

Chapter 61

Intra-Aortic Balloon Pump (IABP) in ICU: Cerebral Hemodynamics Monitoring by Transcranial Doppler (TCD/TCCS)



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Key Points

1. Intra-aortic balloon pump (IABP) modifies the pattern of arterial blood pressure pulsatility leading to concurrent changes in the cerebral blood flow (CBF) waveform during the cardiac cycle.
2. Transcranial Doppler ultrasound (TCD/TCCS) allows measurement of CBF velocity (CBFV) in the middle cerebral artery, or other large intracranial arteries, with considerable potential to identify cerebral hemodynamic disturbances in critically ill patients.
3. TCD can detect reversal of diastolic CBFV which is likely to be iatrogenic and should be minimized.
4. Microemboli produced by IABP should be evaluated with TCD to avoid neurological complications.
5. Patients suspected of clinical brain death need IABP to be on standby to allow a proper determination of the actual CBFV by TCD/TCCS.

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61.1 Introduction

Intra-aortic balloon pump (IABP) remains the most widely used form of mechanical circulatory support in current clinical practice [1]. The accepted clinical indications for IABP use are wide ranging, but the available clinical evidence is largely limited to cardiogenic shock, myocardial infarction without shock, high-risk percutaneous coronary intervention, and perioperative of cardiac surgery [1]. In neurological critical care patients, IABP has been used in subarachnoid hemorrhage (SAH) [2–4].

The physiological premise underlying the utilization of IABP was first described by Kantrowitz in animal models in 1953, based on the diastolic augmentation of aortic root and coronary pressure [5]. Percutaneous IABP was first applied clinically in 1980 and remains the most widely used form of left ventricular mechanical support in most centers [6].

The IABP is a flexible balloon catheter with two lumens—one to allow flushing, aspiration, and for aortic pressure to be transduced; the other to allow inflation/deflation with the rapid shuttling of gas (usually helium) to and from the balloon [7]. Triggering of the insufflation and deflation is synchronized by the trace of the electrocardiogram (ECG) or the systemic arterial blood pressure (ABP) [8]. Of considerable relevance, the control of triggering can be changed to change the time delay when inflation takes place after the QRS complex of the ECG, leading to changes in the temporal pattern of the ABP waveform as it will be discussed later.

In this procedure, a balloon is placed into the aorta just distal to the left subclavian artery, generally via the femoral artery [6]. The balloon is connected to an external pump through a catheter. The pumping is timed so that the balloon inflates immediately after the aortic valve closes. Balloon inflation decreases diastolic aortic runoff and increases diastolic aortic pressure; these changes increase perfusion of the coronary arteries. The balloon deflates just before systole. Balloon deflation results in systolic unloading and a decrease in the aortic impedance to left ventricular ejection [1, 9] (Fig. 61.1). This combination causes relatively little change in the mean aortic pressure but decreases the left ventricular pressure by approximately 20% and increases coronary perfusion and cardiac output by up to 40%. As a consequence of these hemodynamic changes, cardiac workload and myocardial oxygen consumption are reduced in patients with a failing heart in cardiogenic shock or heart failure [10, 11].

IABP use is not free from complications. Postmortem examinations have shown multiple etiologies, including cerebral air embolism secondary to IABP rupture, compromised spinal cord blood supply due to mechanical trauma, dissecting hematoma of the thoracic aorta, spinal cord necrosis, microatheroembolism to radicular arteries, and other minor cerebrovascular events [12–15].

Given ongoing concerns about the effects of cardiac surgery on cognitive impairment and perioperative hypoperfusion stroke, the role of IABP on alterations of cerebral hemodynamic parameters is of considerable interest.

