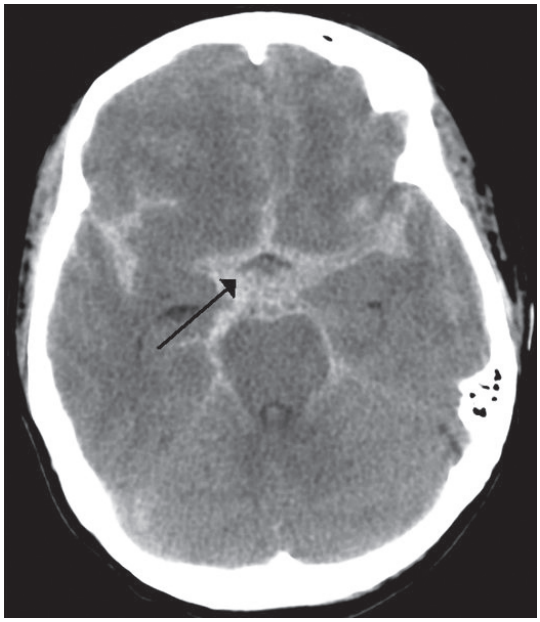

Figure 4. Cranial computed tomographic (CT) image of a patient with subarachnoid hemorrhage. In this technique, brain matter is gray and bleeding is gray-white. This patient has an extensive hemorrhage, with blood tracking in the fissures of the cerebral cortex, and also collecting in the perimesencephalic cistern and supracellar cisterns (arrow), near the brainstem and pituitary. It is instructive to compare this image with Figure 5. Image by James Heilman MD; reproduced from Wikipedia under Creative Commons License.

enclose the brain. SAH occurs between the two innermost layers, the ***arachnoid mater*** and the ***pia mater***.

Subarachnoid hemorrhage (SAH) is most commonly caused by rupture of a ***saccular cerebral aneurysm*** (CA). (SAH can also arise from other lesions, including tumors, arteriovenous malformations, and from trauma. We will not consider such lesions here.) The saccular aneurysm is the most common form of CA, the so-called "berry aneurysm." These are small, spheroid outpouchings of a cerebral artery that occur primarily at branch points in the arterial tree, particularly in the ***Circle of Willis***. The Circle is a roundabout formation of cerebral arteries at the base of the brain uniting the anterior circulation from the carotid arteries with the posterior circulation from the vertebral arteries.

Berry aneurysms lurk in the heads of about 1-6% of the general population[18]. Berries are congenital, although environmental and behavioral factors appear to have an impact on their postnatal development and risk of rupture. Genetic factors that predispose to CA include female sex and alterations in genes for various connective tissue proteins and proteases. Risk factors contributing to development and rupture of congenital CA include smoking, hypertension, heavy alcohol use, and increasing age.

The most important factor in the rate of CA rupture appears to be aneurysmal size. The International Study of Unruptured Intracranial Aneurysms[19] investigated the natural history and clinical outcome in 4060 patients with unruptured CA. They found that the rate of rupture for lesions less than 7mm in diameter was 0-2.5% over five years, while the five-year rupture rate for very large aneurysms (>25mm) was up to 50%. A larger aneurysm creates hemodynamic, histological and biophysical conditions that favor rupture[18].

Looking at the literature on ICH and lifting, we find that most ICHs were of the SAH variety, and those that underwent anatomical investigation were almost always associated with ruptured berry CAs. The epidemiology of this rare phenomenon is still sketchy, but it is fair to say *the literature gives us no indication that resistance training increases the risk of ICH in the absence of severe uncontrolled hypertension, coagulopathy, congenital aneurysm or other underlying cerebrovascular pathology*. In other words, if you don't already have a time bomb in your head, it probably won't go off, no matter how much you lift and grunt.

