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Application note

Philips HeartStart Intrepid monitor/defibrillator TBI advisory

Overview

The TBI advisory feature on the HeartStart Intrepid assists in the monitoring of patients who are determined at high risk of having suffered a traumatic brain injury. The TBI advisory provides visual guidance to help prevent the following conditions:

- Hypoxia – low blood oxygen saturation (as measured by SpO_2)
- Hypotension – low systolic blood pressure
- Hyperventilation-induced hypocapnia – decreased carbon dioxide in the blood (as measured by $EtCO_2$).

The TBI advisory is available for both adult and infant/child patients on devices in Monitor Mode configured with SpO_2, NBP, and $EtCO_2$. The TBI advisory display reflects the appropriate target limits for each TBI Care parameter.

Introduction

Every year, 2.2 million people in the United States suffer a traumatic brain injury (TBI), with approximately 52,000 deaths and 280,000 hospitalizations.[1] The economic burden of TBI is more than USD 60 billion per year.[1] TBI occurs when an external mechanical force inflicts sudden trauma to the head disrupting the normal function of the brain.[2]

However, subsequent preventable (secondary) injury to the brain can add dramatically to the primary injury, and the extent of damage during this time is significantly influenced by the early care the patient receives.[3] The emphasis on early recognition and early treatment of TBI should be similar to that of other pathologies where survivability and recovery are time-dependent, i.e., sudden cardiac arrest.[4]

TBI Pathophysiology[5]

The brain accounts for the consumption of 20% of bodily oxygen and 25% of the blood glucose. Because of this, cerebral blood flow (CBF) is critical for ensuring adequate brain metabolism and preventing cerebral ischemia. CBF is a function of cerebral perfusion pressure (CPP) and cerebral vascular resistance (CVR), CPP is calculated by subtracting intracranial pressure (ICP) from mean arterial blood pressure (MAP) (CPP= MAP-ICP). Normal ICP ranges from 10-15 mmHg, and normal MAP ranges from 70-95 mmHg; therefore, the average CPP is approximately 60-80 mmHg. CBF is regulated through several mechanisms in the healthy patient; however, disruption of those mechanisms can have catastrophic consequences on the brain and its control of body systems.

ICP and TBI[5]

The cerebrum, cerebellum, and brainstem use approximately 80% of the intracranial space (1,200 cc), with blood vessels and cerebral spinal fluid utilizing the remaining volume at 12% (150 cc) and 8% (90 cc), respectively. Cerebral swelling and hemorrhage secondary to TBI increase the cerebral volume and require the body to compensate for the increased volume by reducing the volume of other brain structures and cerebrospinal fluid within the cranium. If the amount of room available to compensate for the increasing volume is less than the expanding volume, then an increase in ICP will occur. Initially, the brain compensates for an increase in cerebral mass by compressing the cerebral venous blood vessels and, as cerebral volume continues to increase, cerebrospinal fluid is pushed from the skull into the spinal space. The initial compensatory mechanisms can maintain ICP close to normal; however, once those mechanisms are exhausted, ICP quickly rises.

The continued increase in ICP impedes arterial flow into the cranial space and constricts cerebral blood vessels. These mechanisms decrease CBF and, subsequently, the cardiocerebrovascular system attempts to compensate for the decreased CBF by increasing SBP. This further increases ICP.

The decreased CBF also causes cerebral ischemia and neuronal hypoxia. If spontaneous ventilation is compromised, pCO_2 increases, and this results in cerebral vasodilation and additional increases in ICP. Cerebral edema secondary to neuronal injury and cerebral ischemia leads to a further exacerbation of ICP. Ultimately, the continued increase of ICP will lead to profoundly compromised CBF, resulting in death. Figure 1.

