The well known clinical picture of "coma depassé" which follows lethal post-anoxic cerebral oedema (e.g. in cases of severe head trauma or prolonged cardiac arrest with isoelectric EEG) is sometimes accompanied by the angiographic phenomenon of non-filling of cerebral vessels. It is accepted—but has not been proven—that this non-filling phenomenon represents an arrest of cerebral circulation, supposed to be due to an increase in intracranial pressure to levels above systolic or diastolic blood pressure. However, "minimal residual carotid blood flow when intracranial pressure had been elevated far above the systolic pressure" has been observed. Further, the fact that apparently no angiographic contrast has penetrated into the cranium by the carotid arteries is no proof that there is no cerebral blood flow (CBF) because the amount of contrast medium able to enter the cerebral vessels may be too little to be visualized. Further attention will be directed to this point in the discussion.

The isoelectricity of the EEG often observed in such cases, and which seems to precede the non-filling phenomenon, has been invoked by some as a proof of cerebral (and total) death. Since there are several clinical conditions compatible with life which may also be associated with an isoelectric EEG, even for long periods of time, recovery having been recorded even after four and five days of isoelectricity, and since, on the other hand, in patients with isoelectric EEG and lethal outcome evoked potentials or central reflexes may still be obtained, recovery cannot be considered a reliable criterion of so-called cerebral death—even if the basal ganglia are also silent.

Modern resuscitative and intensive care techniques have allowed such irreversibly comatose patients to be maintained in a state of vegetative life for longer periods of time. However, the outcome has not been altered...
by such technical improvements. On the contrary, these improvements have augmented the tremendous and unrewarding burden this particular type of patient represents to the staff, to his family, and to society. This problem will persist as long as there is no reliable criterion to answer the question of when it is correct to consider a patient as dead and to discontinue the aids which are maintaining "Life". Since medical teams performing organ transplantation in Homo have been increasingly faced with this delicate subject, it has received more and more attention and has been discussed with growing intensity.

If it is true that nowadays death means cerebral death, and if it is also true that cerebral life is impossible in the absence of cerebral blood flow, the demonstration of an arrest of blood flow within the brain would, then, be a reliable criterion for the verification of death. In such cases, obviously, the usual clinical manifestations of cerebral death will be present.

We have recently been able to study the CBF of four such patients. Since no similar report has been found in literature, it seems worthwhile to present our findings as illustrated by one typical case.

**Case:** A. Z., a 4½ years-old white boy was injured in a car accident at 2:30 p.m. on July 12, 1968. The boy lost consciousness immediately and was brought to a nearby hospital. Simple roentgenograms of the skull showed no abnormalities. There was a transverse fracture of the proximal third of the shaft of the left femur. The child remained comatose and was transferred to our department the next morning, some 20 hours after the accident. The pupils were moderately but symmetrically dilated and reacted sluggishly to light; there was a generalized hyporeflexia. No plantar response could be evoked. Painful stimuli elicited a barely perceptible reaction of all limbs. Blood pressure was 120/80 mm Hg, pulse 95 per minute, temperature 37.7°C and respiratory rate 25 per minute. The echoencephalogram showed a midline shift of 2 mm (within normal limits) from right to left. Laboratory findings were normal.

In the absence of focal signs it was decided to keep the child under close observation in the intensive care unit. Chloramphenicol and Comital were given prophylactically.

The clinical picture remained entirely unchanged until 9:00 a.m. on July 15 (about 67 hours after the accident), when the child had a sudden cardio-respiratory arrest. Reanimation was immediately begun and the child was intubated whilst external cardiac massage was being performed. Spontaneous heart activity returned after one minute, but the child had to remain connected to a Bird respirator. At this point the pupils were maximally dilated and unresponsive. Blood pressure could not be measured by auscultation. Although a control echoencephalogram still showed no pathological midline shifting, it was decided to perform an angiographic study.

*Bilateral percutaneous serial carotid angiography* (puncture of the common carotid arteries) at 10:00 a.m. revealed the non-filling phenomenon on both sides (Fig. 1). The major part of the injected contrast medium (7 ml) circulated through the external carotid vessels, while a fraction of it still remained within the extraeranial internal carotid at the end of the series (about 10 seconds after injection). The "waterfall phenomenon" of the contrast medium could be observed on both sides, being more distinct on the right.