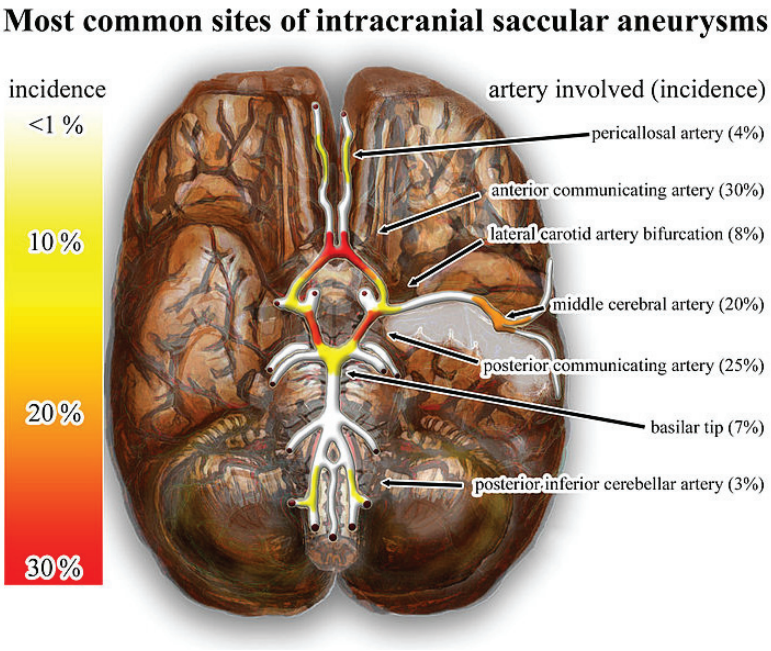

Figure 5. The Circle of Willis. The circle connects the anterior circulation (from the carotid via the middle cerebral artery) and posterior circulation (from the vertebral arteries via the basilar and posterior communicating arteries) in a ring of vessels at the base of the brain (center). The incidences of congenital aneurysm by vascular site are indicated by the vessel color and corresponding thermogram at the left. It is instructive to compare this image with Figure 4. Image by Nicholas Zaorsky MD; reproduced from Wikipedia under Creative Commons license.

This means that any consideration of an increased risk of hemorrhagic stroke while lifting under Valsalva is *practically* restricted to those who have such lesions. This is both a reassurance (because the incidence of such lesions in the population is so low) and a concern (because people who have such a monster in their head generally don't know it, and there's no quick-cheap-and-easy way to screen for them).

Nevertheless, the laws of chance dictate that *some* people with berries are going to get under the bar. Many if not most of them are going to hold their breath. When they do, blood pressure will shoot up, increasing the intravascular stress on the aneurysm. Doesn't the Valsalva pose a clear and present danger to them?

There are two complementary approaches to analyzing this question, and neither of them can call on enough data to give us a definitive answer. The first approach is to consider the dynamics of aneurysmal rupture in the setting of resistance training with Valsalva—the ***physiological evidence***. The second approach is to survey the epidemiologic data—the ***clinical experience*** of what actually happens to human populations when they lift under Valsalva. We will consider each in turn.

The Physiological Evidence

Several factors influence aneurysm rupture, but for our purposes the critical variable is ***cerebrovascular transmural pressure*** (TMP), the net force across the wall of the aneurysm. Transmural pressure is the difference between the (1) internal (arterial) pressure, which is more-or-less equivalent to the ***cerebral perfusion pressure*** (CPP), and (2) the ***intracranial pressure*** (ICP). The intracranial pressure is the pressure transmitted through the cerebrospinal fluid, which circulates throughout the central nervous system (brain and spinal cord) within the closed sac of the meninges. The CPP normally ranges from between 70 and 85 mmHg in a resting adult, while the normal value for the intracranial pressure is about 5-15 mmHg[20]. This means the resting mean TMP should be about 55-75 mmHg, although direct measurement of this value in humans is rarely reported in the literature.

Now, what happens when you lift? Let's call this condition ***RT***, or resistance training *without* Valsalva. Resistance training jacks up the systemic blood pressure, which drives up the CPP, increasing the pressure gradient across the aneurysmal wall (TMP)[4, 17, 21].