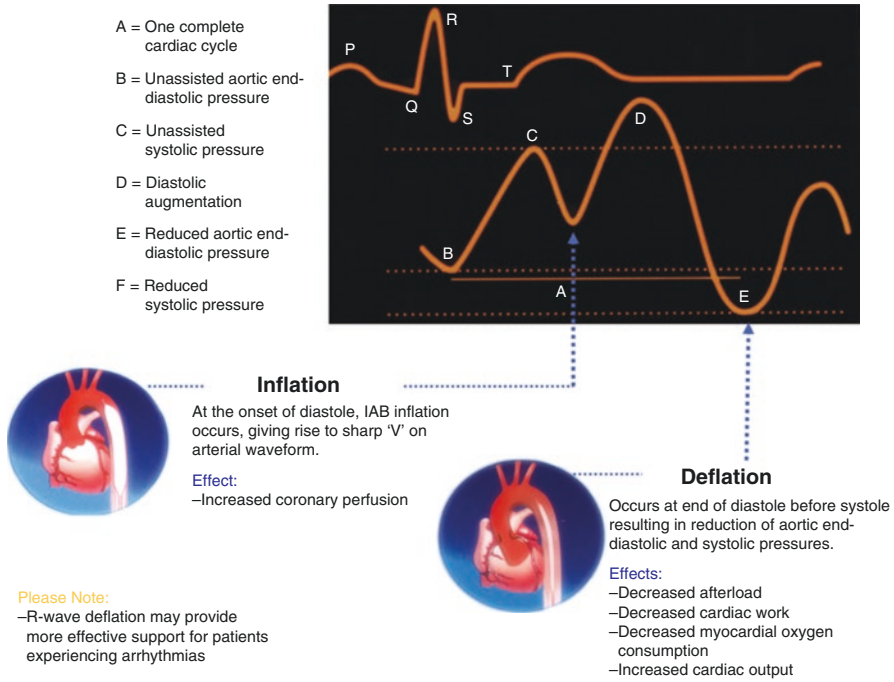


Fig. 61.1 The pressure waveform transduced from the tip of the IABP demonstrates a reduction in systolic pressure and augmentation of diastolic pressure with counterpulsation. (Courtesy: Murlin and Zacharowski [51])

61.2 Neuro ICU: Intra-Aortic Balloon Pump (IABP)

In the neuro intensive care unit, the IABP has been used in patients with subarachnoid hemorrhage (SAH) [2, 4, 16] in two distinct clinical situations. The first one is the neurogenic stunned myocardium, that is, a severe condition characterized by reversible left ventricular dysfunction [17]. Neurogenic stunned myocardium following SAH occurs in 20–30% of SAH patients and may result in several complications such as cardiac arrhythmia, pulmonary edema, and prolonged intubation, which can negatively impact morbidity and mortality as well as long-term recovery [17]. Patient management remains basically supportive with case reports acknowledging the utility of contractility vasopressors such as dobutamine and milrinone [17]. Limited data also suggested the utility of IABP in patients who require hemodynamic support when neurogenic stunned myocardium is accompanied by cardiogenic shock [4, 18].

The second clinical situation reported is when patients develop refractory cardiogenic shock despite vasopressor use in cerebral vasospasm after SAH [2, 18]. Cerebral vasospasm is a major cause of morbidity and mortality following

aneurysmal SAH and contributes to delayed cerebral ischemia [19]. The combination of induced hypertension, hypervolemia, and hemodilution (triple-H therapy) is often used to improve cerebral perfusion pressure following cerebral vasospasm [20]. Although this approach has gained widespread acceptance over the past 20 years, the efficacy of triple-H therapy in the management of the acute phase of SAH may be questioned [19]. The IABP can improve the hemodynamic status of patients, who would probably die without such therapy and allow the continuing use of triple-H therapy [3].

TDC is a standard screening tool for vasospasm, but the predictive value of mean flow velocity (MFV) is likely inaccurate in the setting of an IABP, as the aforementioned alterations in the arterial waveform are reflected in the TCD velocity waveform (Fig. 61.1). There is just one study that explored this topic; Morris and colleagues reviewed cases of SAH that underwent same-day TDC and angiography [2]. TDC/TCCS waveforms were assessed for MFV, peak systolic velocity (PSV), balloon pump-augmented diastolic velocity, and a novel feature that they called “delta velocity” (balloon pump-augmented velocity—systolic velocity) [2]. The authors concluded that delta velocity, a novel transcranial Doppler flow velocity feature, may reflect vasospasm in patients with SAH with IABP support. The MFV, usually considered the most accurate TCD/TCCS measure of proximal vasospasm, was not significantly correlated with proximal vasospasm, although it was correlated with distal vasospasm [2]. However, these data are from a single, retrospective observational study. Clearly, interpretation of the waveform in patients with IABP for vasospasm detection requires further investigation.

