Brain venous study: the role of TCCD

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The use of ultrasound to study cerebral venous circulation is a very recent application of transcranial color coded Doppler sonography (TCCD). Several authors have attempted to use trancranial Doppler (TCD), which is a pulsed blind method, but in this area (the study of venous circulation) TCD reveals all its limitations. The insonation takes place in a blind manner and failure to detect the vessel can readily lead to misinterpretation. Whereas, through compressive tests (whose safety and utility is still debated), we are able to obtain useful information from arterial studies, in venous studies these tests cannot be performed – we absolutely have to "see" the vessel and/or venous sinus in order be completely certain of their insonation.
To detect correct peak systolic velocity (PSV) and peak end diastolic velocity (PEDV) values, guided correction is indispensable because a correct angle can change the values enormously (in the venous context, where velocimetric parameters are basically very low, angle correction is vitally important). But this correction is possible *only* if we can detect the vessel and its direction for about a centimetre at least. Moreover, bi-dimensional imaging is irreplaceable in the search for important reference points (protuberantia occipitalis interna, falx cerebri, tentorium etc.) which, in turn, help us to find suitable insonation planes in which to look for the venous vessels themselves (1,2).
Several structures can be insonated. They include:

- **the deep middle cerebral vein (DMCV)**
- **the vein of Rosenthal (RV)**
- **the vein of Galen (GV)**
- **the sphenoparietal sinus (SPaS)**
- **the superior sagittal sinus (SSS)** (distal part)
- **the straight sinus (SRS)**
- **the superior petrosal sinus (SPS)**
- **the inferior petrosal sinus (IPS)**
- **the transverse sinus (TS)**
- **the sigmoid sinus (SS)**
- **the basal venous plexus (BPV)**

It must be remembered that:

– vessel "detection rate" depends on the patient's age. Several trials in the literature have been based on the venous "detection rate" (3-5). Brain venous thrombotic pathology is increasingly present in young patients, in whom detection of venous vessels with TCCD is easier.

There are certain conditions indispensable to a correct approach to intracerebral venous study using ultrasound. These are:

– a *correct setting* of the equipment. It is unthinkable to try and detect veins correctly using the arterial flow setting. In this context, the concept of a "low-flow setting" acquires vital importance (decreasing the pulse repetition frequency-PRF-, small colour box, wall filter etc.).
– good anatomical knowledge of the course of the main venous branches and of the cerebral sinuses is another very important attribute that we must have before positioning the sample volume and moving the

probe in different planes. Thus, in the venous study the use of the traditional arterial insonation planes (mesencephalic, diencephalic, sovradiencephalic) must be supplemented by the use of many further venous planes which have to be found by means of small movements of the transducer, and also thanks to particular reference points: the protuberantia occipitalis interna (torcularis Herofilii), the free edge of the tentorium (SRS), the P2 segment of the posterior cerebral artery (RV), etc.

Another basic concept is that of

– venous colour coding: and thus, ultimately, the *direction* of the venous flow. We will see that detection of inverted flow direction in a venous vessel is often a very important indicator of an obstruction **(**thrombosis?) of the downstream vessel. If the DMCV flows next to the corresponding artery, the RV embraces the mesencephalon (peduncles and lamina) and then flows into GV. Since we are dealing with the venous system, many anatomical variants are possible. The DMCV and RV show flow directed away from the transducer, and the flow will appear below the zero flow line. The SPS can easily be insonated through the temporal bone window (flow directed towards the probe: positive-flow), while for the IPS the occipital approach is preferred (positive-flow) (6).

Study of the venous vessels is not an end in itself, but can give us important information in the event of brain venous thrombosis (a pathology increasingly frequent in young patients) and in the presence of arteriovenous malformations (AVM); this is true particularly of venous outflow studies of the AVM "nidus" (7-9). In determining the presence of brain thrombosis, given the wide anatomical individual variability of the venous formations, failure to detect a vessel (direct criterion) may be due to an obstruction or to hypoplasia of the vessel itself. The use of an ultrasound contrast agent, together with an "indirect criterion" (inverted flow due to vessel obstruction), can facilitate the work of a neurosonologist.

When carotid-cavernous fistulae are present (10,11), direct visualisation of the "nidus", together with the flowmetric pattern (low resistence index-RI) and the "sound" one, will be very useful for correctly detecting and differentiating them. Recourse to the ultrasonographic methodology can certainly help us to evaluate *"on line"* the possible

– vessel recanalisation, and the consequent
– improvement of the cerebral venous congestion.

The possibility of studying venous outflow variations in a repeatable and continuous way and thus, ultimately, of monitoring the cerebral venous decongestion, is surely the strength of venous cerebral study through TCCD. Similarly, the possibility of detecting not only tributary venous branches but in particular the presence of effluent *high-flow* ones, constitutes a possible future TCCD application.

We still have to prove, even though theoretically this is possible, the presence of a *therapeutic* action due to "sonification" of the venous thrombus (maybe helped by contrast agents and due to breaking of the bubbles near the thrombus itself by the high-power ultrasound beam), which can be likened to arterial sonothrombolysis!