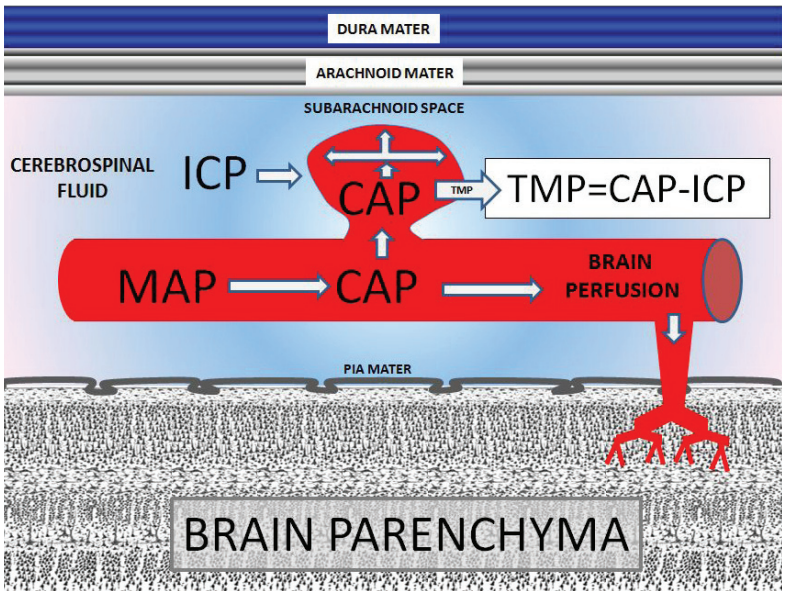

Figure 6. Hemodynamic and anatomic relationships pertaining to aneurysmal rupture. The cerebral artery and its aneurysm occupy the subarachnoid space, overlying the brain tissue. Mean arterial pressure (MAP) is transmitted to the cerebral artery as the cerebral arterial pressure (CAP). CAP is defined here as the pressure applied to the walls of the aneurysm from within, and is counteracted (but not necessarily balanced) by the intracranial pressure (ICP) which is transmitted through the cerebrospinal fluid. Image prepared by the author.

What happens when we add Valsalva to the mix? Let's call this condition ***RT+V***. Existing data[10, 22, 23] indicates that RT+V drives up the arterial blood pressure even further, with a corresponding dramatic increase in CPP and intravascular stress. Rupture is now imminent. *She's gonna blow*.

Except it's not that simple. Our analysis must take into account the profound effect of Valsalva on intracranial pressure, the other critical factor in the pressure across the aneurysmal wall.

In the 1930s, Hamilton developed a new "differential manometer" which allowed direct invasive measurement of rapid changes in vascular and cavitary pressures[14, 24]. This work yielded the first modern picture of the hemodynamics of the Valsalva maneuver. Hamilton's technique allowed him to record cerebrospinal fluid pressure, and he showed that the increase in thoracic pressure produced by Valsalva is transmitted directly to the cerebrospinal fluid, thereby increasing the ICP.

> *…changes occur in the spinal pressure during straining and coughing which protect the arterial tree within the craniospinal canal from these unusual stresses. Thus a simultaneous sudden rise of arterial and spinal pressures of 100 mmHg…leaves the net arterial pressure unaffected.*

This was such a seminal observation that Hamilton's papers are still widely quoted today in the literature on Valsalva and intracranial pressure.

The increase in ICP with Valsalva has been verified in multiple investigations since Hamilton's paper appeared. Of particular interest are Prabhakar's measurement of CPP and ICP (allowing derivation of TMP) in patients undergoing neuroendoscopic procedures via a cerebral ventriculostomy[25], and Haykowski's measurements of ICP during bicep curls in patients with neurosurgical drains[26]. Haykowski's contribution is particularly important. His invasive measurements of ICP and MAP demonstrated that the RT condition generated *higher* calculated TMPs than the RT+V condition. If the results of Haykowski's direct measurements in living human beings are correct, then lifting weights with a Valsalva generates *less* stress across the vascular wall than lifting weights without a Valsalva. These findings are a direct and important challenge to the conventional view of the "dangers of Valsalva."