61.3 Cerebral Blood Flow: IABP Effects

Although its benefits for improving cardiac function have been well documented, the effects of IABP on the cerebral circulation are still not well understood. Studies investigating the effects of IABP on the cerebral circulation provide conflicting results and have suggested that IABP either decreased, increased, or even transiently reversed cerebral blood flow [21–23]. What is clear though is that the use of IABP can change the temporal pattern of CBFV waveforms during the cardiac cycle, leading to characteristic double-peaked waveforms (Figs. 61.2 and 61.3) and the occurrence of transient reversed diastolic (i.e., negative) values of CBFV recorded with TCD [21, 24].

Cheung et al. reported in their study with 19 patients that IABP modified the pattern of CBFV in the middle cerebral artery (MCA) as measured by TCD but did not affect mean CBFV, even at different magnitudes of augmentation or trigger ratios [21]. They also concluded that IABP modified the phasic profile of cerebral blood flow to reflect the arterial pressure waveform (Figs. 61.2 and 61.3), and this pattern was not showing on standby mode.

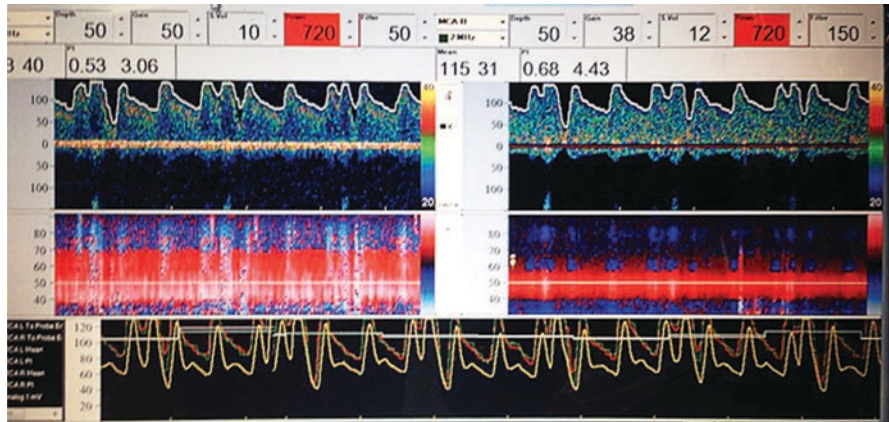


Fig. 61.2 Continuous recording of blood pressure (yellow tracing) and cerebral blood flow velocity (top, right and left middle cerebral arteries) from a 55-year-old male patient with IABP ratio 1:2 showing the double-peaked waveform

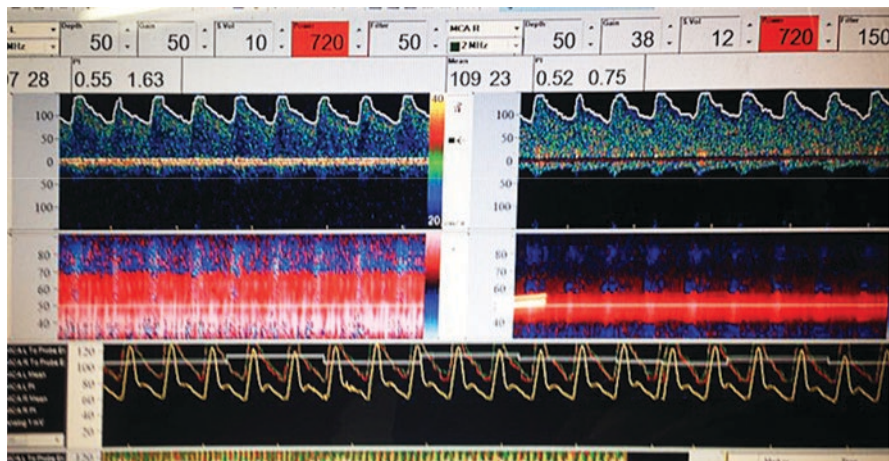


Fig. 61.3 Continuous recording of blood pressure (yellow tracing) and cerebral blood flow velocity (top, right and left middle cerebral arteries) from the same patient in Fig. 61.1 showing the IABP in standby mode