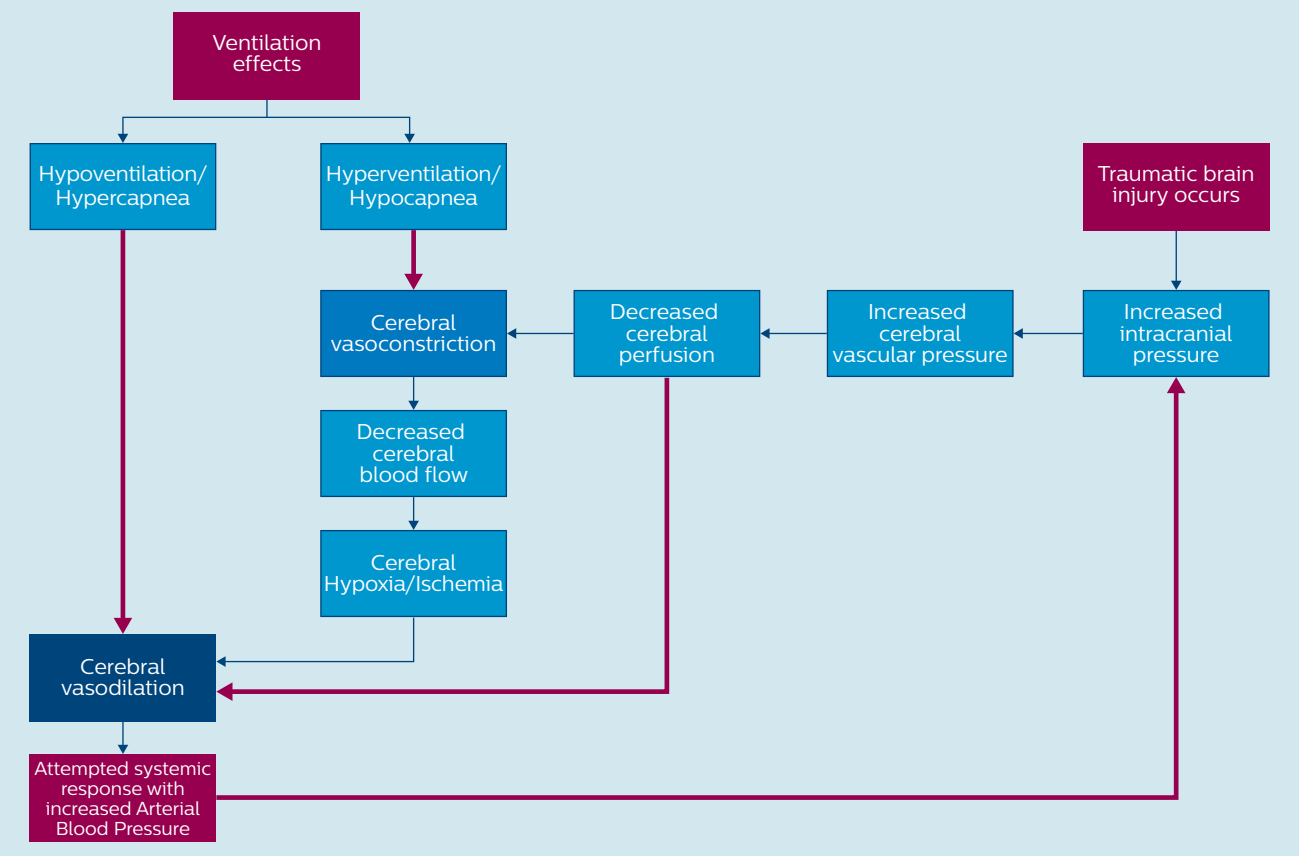


Figure 1: Relationship between increased intracranial pressure and decreased cerebral perfusion.

Three critical factors of TBI (the 3 H-Bombs)

The National EMS TBI Guidelines published by the Brain Trauma Foundation emphasize the importance of maintaining adequate cerebral oxygenation and perfusion by avoiding three critical factors.[7]

- Hypoxia
- Hypotension
- Hyperventilation (that leads to hypocapnea and cerebral vasoconstriction)

A single incidence of any one of these factors dramatically increases patient mortality.[3] However, maintaining End-Tidal Carbon Dioxide ($EtCO_2$), arterial blood oxygenation (SpO_2), and a systolic blood pressure (SBP) within guidelines established by the Traumatic Brain Injury Foundation have demonstrated improved outcomes in patients with severe TBI.[3]