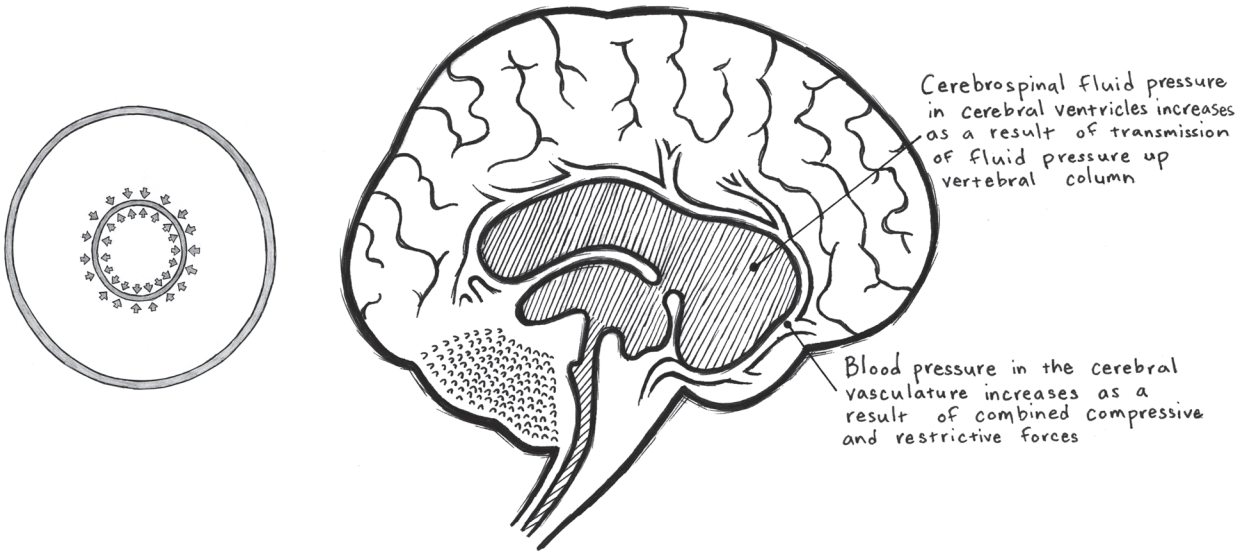

Figure 7. Mechanism for vascular protective effect of Valsalva. The rise in vascular pressure caused by work against a load under Valsalva is counteracted by a simultaneous increase in intracranial pressure transmitted via the cerebrospinal fluid. The volume of the skull, which is fixed, limits the volume and pressure of these two systems and stabilizes vascular structures, rather than predisposing them to rupture. Reproduced with permission from *Starting Strength: Basic Barbell Training*, 3d Ed 2011; The Aasgaard Company.

In 2012 Niewiadomski et al[27] published an important investigation of the effect of the Valsalva maneuver on the hemodynamic response to resistance exercise. Twelve Polish bros ("*broskis*") performed concentric and eccentric leg press exercises while their blood pressure, heart rate and mouth pressure (an indirect measurement of intrathoracic and intracranial pressures) were recorded. As expected, Valsalva markedly increased systolic and diastolic blood pressures at rest and during exercise. Stronger Valsalva in conjunction with heavier loads produced the largest blood pressure spikes. The authors derived values for arterial transmural pressures during systole (when pressure is the highest). They found that Valsalva *decreased* transmural pressures. No broskis died or stroked out during the experiments. Again, this work directly and substantially challenged the idea that Valsalva increases the risk of ICH in the RT+V condition, and supports a model in which the increased ICP with breath-holding moderates the TMP.

The accumulation of papers like those of Prabhakar, Haykowski and Niewiadomski allowed for an important systematic review by Hackett in 2013[10]. The best evidence showed that RT+V increases blood pressure, *but not as much as Valsalva alone*. The authors recommend that the Valsalva should not be exaggerated under a load, but it should not be avoided, either, considering the moderating effect of Valsalva on TMP. A theoretical caveat to this model is that release of the classical prolonged Valsalva produces an overshoot in systemic blood pressure, even as ICP falls, potentially increasing the TMP. This dynamic has not been described, however, and there is data suggesting that CPP does not overshoot after Valsalva[28]. In summary, the physiologic data, while flawed, clearly indicates that (a) RT raises blood pressure; (b) RT+V raises it even more; and (c) the high intrathoracic pressures generated in the RT+V condition are transmitted to the cerebrospinal fluid and cranial vault, increasing ICP and moderating changes in transmural pressure – a *protective* effect[29].

Another critical point emerges from the physiological data. Multiple authors have observed that, notwithstanding any physiologic effects the Valsalva may have, it is an everyday occurrence and is *virtually unavoidable under heavy loading, even when the lifter is instructed not to do it*[15, 17, 10, 16, 30]. Of course, anybody who's ever squatted or deadlifted any serious weight, or found themselves confronted by the exigent necessity of lifting a heavy object off an injured child, already knows this.

The implication is as obvious as it is far-reaching.

Prohibiting the Valsalva is tantamount to a prohibition of heavy lifting itself.

Clinical and Coaching Experience

It's one thing to drill holes in people's heads, ram catheters into their spinal canals, cannulate their arteries, and then make them do leg presses. That gets us *physiological* data, which is important for our overall understanding, but not definitive. Physiological reasoning, even when based on the best data, often turns out to be dead wrong in the clinical setting.

For a definitive, practical, real-life understanding of the interaction between Valsalva and the risk of hemorrhagic stroke from aneurysmal rupture, we need to look at the large, controlled, longitudinal clinical studies with relevant clinical endpoints that examine *what actually happens* to lifters, with and without intracranial lesions, with and without Valsalva. So let's look at those.

Oh, wait. Sorry. *There aren't any.*

That's right. For all the *Sturm und Drang* about Valsalva and popped berries, nobody has *ever* demonstrated a cause-and-effect relationship between RT+V and intracranial hemorrhage, even in susceptible populations. It's easy to see why. Berry aneurysms are rare. Atraumatic SAH is quite rare – about 9 strokes/100,000 patient-years[31]. And blowing an SAH under the bar? That is exceedingly,

incredibly, *fantastically* rare. Studying such a rare clinical phenomenon in a controlled manner is virtually impossible. A phenomenon this uncommon forces us to rely on isolated case reports, laboratory data, and physiological reasoning. It also throws open the door to guesswork, speculation, and crazy-ass bullshit.