Notwithstanding some studies reported that the IABP device probably not changes the MFV. Currently, this device uses a square-wave function to control gas flow into the balloon [8], and with TCD, it may be possible to devise nonlinear inflation/deflation sequences for intra-aortic balloon pumps that can maximize forward blood flow in the intracranial circulation [24].

61.3.1 Transient Reversed Diastolic CBFV

Late diastolic IABP flow reversal in CBFV was first described in 1990 when TCD was used to evaluate CBF in three patients with an IABP [24]. This phenomenon is not uncommon; studies suggested that waveform flow reversal in intracranial vessels may occur in up to 35% of cardiac surgery patients supported by IABP [22].

Under normal conditions, forward CBFV is detected in both systole and diastole with TCD. The reversal of diastolic CBFV implies negative CBF in diastole (Fig. 61.4) and has also been described in other cerebrovascular conditions such as intracranial hypertension, intracranial circulatory arrest, in the first minutes after acute SAH, in metabolically compromised comatose patients, and, in the posterior circulation, the subclavian steal syndrome [24, 25]. However, the clinical significance of this dramatic alteration in the CBF waveform pattern is still unknown and more research on its implications is urgently needed [26].

Reversal of diastolic CBFV has been reported in a few case studies in the literature [27, 28]. The reversal or absence of diastolic flow at late diastole is probably due to diastolic pressure rapid reduction induced by deflation of IABP, so this phenomenon is a direct cause of IABP utilization (Fig. 61.4). These case reports suggest that reversal of diastolic CBFV is iatrogenic and it should be avoided. One

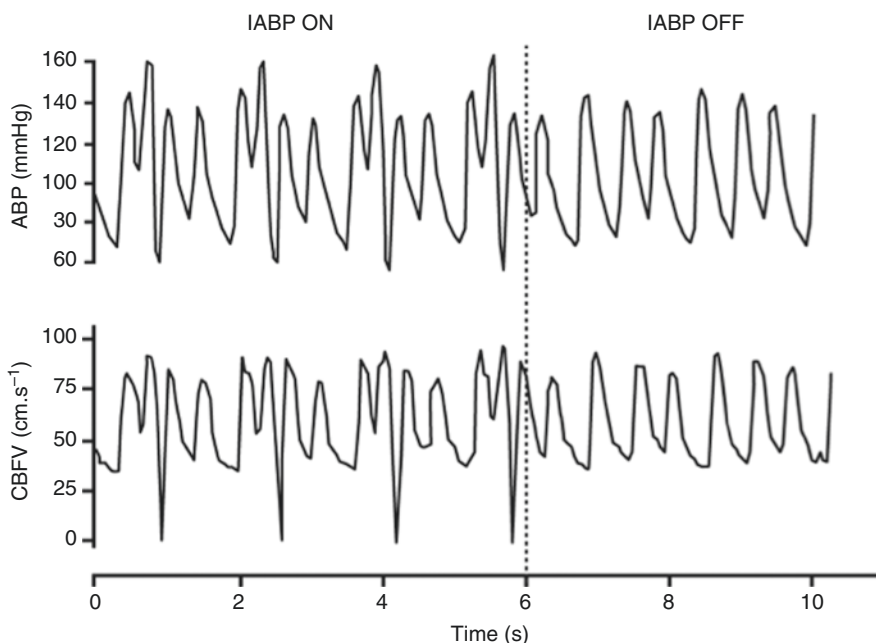


Fig. 61.4 Continuous recording of blood pressure and cerebral blood flow velocity from a 63 year-old male patient with IABP ratio 1:3 showing the transient reversed diastolic and the moment of balloon withdrawal (vertical dashed line). ABP Arterial blood pressure, CBFV Cerebral blood flow velocity, IABP Intra-aortic balloon pump. (Courtesy: Caldas et al. [37])

possible approach is to optimize the balloon inflation/deflation cycle, by moving deflation to the absolute end of diastole [24, 27]. Although the precise effects of CBFV diastolic reversal on cerebral hemodynamics are still unknown, its departure from normal physiological waveforms is likely to be undesirable and to have a negative effect on CA.