Hypocapnia

Preventing hyperventilation induced hypocapnia ($EtCO_2$<32 mmHg) is critical because of its direct correlation to CBF.[3,7] For example, a patient who has a pCO_2 of 40 mmHg has a CBF of 50 ml per 100g of brain tissue per minute.[8] However, if pCO_2 drops to 30 mmHg, then CBF will drop to 40ml per 100g of brain tissue per minute, a 20% decrease in CBF.[8] The relationship between CBF and pCO_2 is so delicate that a 1 mmHg change in pCO_2 can alter CBF up to 5% and decreasing pCO_2 by relatively little (i.e., <32 mmHg) is associated with significant mortality.[3,8-10]

Unfortunately, unrecognized inadvertent hyperventilation in TBI patients is still a major problem, at least in the prehospital environment. In the San Diego RSI Trial, 59% of patients intubated before arriving at the hospital experienced at least one incident of hyperventilation resulting in an $EtCO_2$ value of less than 25 mmHg.[9] The importance of monitoring $EtCO_2$ cannot be overstated. One study observed that prehospital intubation in a setting where capnography was not used reported a mortality rate of 33%; conversely, the ability to monitor patients' $EtCO_2$ level and use a ventilator was associated with a statistically significant, 18% lower relative rate of death.[11]

Hypotension and hypoxia

Multiple studies have demonstrated a strong correlation between a single episode of hypotension (SBP<90 mmHg) or hypoxia (SpO_2<90%) and decreased patient survival.[7,12-14] Independently, hypotension or hypoxia have a dramatic deleterious effect on patient mortality. However, when a patient experiences both hypotension and hypoxia, the adjusted probability of death doubles again (beyond that of either alone).[15]

The Excellence in Prehospital Injury Care (EPIC) TBI study examined the outcome of 13,151 patients who had suffered major (moderate/severe/critical) TBI over a seven-year period. The researchers compared the mortality rate of patients who experienced neither hypoxia nor hypotension (n=11,545), hypotension only (n=604), hypoxia only (n=790), or hypotension and hypoxia (n=212) in the prehospital environment.[15] The study concluded that having at least one episode of hypotension or hypoxia (alone) resulted in a 20.7% (n=125) and 28.1% (n=222) mortality rate, respectively. The combination of both hypoxia and hypotension was associated with a 43.9% (n=93) mortality rate.[15]

Comparatively, patients who did not experience hypoxia or hypotension had a 5.6% (n=644) rate of death.[15] The adjusted analysis found that a patient that experiences both hypotension and hypoxia in the prehospital environment has a 600% (aOR = 6.1) higher likelihood of dying than someone who experiences neither.[15]

Another important finding of the EPIC TBI study was the absence of any identifiable SBP threshold in relation to patient mortality. The study identified that over the range of 40 mmHg to 120 mmHg SBP there was a consistent decrease in survival for every 10 mmHg drop and a progressive association between decreasing SBP and both unadjusted and adjusted risk of death.[1] In other words, a patient with a SPB of 60 mmHg is 4 times as likely to die than a patient with a SBP of 135 mmHg.[1] The implications are obvious: the injured brain is highly sensitive to a lack of perfusion, and the levels of systemic blood pressure associated with increased mortality may be far above the "classic" threshold for "hypotension" (SBP<90 mmHg).[1]

Importance of treating and preventing hypotension, hypoxia, and hypocapnia[10]