Let's leave the speculation and bullshit to the Chicken Littles and Ambulance Chasers for the moment, and turn our attention to the only clinical data we have: case reports and case series.

Of these, the most important and frequently quoted is Haykowski's series of 3 cases[32]. The first case was a 24 year-old who developed headache while performing leg presses. He was found to have an SAH due to a ruptured aneurysm, which was successfully clipped. He went home.

The second case was a 36 year-old man doing bicep curls, who developed a headache, puked, and passed out. He was found to have a large SAH due to a ruptured aneurysm, which was successfully clipped. He went home.

The third case was an 18 year-old bro doing brocurls when he developed a "shock-like" sensation in his limbs, and then puked. An SAH was diagnosed on lumbar puncture. Angiography revealed an aneurysm, which was successfully clipped. He went home.

Valsalva was not documented in any of these cases. Haykowski assumes that Valsalva was performed by these lifters, arguing quite reasonably that lifting heavy without Valsalva is difficult to impossible, depending on the intensity. But we don't know the relative intensity at which these guys were working out when they blew their gaskets. Maybe they were holding their breath. Or maybe they were doing *T'ai Chi* breathing with their curls and leg presses. We just don't know.

Haykowski's series is obvious fodder for exponents of the anti-Valsalva position. In that light, the following paragraph from his paper is particularly fascinating:

> *In all of the patients, the length of time from surgery to return to activity was 3 months. After this recovery period, the patients were encouraged to return to their previous occupational and recreational activities of daily living.* ***In addition, weight training with Valsalva maneuver was not proscribed at this time.***(Emphasis added.)

In other words, these patients were told to go on back to the gym and lift under Valsalva if that's what kept their taters toasty. Seems pretty ballsy, doesn't it? Or perhaps not, based on what we know about the dynamics of rupture. In fact, as we've already seen, Haykowski went on to do important laboratory work supporting the view that Valsalva is protective[26].

Haykowski's series of three guys who got clipped and sent home to start lifting again under Valsalva represents *the lion's share* of peer-reviewed descriptive clinical data focused on weightlifting and aneurysmal rupture. As we'll see, other such cases are registered in injury databases, without peer-reviewed clinical description. There are other scattered case reports of SAH, subdural hemorrhage, barotrauma, retinal injury, coronary dissection, and other misadventures[10]. They all have one thing in common, and it's not Valsalva. It's that *they're all case reports*, which is the standard format for communicating information about *fantastically rare* clinical entities.

When we look at the clinical literature on SAH independent of resistance training, we get a more complete and interesting picture. For example, Matsuda et al published a retrospective chart review of 513 consecutive patients admitted to a neurosurgical service with SAH, identifying circumstances of rupture[33]. Although the authors invoked Valsalva as a potential contributor in some cases, no association of the maneuver with any of these hemorrhages is documented. Rupture was associated with "sporting/exercising" in 2.7 % of cases. This made exercise one of the most infrequent associations with SAH, behind eating and drinking (4.7%), shopping (6%), housework (7.6%),

sleeping (8%), or shitting and pissing (12.7%). The big winner, at about 14%, was "chatting/watching television/staying home." In other words, you were more likely to pop your cork *just sitting in front of the tube* than while working out.

A case-crossover study by Vlak et al[34] looked at 250 survivors of intracranial hemorrhage to identify precipitating factors. Eight triggers increased risk for ICH: coffee, cola, anger, startling, straining to poop, sexual activity, nose blowing, and vigorous exercise. (This sounds to me like the agenda of a day well-spent.) Valsalva during lifting was associated with a lower risk than during sex, masturbation, anger, and blowing one's nose.

One more, just to hammer this home: Shievink published a 1989 series of 500 consecutive cases of SAH presenting to a neurosurgical center[35]. As usual, there was no reliable documentation of Valsalva. "Lifting heavy loads" and "Sporting/exercising" preceded 2.4 and 3.8% of events, respectively – less than sex (6.4%), pooping (7.6%), standing up (5.4%) or *sitting down* (8.8%) **.**

I did find a case report in the literature in which a Valsalva maneuver appeared to be clearly associated, at least temporally, with an intracranial hemorrhage. Carlson et al report a case of *non*-aneurysmal cerebellar hemorrhage, with no identifiable vascular anomaly, in a 60 year-old man with untreated severe hypertension (presumed chronic) who suffered onset of headache and nausea while playing the trumpet[36]. The authors cite several case reports of neurological complications while *blowing on things*, none of them due to aneurysmal rupture.

People with *known* intracranial lesions or uncontrolled hypertension probably should not lift weights. They shouldn't take up the bassoon, either.

