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# 10 or 15 or 20 or 40 mmHg? What is Increased Intracranial Pressure and Who Said So?

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Increased intracranial pressure (ICP) has been recognized since the early 1900s as an important mechanism for additional brain injury after the first impact. Increased ICPs are not always consequential; in fact, we all experience them daily. For example, coughing or straining has been shown to cause large, abrupt cerebrospinal FLUID pressure fluctuations [1]; these arise out of communication between the cerebrospinal fluid and intrathoracic pressures through the venous system, but the surges are absorbed without damaging brain tissue. Head positioning (whether elevated or rotated) is also critical if the hydrodynamics are in free communication. When a new intracranial mass appears (e.g., blood clot, swollen infarcted tissue, tumor swelling), a resulting increase in ICP may cause the brain to shift, resulting in pressure necrosis in the cingulate gyrus and occipital cortices. Diffusely increased pressure most significantly impacts the parahippocampal gyri. Increased pressure in the posterior fossa, mostly from a cerebellar mass, causes the cerebellar tonsils abutting the rim of the foramen magnum to turn necrotic.

Crucial information came with experimental studies by Langfitt et al. [2] and Sullivan et al. [3]. These teams plotted the time course of the ICP during the slow, constant-rate expansion of an extradural balloon. The initial segment of the curve shows only a modest increase in ICP with time; then, the curve breaks sharply so that ICP increases dramatically as the mass expands. Thus, the ICP may remain relatively low at a point in time when an intracranial mass has reached a critical size. The plot of ICP compared with mass volume thus has two segments: a slowly ascending segment and a steep segment. Fig. 1 shows a polygraph recording demonstrating the ICP changes at the time of one of the rapid intraventricular saline injections made in these experiments and subsequent plotting of the pressure–volume curve.

Two questions remained. First, how did this experimental information translate to the bedside? Second, there is now reasonable consensus among neurointensive care unit staff that sustained levels above 15 mm Hg are abnormal and that progressively increasing ICP when brain compliance is poor is equally worrisome, even if the level of 15 mm Hg is not reached. But where did that "magic number" come from? This vignette provides scrutiny of some key studies while acknowledging a much larger body of work in this field.

## Historical Definition of Increased ICP

Lundberg in his 1965 classic studies on continuous ventricular fluid-pressure recordings felt that ICP of 10 mm Hg was normal, slightly elevated if sustained above 15 mm Hg, moderately elevated at 25 mm Hg, and severely elevated above 40 mm Hg [4]. His "A" waves (plateau waves) reflected ICPs in the 50–mm Hg range, and this resulted in vasodilatation subsequently resulting in another A wave. This was different than his B and C waves, in which ICPs would not pass the threshold of 20 mm Hg [5].

The first and possibly most influential study came from Miller et al. [6] (Fig. 2). The overwhelming proportion of patients with traumatic brain injury with documented high ICP were comatose. If a hematoma was present and subsequently evacuated, the elevated pressure was considered important for prognosis. If