TDC available in ICU, as an easy-to-use, safe, and low-cost bedside tool, is advantageous to identify the reversal of diastolic CBFV in patients with IABP. Furthermore, it could be helpful to assess the hemodynamic response to changes in the balloon deflation timings to minimize the flow reversal phenomenon.

61.4 IABP: Cerebral Autoregulation

The main reason why IABP might disturb CBF and/or its regulatory mechanisms is due to observed changes in CBF temporal patterns. Chiefly among these regulatory mechanism is cerebral autoregulation (CA) that maintains cerebral perfusion within strict limits despite changes in mean ABP in the range 60–150 mmHg [29].

Assessments of CA are generally classified as being “static” or “dynamic” [30]. Static CA refers to the steady-state relationship between ABP and CBF [30, 31]. Dynamic CA reflects the transient response of CBF, often recorded as CBFV with TCD to rapid changes in ABP [31]. Impairment of CA renders the brain less tolerant to both low and high ABP, with increased risks of significant brain oligemia and hyperemia, respectively [32]. Assessment of CA in critical care patients and those with IABP could be an important bedside tool to avoid these complications [50].

There are numerous methods of quantification and assessment of CA in use at the current time, each with their own inherent assumptions, caveats, and specific analytical models. Importantly, no particular method is currently considered to be the “gold standard” [33].

Hypo- and hyper-perfusion, resulting from disturbances in cerebral autoregulation, are thought to be contributing factors in the neurological complications following cardiac surgery with cardiopulmonary bypass, and the use of IABP could also play a part in the development of these complications [34–36].

Hitherto, only two studies evaluated cerebral autoregulation in patients with IABP [37, 38]. These studies used different methods to assess cerebral autoregulation. Caldas et al. (2017) used transfer function analysis and the autoregulation index (ARI) to quantify the efficiency of CA [31, 37]. The second study (Bellapart 2010) was based on the time-delay between fluctuations in CBFV and ABP, estimated using a cross-correlation technique. Both studies found that cerebral autoregulation was intact in patients with IABP [37, 38].

One important corollary of these studies though was demonstrating the feasibility of performing bedside assessments of CA in critically ill patients requiring the use of IABP or different invasive support devices. Several critical care conditions were reported with impairment of cerebral autoregulation, such as sepsis, perioperative of cardiac surgery, ischemic heart disease, or SAH, and these conditions may be

requiring the use of IABP [39–44]. Patients who have manifested impairment of CA should have close monitoring and management of arterial blood pressure, to prevent ischemia or hyper-perfusion.

61.5 IABP and TCD/TCCS: Other Miscellaneous Conditions

61.5.1 Microemboli

Monitoring of the cerebral circulation with TCD can identify microemboli within the MCA during cardiopulmonary bypass procedures [45–47]. Similar techniques during the use of left ventricular assist devices may help identify occult cerebral embolization with IABP. Cerebral air embolism is a neurological emergency most commonly iatrogenic in origin [14]. It has been reported following invasive procedures such as cardiac catheterization, central venous catheter insertion, and cardiothoracic surgery. This complication, previously reported in patients with IABP when there is a rupture with gas embolization, is a rare but potentially fatal event [48].

61.5.2 Brain Death

The classical TCD pattern in brain death consists of three stages: oscillating or biphasic flow, systolic spike flow, and no flow [49]. With increasing intracranial pressure, diastolic flow ceases and systolic peaks become sharper. As cerebral perfusion pressure approaches zero, there is collapse of blood vessels during diastole, and so absent or reversed diastolic flow can be demonstrated.

