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HYPERVENT-CHECK: A Checklist for Therapeutic Hyperventilation in Neurocritical Care

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Introduction

In the past 30 years, changing evidence has led professional societies to update their position regarding therapeutic hyperventilation (HV). The Brain Trauma Foundation 2017 guidelines[1] discourage prophylactic HV ($PaCO_2 < 30$ mm Hg) in the initial 24 hours following severe traumatic brain injury due to the risk of worsening cerebral ischemia and eliminating any neuroprotective effect. Instead, they endorse restricted HV as a temporary "rescue" intervention in the context of acute, life-threatening intracranial hypertension—ideally only until definitive therapies (e.g., osmotherapy, decompressive surgery) can be established.[1] Likewise, the American Association of Neurological Surgeons and the Neurocritical Care Society have endorsed these recommendations, noting that extended hypocapnia can be detrimental by impairing microcirculatory perfusion and autoregulatory function.[2] Despite the presence of these concordant statements, clinical practice remains heterogeneous.

Therapeutic Hyperventilation

HV has remained a contentious but long-standing function in the treatment of acute intracranial hypertension.[3–6] By decreasing arterial carbon dioxide tension ($PaCO_2$), HV causes cerebral vasoconstriction, which subsequently decreases cerebral blood volume and promptly lowers intracranial pressure (ICP).[5] The physiological rationale for this intervention was well illustrated in animal studies and early clinical series, in which moderate lowering of $PaCO_2$ (to ~30–32 mm Hg) caused a prompt and reproducible lowering of ICP. Yet, the same studies—most importantly by Muizelaar et al.[7] in 1991—demonstrated that vasoconstriction may impair cerebral blood flow (CBF) and regional oxygenation, potentially causing ischemic damage in the penumbra of injured brain tissue. In an attempt to solve this loophole, we hereby introduce HIPERVENT-CHECK. This is a six-point, suggestion-only, concise, evidence-based transient therapeutic HV initiator and protective list.[3–6]

Conceptual Development and Rationale

An exhaustive, formal decision aid for such "gray-zone" settings is surprisingly not available in the literature. Current scales of severity—like the Glasgow Coma Scale, Marshall CT (computed tomography) score, or FOUR score—yield prognostic information but are not used directly to inform the timing or appropriateness of HV. Likewise, multimodal monitoring algorithms include HV as part but presume easy access to specialty hardware. There is still an acute disconnect between high-fidelity physiology and the street-level requirements of emergent neurocritical care providers. To create a feasible bedside cue for HV, we synthesized essential evidence and guideline suggestions into four broad areas:

Indications for HV

Sustained ICP increase, i.e., >25 mm Hg despite optimized initial first-line treatments (head elevation, sedation, osmotherapy), is the primary physiologic stimulus for transient HV.[7] Acute herniation symptoms (e.g., unilateral mydriasis or decorticate posturing) are robust, high-specific indicators that prompt ICP reduction and are required to avert catastrophic brainstem compression.

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Risks of Sustained HV

While HV rapidly decreases ICP, vasoconstriction secondary to hypocapnia can compromise CBF and precipitate ischemia in the injured penumbra. Positron emission tomography and near-infrared spectroscopy studies demonstrate that $PaCO_2$ declines below 30 mm Hg are correlated with regional hypoperfusion and decreased oxygen delivery, emphasizing the narrow therapeutic window for HV.

Need for a Checklist

Under emergencies or low-resource environments, sophisticated neuromonitoring equipment like brain tissue oxygen tension ($PbtO_2$), jugular venous oximetry, or continuous transcranial Doppler could be unavailable.[8] Even in well-resourced centers, the logistics of invasive probe deployment can cause delays when every second matters.[9] Physicians in such situations fall back on simple ICP monitoring, stat arterial blood gas, and patient neuro-examination to implement HV, underutilizing in extreme situations or overutilizing with resultant ischemic injury.

HIPERVENT-CHECK Tool

The HIPERVENT-CHECK tool is a suggestion-only checklist to guide transient HV in neurocritical care. Consider HV (target $PaCO_2$ 30–32 mm Hg) if ≥4 items are met, including Items 1 and 2 (►**Table 1**). In a unifying evidenced record of intracranial hypertension, symptomatology of herniation, basal line $PaCO_2$ monitoring, stratification for the risk of ischemia, uttered time limit words, and multi-man opinion aggregation in one utility, HIPERVENT-CHECK attempts to implement standardized decision-making wherein limited or absent neuromonitoring is employed. It is offered crisply as an unvalidated best-practice query—intended for immediate adoption during emergency contexts to provide increased situational awareness, safety of the patient, and establishment of the framework for eventual formal assessment. In the subsequent sections, we introduce the mechanistic underpinnings of HV, summarize the landmark clinical trials and guideline recommendations, and sketch the rationale for developing the HIPERVENT-CHECK tool. Next, we fully detail the checklist, its possible uses and limitations, and provide recommendations on how to integrate it into neuro-intensive care unit (neuro-ICU) workflows. We hope to provide clinicians with an actionable, evidence-based approach that closes the gap between physiologic principles and emergent bedside decision-making.

Potential Advantages

HIPERVENT-CHECK converts successful concepts into a quick, six-step bedside prompt. By mandating documented intracranial hypertension and clinical evidence of herniation, it protects against indiscriminate HV. Mandatory $PaCO_2$ monitoring provides for appropriate titration, and ischemia screening by simple imaging increases safety awareness. Time constraints (<6 hours) and clear team communication are factors in risk reduction against clinically impacting hypocapnia. Though this checklist has not been validated, it does encapsulate expert consensus recommendations and major evidence. It can also act as an initiating discussion document in ICUs that lack full neuromonitoring capabilities or in disordered emergency environments to engender even-handed situational awareness and ensure patient safety.

Limitations

This checklist is nonvalidated; in other words, the tool has not been tested retrospectively or prospectively, and performance parameters (sensitivity/specificity for good outcome) are unknown. We assume the availability of at least minimal ICP monitoring and imaging may lack generalizability in all low-resource environments. Finally, the dichotomous yes/no format may have the potential to oversimplify sophisticated physiology; clinical judgment remains important.

Conclusion

HIPERVENT-CHECK is a brief, suggestion-only checklist to support the judicious, short-term use of HV in neurocritical care. HIPERVENT-CHECK aggregates high-grade evidence-based initiators and safety screens into one tool for use with rapid decision-making. We recommend that ICU teams implement HIPERVENT-CHECK as an ideal practice prompt and make their experience available to support subsequent validation studies.

Table 1 HIPERVENT-CHECK tool

Item	Question	Yes/No
1. Sustained ICP elevation	Is ICP >25 mm Hg despite first-line measures?	[ ] Yes [ ] No
2. Clinical herniation signs	Are there acute herniation signs (e.g., unilateral mydriasis, decorticate posturing)?	[ ] Yes [ ] No
3. $PaCO_2$ monitoring	Can you measure $PaCO_2$ via arterial blood gas or end-tidal CO_2?	[ ] Yes [ ] No
4. Risk of ischemia	Does neuroimaging show no extensive infarction or low-flow areas?	[ ] Yes [ ] No
5. Defined duration	Is HV intended only as a transient bridge (<6 hours) to definitive care?	[ ] Yes [ ] No
6. Team awareness	Has the care team been briefed on HV risks and the plan for tapering?	[ ] Yes [ ] No

Abbreviation: HV, therapeutic hyperventilation.

Conflict of Interest
None declared.

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