I've saved the most important article of "clinical data" for last. It is not a peer-reviewed study, case report, or case series. It's the article I mentioned at the beginning of this piece, published in *The Exercise, Sports and Sports Medicine Standards and Malpractice Reporter* by Debra Bursik and Gregory Conway[1]. The authors cite the now-familiar view that RT+V leads to cranial hemorrhage and other catastrophes. They cite none of the basic investigations arguing either for or against RT+V, but only expert opinions and a position paper from the American Heart Association[2].

They go on to describe a case brought against a fitness center and personal trainer for failing to warn a client against the dangers of Valsalva. The client suffered an intracranial hemorrhage. Plaintiff produced expert witnesses to testify that the Valsalva is dangerous and should not be permitted. The defendant also produced expert witnesses to testify to the contrary, but, as Bursik and Conway tell us:

> *…several professors of exercise science as well as practitioners said they didn't bother to warn about the Valsalva maneuver because a stroke during weightlifting was so rare. Upon further questioning, it turned out that* ***none of them knew the NEISS material from the Consumer Product Safety Commission was in existence****.* (Emphasis added.)

If the word "ambush" just popped into your head, you're still paying attention. The material in question, which was used so effectively by plaintiff's counsel to surprise and discredit defendant's expert witnesses, had not been published in the peer-reviewed literature. Rather, *it was data abstracted by the plaintiff's litigation team* from the National Electronic Surveillance System Database. Bursik and Conway report that plaintiff's data documented "***32 cases from 2002-2010 in which the injured individuals suffered stroke or subconjunctival hemorrhage in association with weightlifting.***" Plaintiff claimed that extrapolating this data (obtained from 100 hospitals nationwide) shows that 1287 such cases must have occurred during the same period – more, in fact, because many such cases probably did not present to the ER.

Based on this data and testimony of plaintiff's experts in this case, along with literature arguing (without good evidence, as we've seen) against Valsalva, Bursik and Conway tell us that stroke caused by RT+V is "not rare at all." Trainers and coaches have a duty to warn their clients against performing Valsalva—presumably upon pain of litigation.

I have *huge* problems with this "data," as will become apparent. But for the sake of argument, let's give it the benefit of the doubt. Let's take that data, a crayon, and the back of an envelope, and do some quick-and-dirty calculating, using a few very conservative assumptions of our own.

Say Bursik and Conway are right – that plaintiff's extrapolation of 32 reported cases to 1287 in 9 years is not only valid but in fact lowball, because not all such cases present to hospital ERs. Let's say fully half of such cases end up somewhere else. (Where? I don't know. The local Steak-n'-Shake, say.) Let's *double their number*, to 2574 such cases over 9 years, or 286 cases per year.

And let's start with a very conservative estimate of the number of people lifting weights in the US. This number is between 37 to 45 million[37]. Let's tilt the argument further in favor of Bursik and Conway, and cut that by about half, to 20 million.

Let's say each of these 20 million lifters works out for only 3 hours a week (I wish). But they're not spending all that time under Valsalva – and we're assuming, along with Bursik and Conway, that virtually all of these injuries occurred under RT+V conditions. So let's say these lifters are spending about 15% of their workout time in Valsalva. That number is inflated, but we're at pains here to make assumptions in favor of plaintiff's argument. We get a total of 0.45 hours/week spent by each of these 20 million lifters under Valsalva. Let's call these ***hours-at-risk***, or ***Hr .*** That turns out to be 23.4 Hr/year, or almost 24 hours annually that each of these lifters is exposed to the risk of hemorrhage. If we multiply that by the number of lifters, we get 468 million Lifter-Hr/year of risk exposure across the entire United States. That's the denominator. The numerator is the total number of hemorrhages per year: 286, using our generous interpretation of Bursik and Conway's data.

Dividing the numerator by the denominator gets us an injury rate of 6.1×10^{-7} hemorrhages/Hr, (or, to use the more standard metric, 0.0006 injuries/1000 Hr.)That's 0.0000006 hemorrhages per hour spent *under Valsalva*. Taking the reciprocal, we find that our lifters will sustain one stroke in *1.6 million hours* of working out under Valsalva.

But our lifters are exposed to this risk for only 23.4 hours a year. Therefore:

(1,636,364 Hr/Inj) x (1 y/23.4 Hr) = 69,930 y/Inj

This is a preliminary, back-of-the-envelope calculation, but it comports with the general approach to evaluating and comparing injury rates in sport[38], and I think it gives you some idea of the magnitude of hemorrhagic stroke risk in the setting of resistance training. Interestingly, if we do a similar calculation based not on time under Valsalva, but rather on heavy workset reps under Valsalva, we get exactly the same answer.