Ultimately, cerebral venous study through TCCD introduces **a new philosophy** in ultrasound cerebral study. Whereas in recent years we have dwelled, in particular, on *how blood reaches the head*, now we are observing (12) *how blood "leaves" the head*. The cerebral decongestion mechanisms are enormously important, particularly if we consider that, according to the Monroe-Kelly theory, since the encephalon is contained in a closed box, any variation of the intracranial pressure impacts, in a very evident way, on the patient's outcome (13,14). Several recent papers have shown how a patient's prognosis is connected in an absolute way to the cerebral outflow mechanisms. While this is true mainly of primitively venous pathologies, it also applies to extensive arterial processes (one need only think of the problems consequent upon the mid-line shift in cases of extensive ischaemic infarcts with obstruction of the venous outflow at the level of the basal meso-diencephalic structures). In these cases we have, to date, dwelled upon the size and location of ischaemic arterial parenchymal areas as outcome predictors, but study of the venous circulation associated with these pathologies will be able to supply us with invaluable dynamic information.

TCCD-multigate

The TCCD with "multi-gate" technique is useful for demonstrating local venous local flow alterations (15). This is the novelty of the technique: using a sectorial probe, we can position two sample volumes in *different vessels* in this *sectorial plane*, and they may also be situated at *different depths*. At the *same time* we can also study homolateral and contralateral venous vessels. With this technique we can study the arterial flow and, at the same time, the *local venous outflow*, i.e., with a dedicated sectorial plane and using a wider sample volume we can study simultaneously the middle cerebral artery (MCA) and the DMCV(homolateral) and RV(homo- or controlateral).

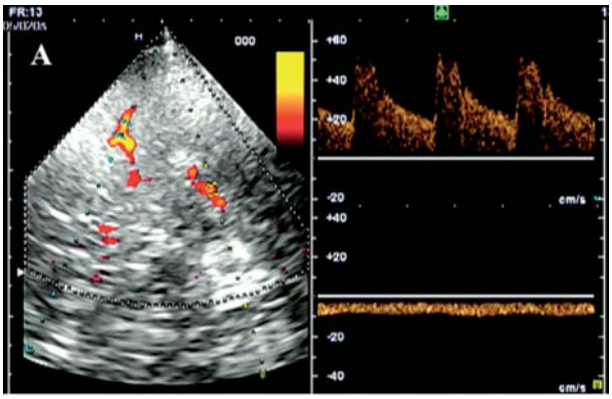

Fig. 1 - A-B: "Apnoea test" in MCA and BV.
A) Basal conditions. B) Increase of the flow in the MCA (with aliasing effect) and the correspondent venous pattern in BV (note the pulsated "arterial-like" flow). C) With a dedicated sectorial plane and using a wider sample volume we can study simultaneously the MCA, DMCV and BV.

Transient global amnesia

Transient global amnesia (TGA) is a clinical syndrome characterised by sudden-onset, transient memory disturbances without loss of consciousness or personal identity. These symptoms usually disappear within several hours. The main pathogenic hypotheses for TGA are: ischaemia, epilepsy, migraine and finally venous origin. Recent studies suggest involvement of memory circuits in the temporo-mesial region and in particular the hippocampo-mammillo-thalamus-cortical fibres ("Papez-circuit"). If, conceptually, cerebral ischaemia constitutes the main pathogenic hypothesis, Enzinger(16), in a recent article, studied TGA patients with MRI and remarked that their findings "do not support a cerebrovascular etiology of TGA, even in those individuals showing acute DWI lesions. Other pathophysiologic mechanisms need to be explored as indicated by more recent reports on hemodynamic disturbances in venous flow patterns in TGA" (17,18). In 1998, Lewis proposed a venous hypothesis (19): a causal bridge between cerebral venous congestion due to Valsalva-like activities and TGA. Sander (17) suggested that TGA may be due to venous congestion and consequent venous ischaemia at the level of basal cerebral venous system. He used TCCD to study changes in the internal jugular venous flow in patients with TGA during the Valsalva manoeuvre (VM).
Let us recall the arguments for a venous origin of TGA: 1) A VM can often cause TGA; 2) Situations increasing blood flow to the superior vena cava can often cause TGA (emotion, arm exercise, etc.); 3) Patients with a thrombosis of the Galenic venous system may present a transient amnestic syndrome.
Thus, the VM may increase venous pressure in the Galenic system, especially in patients with jugular vein valve incompetence.

Intracerebral arteriovenous conflict

Several studies in the literature have stressed how the presence of an "isolated headache" can often be caused by intracerebral sinus thrombosis (20,21). Thus, isolated headache can be a major problem in routine neurological examinations. In our department, we found that patients with this clinical symptom can present an ultrasonographic pattern of recent *arteriovenous conflict between the RV and the posterior cerebral artery(PCA).* The P1 or P2 segment of the PCA can often come into conflict with the pre- or post- peduncular part of the RV. Using the new generation software we can see (with a good ultrasonographic bone

window!) the arterial and venous vessel walls. In this situation, the venous local flow will be very slow and it could be related to these pathologies and ultimately predispose patients to the subsequent appearance of local thrombosis.
To our knowledge this is the first report of arteriovenous conflicts that could represent a *pathogenetic bridge* between isolated headache and cerebral venous thrombosis.

In conclusion, TCCD (also with the "multi-gate" technique) is very useful for studying *dynamic and functional* intracerebral venous flowmetric patterns. The venous study must be an essential part of every acute TCCD examination, so that useful information, which is within our reach using modern software, is not missed.

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