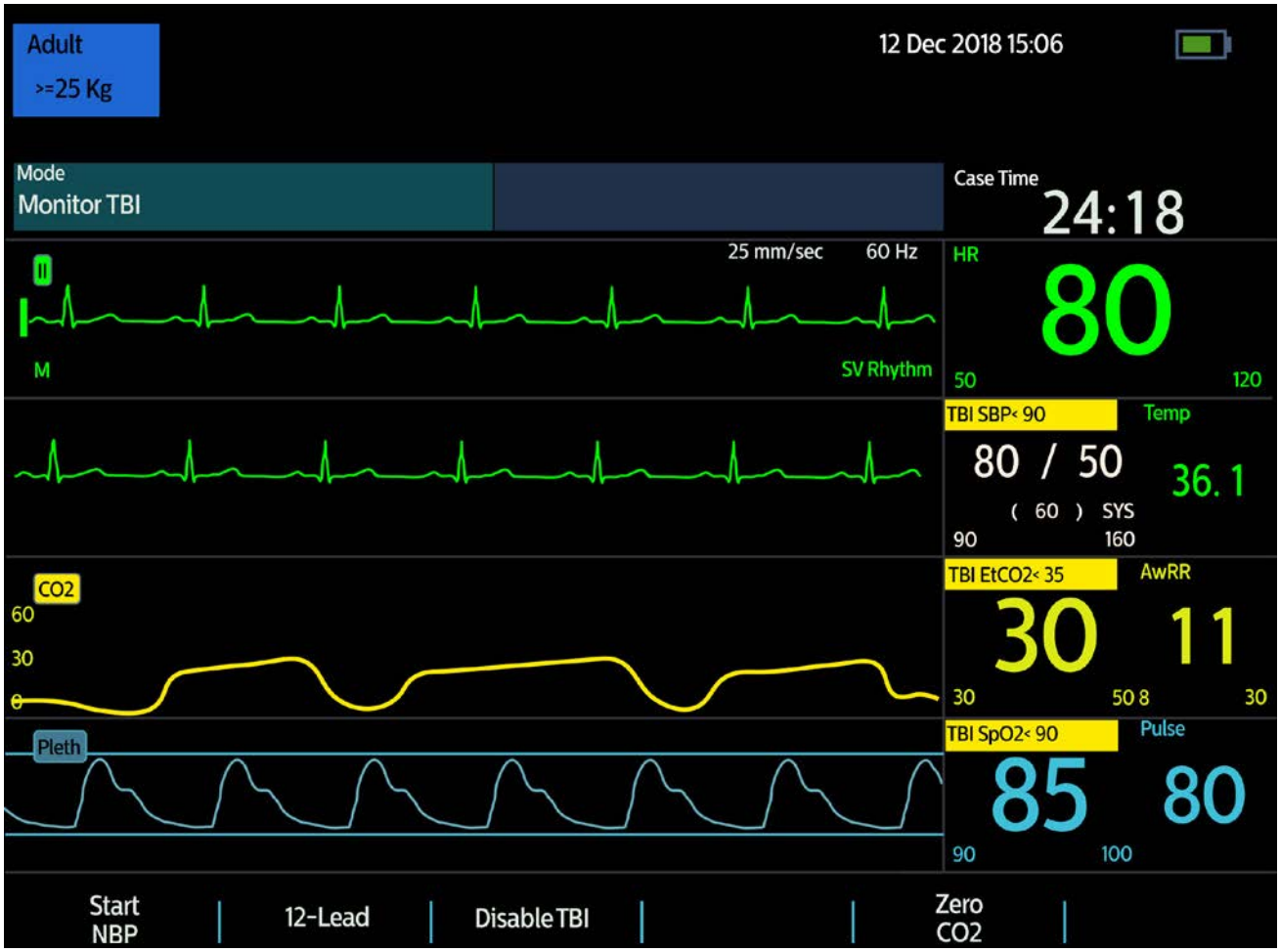
Researchers from the EPIC TBI study worked with Arizona EMS agencies to implement prehospital TBI guidelines focusing on three areas of prevention and treatment (the three "H-Bombs"):

- Preventing and treating hypoxia by use of high-flow O_2 and emphasis on basic airway maneuvers
- Preventing and treating hypotension by rapidly infusing isotonic fluids
- Preventing hyperventilation by using appropriate ventilation rates, adjuncts, and $EtCO_2$

In the EPIC Study, adjusted survival to hospital discharge doubled (aOR 2.03) for patients with severe TBI (head region severity score = 3-4) and tripled (aOR 3.28) for patients with severe TBI that also received positive pressure ventilation (PPV) via bag-valve mask or endotracheal intubation (ETI). The increased survival of patients with severe TBI receiving PPV was attributed to the emphasis on maintaining $EtCO_2$ between 35 and 45 mmHg, rate-timers that gave visual cues for proper manual ventilation and using flow-controlled ventilation bags to help prevent hyperventilation and overventilation.

