EDITORIAL COMMENT



Variation in CT perfusion protocol has implications on defining irreversibly damaged ischemic brain parenchyma

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Key Points

- Computed tomographic perfusion (CTP) is increasingly being used in the characterization of brain ischemia.
- Variations in post-processing protocols continue to be a challenge, resulting in a slight variation of CTP results.
- We need to adopt a universal acquisition protocol to help optimize output of CTP.

Computed tomographic perfusion (CTP) is increasingly being used in the characterization of brain ischemia [1]. Most commonly, these are used for acute ischemic stroke for the characterization of infarct core and ischemic penumbra. More recently, CT perfusion is also being used for the characterization of ischemic injury in critically ill patients—such as those suspected to have brain death in ICU [2, 3], comatose cardiac arrest patients [4], and those with severe traumatic brain injury [5, 6].

In their *European Radiology* article, Peerlings et al report the variations in post-processing protocols continue to be a challenge, resulting in a slight variation of CTP results [7]. The significance of variation depends on whether we look for penumbra (potentially reversible ischemia) or core (irreversible ischemic damage). To define the irreversibly damaged ischemic core, the generally acceptable threshold for cerebral blood flow (CBF) has been 15 mL/min/100 g of brain tissue and that for cerebral blood volume (CBV) of 2 mL/100 g of brain tissue [8, 9]. When comparing CBF and CBV, CBV is more likely to predict the irreversibly damaged ischemic core; i.e., if brain region is dead on CBV maps, it is unlikely to recover on follow-up irrespective of treatment [1, 8]. On the other hand, relative CBF is more sensitive in predicting the irreversibly damaged ischemic core [9].

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The perfusion parameters are calculated based on the time density curve (TDC) that depicts the rate of change of density of contrast during the first pass of the contrast bolus. The TDC is a function of the injection of the contrast bolus, heart rate, and cardiac output. The faster the contrast injection rate, the tighter the contrast bolus, which results in a sharper, narrower, and higher TDC. The faster the heart rate, the sharper and narrower the TDC. The corollary is also true for patients with slow heart rate and low cardiac output. Controlling patient physiological parameters such as heart rate and cardiac output is usually not done in the clinical setting where CTP is used. But the injection rate of contrast agent could be changed and should be standardized to compare CTP results across patients as well as across centers. Bolus arrival time and TDC width are good surrogate markers for the patient's physiology. A patient with a slower heart rate and a lower cardiac output usually has a longer bolus arrival time and a wider TDC.

This brings to attention another important CTP acquisition parameter, time of acquisition. If the total time of acquisition is not long enough, the whole cardiac cycle may not be covered during CTP acquisition, resulting in the truncation of the TDC. The shorter time of acquisition is largely because of excessive concerns of radiation dose. Recent guidelines have suggested a longer time of acquisition of 70-90 s to avoid any possibilities of truncation of TDC [10]. CBV is calculated as the area under the curve of TDC. CBF is calculated based on the ascending segment of TDC. Truncation of TDC negatively affects the calculation of CBV resulting in under-estimation of CBV, thereby over calling the volume of irreversibly damaged ischemic core. This has minimal to no effect in the calculation of CBF. Truncation of TDC is not uncommon and can be seen in up to 30% of cases [9]. This has resulted in transition from CBV to CBF as the preferred parameter to predict the

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irreversibly damaged ischemic core [9]. A relative CBF is better than absolute CBV in depicting the irreversibly damaged ischemic core. A threshold of absolute CBF of < 15 mL/100 g/min correlates with that of irreversibly damaged ischemic core [9].

Effects of truncation of TDC are also significant when CTP is used to predict devastating brain injury in patients other than those with ischemic stroke, e.g., those with severe traumatic head injury, comatose cardiac arrest, and suspected brain death [2–6]. Truncation artifacts can be controlled by increasing the time of acquisition of CTP, which results only in a miniscule percentage increase in the associated patient radiation dose.

As the use of CTP for ischemic stroke is increasing and the scope of CTP is expanding, a better understanding of the different factors that could influence the results of CTP is needed. With a better understanding, we need to adopt a universal acquisition protocol to help optimize output of CTP.

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Methodology

Editorial comment

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