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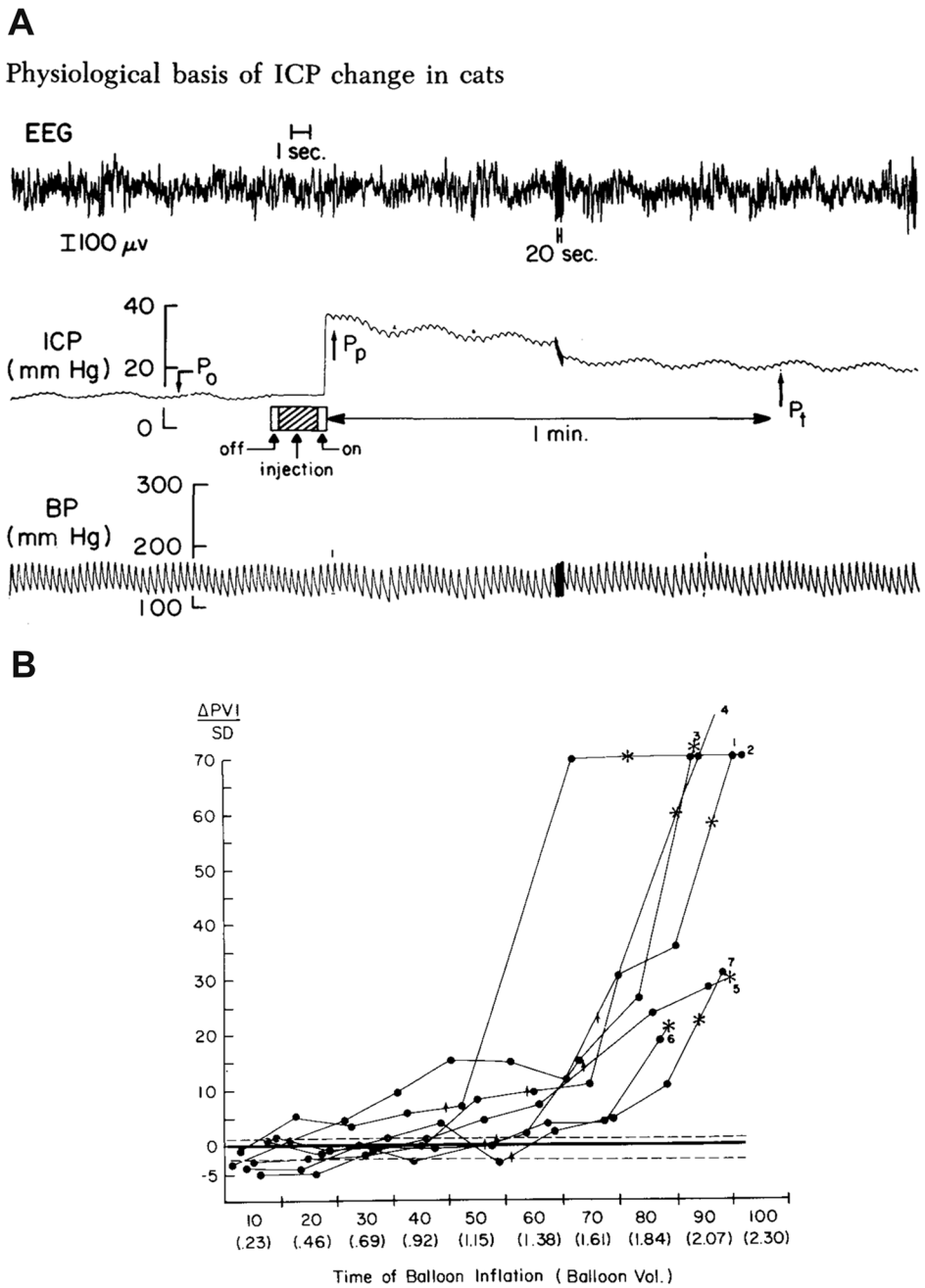
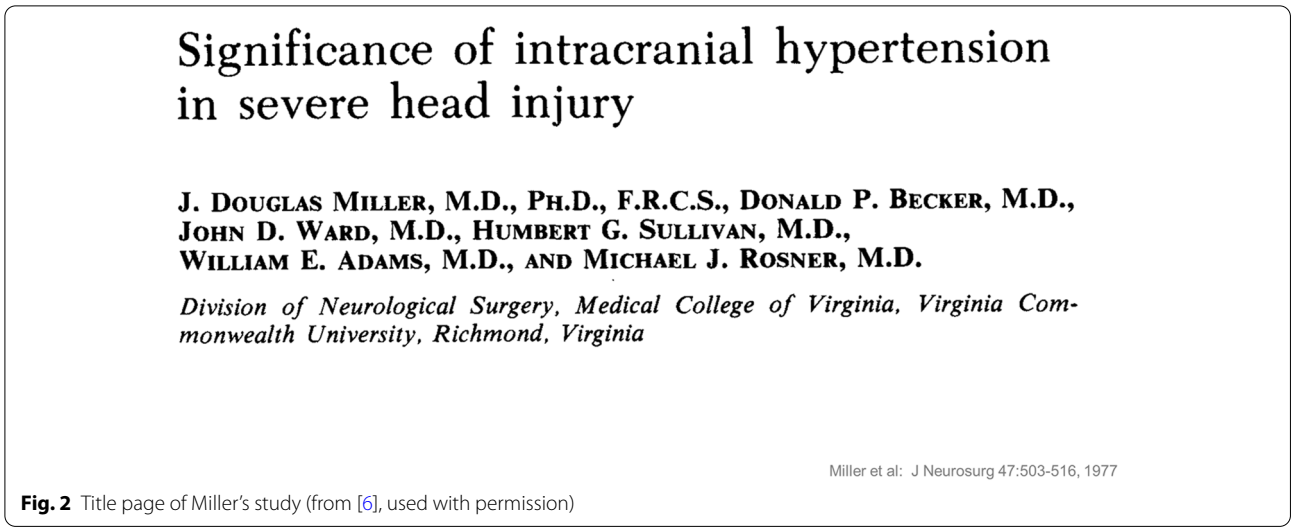