The IABP can induce alterations in the pattern of CBFV measurements and the reversal of flow in late diastole may appear [24, 27]. Therefore, in a patient with an IABP, who is clinically brain dead, the IABP has to be on standby to allow a proper determination of the actual CBFV. The classic TCD pattern has to be detectable for at least 30 minutes [25] to confirm the diagnosis of brain death.

61.6 Conclusion

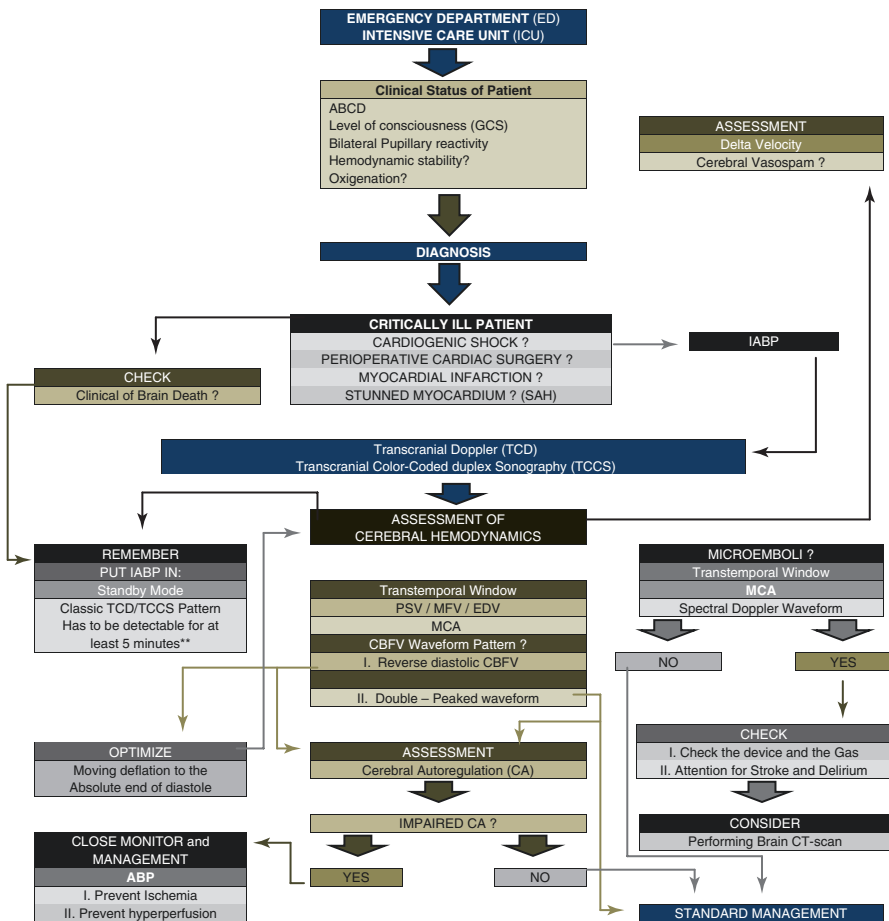
IABP significantly changes CBF temporal patterns in basal cerebral arteries. TCD provides valuable information about cerebral hemodynamics with intra-aortic balloon pumping, given the ability to measure subsecond changes in cerebral hemodynamics.

The IABP is an important resource to support the failing heart, in diverse clinical situations, but the alterations produced in the systemic circulation are transmitted to

the brain, resulting in alterations of the CBFV temporal pattern, whose implications still require more detailed investigation. The use of TCD for monitoring patients with IABP, and also performing a number of dedicated assessments, such as dynamic CA or brain death, is recommended.

Further studies are needed in patients with IABP to assess the extent of cerebral hemodynamic effects, particularly regarding the phenomenon of diastolic CBFV reversal.

Algorithm



ABCD Airway-breathing-circulation-disability, IABP Intra-aortic balloon pump, CA Cerebral autoregulation, ABP Arterial blood pressure, MCA Middle cerebral artery, CBFV Cerebral blood flow velocity, PSV Peak systolic velocity, MFV Mean flow velocity, EDV End-diastolic velocity, SAH Subarachnoid hemorrhage; ** If the complementary exam is needed

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