One of the most important findings of the EPIC Study was that the initial improvement of patient survival faded over time, and this was most likely because of the diminished emphasis on following the prehospital TBI guidelines. The researchers could not require retraining of the study participants. Therefore, a method of ensuring that hypoxia, hypotension, and hypocapnea are recognized early should be available to all prehospital providers. The authors concluded that their findings make it likely that the use of monitoring technology that can give cues and real-time feedback to providers during the resuscitation of brain injured patients will improve outcomes by helping to prevent secondary brain injury from hypoxia, hypotension, and hyperventilation.

HeartStart Intrepid Traumatic Brain Injury advisory



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The TBI Advisory is available for both adult and infant/child patients on devices in Monitor Mode configured with SpO_2, NBP, and $EtCO_2$. The TBI Advisory display reflects the appropriate target limits for each TBI Care parameter.

Notes:

- These limits must be pre-established and pre-configured prior to using this feature (see "Configuration – TBI Advisory" in the HeartStart Intrepid Instructions for Use).
- If a physiological alarm condition occurs, it will have a higher priority than the TBI advisory and will obscure the TBI Indicators.

Enabling the TBI advisory

1. TBI: Ensure an SpO_2 sensor, NBP cuff, and $EtCO_2$ tubing are connected to the patient
2. Press the [Enable TBI] soft key.

If no patient age has been entered, the Patient Age number selector appears.

3. If prompted, enter the patient age.

The patient age is displayed in the status area next to patient name/ID.

When age is entered, mode text changes from Monitor to Monitor TBI.

Notes:

- The TBI Advisory is only available in Monitor Mode and is not available during the acquisition of a 12-lead ECG.
- Devices must be configured with SpO_2, NBP, and $EtCO_2$ measurement parameters, and all three parameters must be in use.
- TBI Advisory can only be enabled when TBI limits are pre-configured on the device.
- The neonatal patient category is not supported.

Displaying the TBI advisory

The TBI advisory display appears in the numeric parameter area. A TBI limit bar is displayed at the top of the corresponding measure box for NBP, $EtCO_2$, and SpO_2.

TBI for systolic blood pressure (SBP)

Systolic blood pressure that falls outside the established TBI limits is indicated by a yellow bar, and TBI SBP > High Limit or TBI SBP < Low Limit is displayed, as shown in Figure 2.

Figure 2: TBI outside limits.

Systolic blood pressure within limits the setting for the TBI configuration is indicated by TBI SBP on a green bar, as shown in Figure 3.

Figure 3: TBI within limits.

TBI for $EtCO_2$

If the $EtCO_2$ value falls outside the established TBI limits, TBI $EtCO_2$ > High Limit or TBI $EtCO_2$ < Low Limit is displayed in a yellow TBI bar as shown in Figure 4.

Figure 4: TBI outside limits.

If the $EtCO_2$ value is within the limits setting for the TBI configuration, TBI $EtCO_2$ is displayed in a green bar as shown in as shown in Figure 5.

Figure 5: TBI within limits.

WARNING: To ensure correct functioning of the CO_2 measurement, use only approved CO_2 accessories as listed in the HeartStart Intrepid Instructions for Use, Chapter 18: Supplies and Accessories.

TBI for SpO_2

For SpO_2, there is no upper limit, only a lower limit threshold for SpO_2 is set. If the SpO_2 value falls below the established TBI limit, TBI SpO_2 < Low Limit is displayed on a yellow TBI bar as shown in Figure 6.

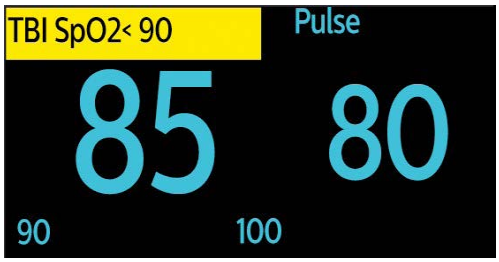

Figure 6: SPO_2 below TBI limit.

If the SpO_2 value is above the limit set for the TBI configuration, the TBI bar is green, and the text TBI SpO_2 is displayed on a green bar as shown in Figure 7.

Figure 7: SPO_2 above TBI limit.

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