**Fig. 1** **a** Experimental set up. **b** Pressure–volume curve (from [6], used with permission). BP, Blood pressure, EEG, Electroencephalogram, ICP, intracranial pressure

pressure continued to climb in patients following surgery, as it had in more than half of the patients, it increased the probability of poor outcome. Miller et al. [6] also found that if no intracranial hematoma was present, an increase in ICP (in this study, defined as > 10 mm Hg), which was found in only a third of the patients, rarely elevated in a significant fashion (> 20 mm Hg). They stated, "the selection of a threshold pressure of 10 mm Hg gives added security…In patients with diffuse brain injury, any increase in ICP more than 10 mm Hg was associated with worsening of the neurological status and a poorer outcome so that this threshold does appear to have real clinical significance." Miller's landmark study consisted of a consecutive series of 160 patients with



Significance of intracranial hypertension in severe head injury

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Miller et al: J Neurosurg 47:503-516, 1977

**Fig. 2** Title page of Miller's study (from [6], used with permission)

severe traumatic head injury [7]. In nearly half of these patients, the best motor response to pain was decorticate response, decerebrate, or none, and the oculocephalic response was impaired or absent in 40% of the cases. In one in four patients, the pupillary light response was absent. It is important to know the management protocol to understand the results of the study. "All patients who had a $\geq$ 5 mm midline brain shift on the ventriculogram" were treated with craniotomy to remove intracranial mass lesion. Cases of no shift but increased ICP underwent an angiogram or computed tomography scan to exclude bilateral lesions. Of these 160 patients, 62 had a mass lesion requiring surgical decompression, 12 had an epidural clot, and 26 had acute subdural hematomas. ICP was monitored through a ventricular cannula or by subarachnoid screw and continued for at least 3 days. They found a trend toward higher ICP as midline shift increased, but not a strong relationship. A normal ICP (defined as 0 to 10 mm Hg) was not seen in any patient with a $\geq$ 5 mm midline shift, and there was a strong association between ICP at 20 mm Hg and shift of $\geq$ 5 mm. Clearly, there was a correlation between motor response, oculocephalic response, and pupillary light response. An abnormal motor response doubled from 48 to 86% in patients with an ICP of 20 to 40 versus 41 to 60 mm Hg. A similar jump was found in patients with absent oculocephalic responses or pupillary light reaction. Miller and associates clearly established that intraventricular pressures between 0 and 10 mm can be "unequivocally regarded as normal." They further concluded that "a sustained ventricular pressure of 18 mm Hg or 250 mm $H_2O$ should be regarded as abnormally high." This article confirms that, in patients with diffuse brain injury, any increase of ICP more than 10 mm Hg resulted in worsening neurologic examination and poor outcome, claiming significance for the threshold (Fig. 3). They suggested using a slightly higher cutoff pressure of 15 mm Hg and "classifying ICP levels during the monitoring period in the intensive care unit and redefine levels more than 20 mm Hg as indicative of unequivocally raised ICP." But "any ventricular or supratentorial pressure more than 10 mm Hg should be regarded with suspicion." Even within this study, however, there was a numerical shift with definition of increased ICP. Initially, a sustained rise more than 40 mm Hg was reduced to 30 mm Hg, whereas later studies established a threshold of 25 mm Hg for 15 min.