In short, based on a generous reading of Bursik and Conway's data and using conservative assumptions that favor their position, a lifter from the general population training three hours a week with 15% of that time under Valsalva will, on average, sustain *one SAH in seventy thousand life-years***.**

And yet, our calculations assumed that the risk is the same for all of us – which it is not, since, as we've already seen, the majority of intracranial hemorrhages occur in those unfortunate individuals with a pre-existing lesion. When we take that 1-6% congenital aneurysm rate into consideration, we start to get into stroke-free lifter life spans that invite consideration of sub-light-speed interstellar travel. Moreover, my analysis of the data cited by Bursik and Conway accepts their assumption that

the 32 *reported* cases can be extrapolated to 1287 *actual* cases. We assumed that their number under-represents the actual hemorrhage rate by 100%, that all such hemorrhages occurred in the context of Valsalva, and that all of these strokes could therefore have been prevented.

But we shouldn't accept *any* of these assumptions.

Because *they're all bullshit.*

The extrapolation of the 32 cases to 1287 assumes that the 100 hospitals surveyed by the NEISS for this data are truly representative of the admission rate for weight-training-associated SAH across the US. Given the extremely small incidence of such strokes, this relationship cannot possibly be demonstrated. The contention that the 1287 figure actually under-represents incidence is based on their further assumption that many cases of hemorrhagic stroke are never evaluated in the ER.

Really? *Where else would they go?* The chiropractor? Coach's office? The In-And-Out Burger? Let's get real: If you have an acute subarachnoid hemorrhage in America, you will end up in one of two places: the ER or the Eternal Care Unit.

It gets worse. Any implication that all of these cases occurred in the context of Valsalva is entirely unsupportable. True, it is difficult if not nearly impossible to lift heavy without Valsalva, as we have seen. But were the 32 hemorrhages reported in the NEISS and cited by plaintiff caused by heavy lifting under Valsalva?

Answering this question required some detective work on my part. But I was able to track down the databases for the years involved, covering accidents and injuries associated with weightlifting. Searching for *hemorrhage*, *bleed*, *stroke*, etc, I extracted all the reported intracranial and subconjunctival hemorrhages for that period I could find, and came up with a total of….32 cases!

And when I looked at these 32 cases, I found that the NEISS data cited by Bursik and Conway does *not* document that these individuals were lifting heavy. It doesn't document which exercises they were doing. It doesn't document the weight lifted, the medical histories, whether they were using barbells – nothing, except that they were "lifting weights" and then had a hemorrhage.

More importantly, my examination of the NEISS database uncovered absolutely no documentation of Valsalva in any of these thirty-two cases. Not a single one.

And still it gets worse. The thirty-two cases I found included only *twenty* intracranial hemorrhages. The remaining 12 cases were not strokes. They were subconjunctival hemorrhages.

Subconjunctival hemorrhage is much like an intracranial hemorrhagic stroke, except for the "intracranial" and "stroke" parts. In fact, a subconjunctival hemorrhage is nothing more than the colorful result of a burst blood vessel in the sclera (white of the eye), which can occur in the setting of sneezing, defecation, sex and, yes, weight training. Sometimes you just wake up with one. It is a *completely benign*, non-vision-threatening, self-limited entity that requires no specific treatment.

So why didn't Bursik and Conway note in their article that the inclusion of subconjunctival hemorrhages in the plaintiff's data was obviously problematic? Not just problematic, in fact, but *completely inappropriate?* If one were unkind, misanthropic and given to speculation, one might very nearly be tempted to entertain the cynical notion that the submission of such data *could* be interpreted as a deliberate attempt to mislead the court. Why didn't Bursik and Conway pick up on that?

Well, here's a clue: Conway was the attorney who brought suit on behalf of the plaintiff. Bursik did the NEISS research for the plaintiff. *Why in the world* would such nice people use irrelevant, non-peer-reviewed, unpublished data in support of a lawsuit? I leave that *entirely hypothetical* question as a stimulating thought-exercise for the reader.

The Bottom Line: Everybody Needs to Take a Deep Breath

The foregoing deconstruction of the Bursik-Conway piece was elaborated at some length, because I think it is terribly important, both in general and in particular.

It is important in the particular sense because it is the most recent and perhaps most legally consequential argument against the use of Valsalva in RT that we have yet seen. The case cited (and litigated) by Bursik and Conway, and their accompanying article, have important negative connotations for those of us who coach the barbell lifts. The case is probably on the public record, and the Bursik-Conway article has publicly declared – *from a legal perspective* – that failing to warn about the "dangers" of Valsalva, or even permitting a client to perform the maneuver, violates the standard of coaching practice. The article also arrogates unto itself an undeserved veneer of academic respectability. Indeed, the Bursik-Conway data has already been cited by a professor of exercise science speaking at a national conference[39], who called it "an NEISS study."

