CT assisted dynamic perfusion imaging (perfusion CT, PCT) has evolved in recent years with the introduction of the multi-slice spiral technique, the use of study protocols with lower injection rates and improved evaluation programmes. The method permits quantitative determination of cerebral blood flow, can be performed quickly, is economical and causes the patient little stress. It offers two decisive advantages in diagnosing strokes:

- Brain areas with disruption to perfusion can be detected without delay directly after the onset of the clinical symptoms.
- The present studies show that native CT, together with the parameter images from perfusion CT, enables the physician to make a distinction between the irreversibly damaged infarct core and the potentially reversibly damaged infarct penumbra. Consequently, it is increasingly possible to perform stroke therapy taking into account the patient’s individual blood flow status over and above strictly therapeutic windows. The following article briefly explains the basics of the method and the significance and interpretation of the various perfusion parameters.

The principle of dynamic perfusion CT

To obtain functional information about cerebral blood flow, in PCT a short intravenous contrast medium bolus is given during which one slice, or with MSCT, several CT slices, can be acquired repeatedly at fixed time intervals. Usually, for instance, 40 ml of contrast medium are administered with a scanning period of 45 seconds and an imaging frequency of 1 image/second.

The examination is based on the indicator dilution theory: following administration of an intravenous contrast medium bolus the X-ray density of

Fig. 1: Typical time/density curves after injection of a contrast medium bolus in perfusion CT.
The density sequences are imaged (idealised view) in an arterial vessel (middle branch of the cerebral artery: blue), a venous vessel (confluence of sinuses: yellow) and in the cerebral parenchyma (thalamus: red). Note the typical staggered time between the arterial and venous time/density curves and the flattened and slightly delayed density sequence in the parenchymal compared with the arterial curve (Fig. modified according to [3]).
the brain temporarily increases (Fig. 1). Conclusions about cerebral blood flow can be drawn from the extent and course over time of this increase in density. Using various mathematical algorithms parameters denoting cerebral perfusion are calculated and represented in the form of colour-coded parameter images. The most usual parameters are CBV, CBF, MTT and TTP.

**Cerebral blood flow (CBF)**

Cerebral blood flow is the most important parameter. It indicates how much blood is flowing through the brain tissues in a specific period, and it is measured in ml blood/100 g brain tissue/min. Normal values for CBF are between 50 and 80 ml of blood per 100 g of brain tissue per minute. Areas of the brain with high energy requirements such as the cortical surface or the basal ganglia exhibit CBF values which are some 2–3 times higher than those for white matter. Cerebral blood flow is controlled by continual changes in the diameter of the vessels and is kept relatively constant (auto-regulation). If the perfusion pressure rises, e.g. when the systemic blood pressure rises, the cerebral vessels constrict, if the pressure is lowered, they dilate. Only if the vessels in a particular area of the brain are already dilated to the maximum and the perfusion pressure is reduced still further, does the CBF decrease.

Below a CBF of 20 ml/100 g/min, the synaptic function of the nerve cells is retarded due to the lack of energy, i.e. there is neurological failure. This loss may, however, be completely reversible if blood flow is normalised again. Below a CBF of 10–15 ml/100 g/min the metabolism of the nerve cells can no longer be maintained. If CBF remains below this so-called ischaemia threshold for 2–10 minutes, the result is irreversible cell damage. In ischaemic cerebral infarcts around an irreversibly damaged infarct core with CBF values below 10–15 ml/100 g/min, there is frequently a margin of brain tissue in which the CBF is maintained by collateral vessels at 10 to 20 ml/100 g/min. The cells of this area known as an infarct penumbra are not neurologically functional, but they are not yet irreversibly damaged. Structural damage does not occur until hypoperfusion has been maintained in the penumbra for a longer time. This period, which may be many hours, cannot, however, be predicted in individual cases. The treatment of ischaemic cerebral infarcts is not directed on the already irreversibly damaged infarct core, but on the

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**Fig. 2: Normal results.**

The image is of a native CCT slice at the basal ganglia (Fig. A) and the corresponding colour-coded parameter images of the perfusion CT. The mean transit time (MTT, scales 0–14 s, Fig. B), the cerebral blood flow (CBF, scales 0–80 ml/100 ml/min, Fig. C) and the cerebral blood volume (CBV, scales 0–6%, Fig. D) have been calculated. The parameter images show largely symmetrical perfusion rates. It can be seen that perfusion of the white matters is slightly delayed compared with the basal ganglia (Fig. B). The CBF and CBV values at the cortical surface and in the basal ganglia are raised compared with those of the white matter (Fig. modified according to [3]).

**Fig. 3: Territorial infarct with penumbra.**

61-year-old patient with acute right hemiparesis. Native CCT (Fig. A) and perfusion CT (Figs. B–D) approx. 1 hour after onset of symptoms. The parameter images of MTT and CBF (Fig. B and C) show a large hypoperfused area in the left portion of the area supplied by the middle cerebral artery. In the initial native CCT (Fig. A) a slight fuzziness can be detected at the boundary between the cortex and the medulla which cannot be safely exploited. The area that can be distinguished in the CBV parameter image (Fig. D) with reduced CBF (infarct core) is clearly smaller than the lesion with disturbed perfusion identifiable on the MTT and CBF parameter images. Areas that show extended MTT or reduced CBF but no decreased CBV are described as infarct penumbra (tissue-at-risk). Thrombolysis therapy could not be undertaken because of contraindications. In the control scan (native CCT, Fig. E) a partial middle cerebral artery infarct could be detected comprised of both the infarct core initially visible and the penumbra (Fig. modified according to [3]).
tissue in the penumbra that may recover after perfusion rates have been brought back to normal (salvageable tissue, tissue-at-risk).

In interpreting perfusion scans the above details about the amount of CBF must be considered only as approximate standard values. The local CBF calculated not only fluctuates depending on the software used for evaluation but can also change depending on methodology, e.g. according to the injection speed of the contrast medium bolus or the patient’s reduced cardiac function. Furthermore, the CBF values of white matter are considerably lower than those of grey matter. Due to the limited spatial resolution of the method partial volume effects are unavoidably produced.

Cerebral blood volume (CBV)

Cerebral blood volume (CBV) is defined as the percentage of blood vessels in a specific volume of tissue. Highly vascularised areas of the brain such as the basal ganglia or the cortical surface therefore have a higher CBV than the less vascularised cerebral white matter. The CBV, however, is also a functional parameter and alters if vessel size changes in the context of vascular auto-regulation.

Unlike CBF, which in ischaemia is reduced both in the infarct core and in the penumbra, the CBV in the penumbra usually increases. This is caused by cerebral auto-regulation: the fall in CBF has to be compensated for by dilation of the vessels concerned. In contrast, in the irreversibly damaged infarct core, auto-regulation usually no longer functions, so that the CBV is decreased. This is very helpful in diagnosing strokes: areas showing reduced CBV in the acute stage of ischaemia are as a rule irreversibly damaged.

Parameters for describing delay in perfusion (TTP, MTT)

The most common of the parameters indicating retarded perfusion are mean transit time (MTT) and time-to-peak (TTP). There is a direct correlation between them and cerebral perfusion pressure. Even slight disturbances to the blood supply can lead to the MTT and TTP being extended. In clinical studies on strokes the MTT and TTP were found to be very sensitive to disruption in regional perfusion of the brain. Indeed this is not specific to ischaemia. Pathological MTT and TTP values are found both in the infarct core and in the penumbra, but may also be caused by prior clinically asymptomatic vessel stenosis (e.g. in the internal carotid artery) or vasospasm.

Literature