A consequent question is whether raising ICP causes brain damage from reduced perfusion pressure and cerebral blood flow. It is important to correlate ICP with cerebral perfusion pressure, but the early data conflict. Bruce et al. [8] found a correlation between cerebral blood flow and ICP but only when there were mass lesions. Marshall et al.'s [9] study in rabbits concluded "when the cerebral perfusion pressure (CPP) is reduced to 20 torr by raising the ICP in dogs, cerebral blood flow (CBF) is reduced to approximately 40% of control, whereas at an equivalent CPP produced by lowering systemic blood pressure (SBP), CBF is reduced to 20% of control. This indicates that CBF is better maintained during a rising ICP than during a falling SBP, thus protecting the animal from the ischemic brain disease (IBD) seen in experimental systemic hypotension at equivalent CPPs."

On the other hand, Enevoldsen et al. [10] determined that ICP had to rise above 45 to affect blood flow. They found that when systemic arterial pressure is in the normal range, ICP above 40 mm Hg correlates with a

**OVERCOME FROM HEAD INJURY RELATED TO LEVEL OF INTRACRANIAL PRESSURE ON ADMISSION***

| Diagnosis | ICP on Admission (mm Hg) | Outcome | | | Total |
|---|---|---|---|---|---|
| | | GR/MD | SD/Veg | Dead | |
| Intracranial mass lesions | 0-10 | 0 | 0 | 2 (100%) | 2 |
| | 11-20 | 7 (41%) | 2 (12%) | 8 (47%) | 17 |
| | 21-40 | 20 (69%) | 3 (10%) | 6 (21%) | 19 |
| | 41-60+ | 4 (50%) | 1 (7%) | 9 (64%) | 14 |
| Total | | 31 (50%) | 6 (10%) | 25 (40%) | 62 |
| Diffuse brain injury | 0-10 | 22 (85%) | 2 (8%) | 2 (8%) | 26 |
| | 11-20 | 28 (64%) | 5 (11%) | 11 (25%) | 44 |
| | 21-40 | 15 (58%) | 3 (11%) | 8 (31%) | 26 |
| | 41-60+ | 0 | 0 | 2 (100%) | 2 |
| Total | | 65 (66%) | 10 (10%) | 23 (23%) | 98 |

*Outcome is graded as good recovery (GR), moderate disability (MD), severe disability (SD), vegetive and dead

Miller et al: J Neurosurg 47:503-516, 1977

**Fig. 3** ICP and outcome (from [6], used with permission). GR, good recovery, ICP, intracranial pressure, MD, moderate disability, SD, severe disability, Veg, vegetative

decline in blood flow, but this also may occur in a normal brain. However, ICP above this threshold in patients with intracranial mass lesions was associated with a severe neurologic examination. Their main point was that in patients with intracranial hematoma, there is no threshold beyond which ICP causes a reduction of cerebral blood flow or indicates brain shift.

## Look Up the Number

On review of several important neurosurgical and neurologic textbooks, the threshold of ICP abnormality has gradually risen to 15 or even 20. The Brain Trauma Foundation guidelines proposed a threshold of 22 mm Hg to start ICP-reducing therapies, further obfuscating the idea of a threshold [11]. The rationale of this increase remains unclear, and therefore it is important to review studies that established a good correlation between ICP and outcome.

Others showed that sustained ICP at lower levels between 15 and 20 mm Hg, could lead to worse outcome [12]. For a period, cerebral blood flow and cerebral perfusion pressure were regarded as equally important—as, indeed, they are—but as previously noted, increased ICP can cause shifts, shifts can cause ischemia, and ischemia can cause increased ICP. This principle was recognized by clinicians and pathologists and remained one of our core principles regarding increased ICP after traumatic brain injury.

At the end of the day, how can we answer the obvious question of what threshold number defines increased ICP? What number should trigger a call from the attending neurosciences nurse? Well…it depends! And even the use of these numerary thresholds does not tell the full picture of changes in autoregulation and cellular dysfunction. Moreover, we have come to realize that refractoriness of increased ICP is more important than absolute ICP values.

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