The Bursik-Conway deconstruction is important in a more general sense because it is one more very salient example of (a) the shit we're up against and (b) why we shouldn't take it lying down. There is no shortage of misguided, misleading or outright dangerous opinions about barbell training confronting coaches and clients. These opinions, positions and policy statements are usually dressed up with fancy language, footnotes, and "expert" opinions. They can even find their way into the peer-reviewed literature, the courthouse and, worst of all, the legislature. They usually reflect personal, commercial or political agendas at odds with the public welfare.

The one thing we have going for us is that such opinions are exquisitely photosensitive: They rapidly decompose into their constituent shit molecules when exposed to the light of evidence and reason. We, as a community, need to shine that light. It needs to be on all the time, and it needs to be painfully bright.

So where does all of this leave us, as coaches and lifters?

I think it's important to point out that hemorrhagic stroke is not the only injury attributed to RT+V. Vascular dissection, retinal hemorrhage and other injuries have been described in this setting[10]. As with ICH, there is no controlled, epidemiological data establishing a causal link between RT+V and any risk for these rare events. *But they do occur*. Also, people drop barbells on their heads – *far* more frequently than they get dissections and retinal hemorrhages.

Putting together everything we've seen, it's fair to say that if you lift heavy, and if you have an aneurysm, you are at risk for subarachnoid hemorrhage. One need not invoke any cause-effect relationship for this statement to hold. Indeed, I believe there is no good evidence demonstrating a causal relationship between RT+V and hemorrhage. In my opinion, *the risk resides in the aneurysm*, not the lifting.

If you're a lifter with an aneurysm, and if the damn thing is doomed to pop, there is a certain nonzero probability that it will just happen to blow while you're under the bar. The magnitude of any contribution of RT+V to this risk is, as we have seen, unclear at best, and probably nonexistent. In fact, there are excellent reasons to believe the maneuver is *protective*. These difficulties are compounded by the fact that if you are one of those rare people who have an aneurysm, you probably don't know it. Sorry, but I have no remedy for this, short of performing cerebral angiography on the entire population.

Similarly, if you're a coach, you confront a spectacularly remote but nevertheless nonzero risk of standing next to a client in the squat rack on the day Providence decides to pull the plug that's been lurking in his head for the last 43 years. Notwithstanding the complete lack of any definitive

evidence that lifting under Valsalva contributes to such an injury, your chances of getting sued in this circumstance are excellent. On the other hand, your chance of reasoning with a jury selected specifically for their lack of higher cognitive function is abysmal. This underscores the importance of covering your ass, to the extent possible, with insurance and signed documentation of assumption of risk by the client, *including explicit assumption of any risk of cardiovascular, ocular, pulmonary and cerebrovascular complications from exercising under Valsalva*.

If it sounds like I just contradicted everything I said in this article…well, I am vast. I contain multitudes. I *do* believe the "dangers of Valsalva" are imaginary. I *also* believe the dangers of our tort system are very real. The pathologically litigious nature of American society has proven highly resistant to evidence and reason. This is unlikely to change. Wear a cup.

People with known aneurysms or other intracranial lesions, known retinal disorders, a family history of aneurysm or SAH, or a history of polycystic kidney disease should not lift, Valsalva or no Valsalva, unless and until cleared by a physician. I make no claim that my list is inclusive of all conditions requiring physician evaluation and clearance. It is not.

Symptoms of intracranial hemorrhage include but are not limited to explosive headache, nausea, vomiting, visual disturbances, syncope (passing out), and neck stiffness. If you (or your client) have any of these symptoms, ***stop***. Stop holding your breath, stop lifting, stop having sex, stop straining at that turd, rack the friggin' bar, *put down the goddam trumpet,* and get thee to the ER at once. People dial 911 for *much* sillier reasons all the time.

Beyond that, your options are limited. You can go out and grab life. Or you can hide…*but where?* You can certainly sustain a brain hemorrhage in The Proverbial Saddle or under the bar. But you can also stroke out while watching *Duck Dynasty* or taking a dump. There's no clear pattern here, no safe refuge, no guarantees. *You pays your money and you takes your chances*. This is life. Nobody gets out alive.

If you want to be strong, you have to lift heavy. And if you lift heavy, you're going to lift under Valsalva. I'd like to think you now have a better perspective on this practice. The rest is up to you.

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Dr. Sullivan is a *deeply* conflicted person, but has no conflict of interest to report.

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