Carotid endarterectomy using routine shunting and the risk of cerebral hyperperfusion syndrome

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BACKGROUND: Cerebral hyperperfusion syndrome (CHS), defined as a cerebral hyperperfusion combined with neurological findings, occurs in approximately 3–5% of patients undergoing a carotid endartrectomy (CEA) procedure and is potentially life threatening [1].Increased post internal carotid (ICA) clamping and end of procedure regional cerebrovascular oxygen saturation (rSO2) more than 5% and 10% of the pre-clamping values correlates with a higher risk of CHS with sensivity and specificity of 100% and 86.4%, respectively [2, 3, 4].

OBJECTIVES: We prospectively studied the incidence of either cerebral oximetric or clinical evidence of cerebral hyperperfusion in patients undergoing CEA using routine shunting.

METHODS: Between July 2013 and January 2014, 34 patients (22 men, 12 women) undergoing 40 CEA procedures with patch closure using routine shunting were included consequently (Table 1&2). Age ranged from 48 to 86 years. Standard anesthetic and monitoring techniques were used in all patients.Regional cerebro-vascular oxygen saturation (rSO2) was recorded continually (of an interval of 5 seconds) before preoxygenation and continued until recovery from anesthesia, using cerebral oximeter (Somanetic INVOS-5100 cerebral oximeter, COVIDIEN, USA). Area under the curve (AUC) was defined as rSO2 20% or more below baseline. An arterial line was placed for blood pressure measurement. Blood pressure was kept stable within a range of ±20% of the pre-operative level. Somatosensory evoked potential monitoring (SEP) was performed from induction of to recovery from anesthesia (ISIS IOM Neuromonitoring, Inomed, GERMANY).

Neurological classification	
Asymptomatic stenosis	**41%**
Transient ischemic attack	**46%**
Amaurosis fuga	**5%**
Stroke	**8%**
Carotid evaluation (ipsilateral)	
Symptomatic patients	
Stenosis 70-99%	50%
Stenosis 50-69%	8%
Asymptomatic patients	
Stenosis 60-99%	42%
Carotid evaluation (contralateral)	
Stenosis 60-99%	18%
Stenosis <60%	50%
Occlusion	2%
Vertebral arteries	
No occlusion	82%
Occlusion (one or both)	18%

Table1. Preoperative neurologic status and diagnostic findings of preoperative carotid evaluation (n=36).

Hypertension	**74%**
Coronary artery disease	41%
Dyslipidemia	38%
Cigarette smoking	32%
PVD *	18%
Chronic kidney disease	18%
COPD	12%
Previous stroke	12%

Table 2. Risk factors for carotid atherosclerosis (n=36). * PVD = Peripheral vascular disease.

The surgical team was informed of any sudden decrease of the ipsilateral rSO2 measurement or ischemic SEP change after insertion of the shunt to check malfunction. All patients were extubated in the operation theater. At this point patients were tested for the development of any neurological deficit. Patients were transferred to the ICU and were followed until discharge from the hospital and further until one month after surgery for any symptom of CHS.

RESULTS: Reconstruction of ICA was successfully completed in all 40 surgeries. During ICA cross-clamp, there were no clinical significant changes in heart rate, mean arterial blood pressure, arterial oxygen saturation (pulse oximetry), end tidal CO2 (capnometry).No deaths occurred during the observation period. One patient presented post-operatively with transient hypoglossal nerve paresis. No patient developed permanent or transient new neurological deficit on the side of CEA. No patient developed CHS during the follow up period. The mean time between ICA cross-clamping and shunt insertion was 140±51 seconds, (range of 70 to 270 seconds). The mean shunting time was 36±9 minutes, (range of 22 to 60 minutes). In 11 patients (27%), post ICA clamping rSO2 was more than 105% of preclamping value and in 10 patients (25%), end of procedure rSO2 was more than 110% of preclamping value. 18 patients (45%) had cerebral oximetric evidence of cerebral hyperperfusion but no patient developed CHS during follow up.

CONCLUSION: In our series, 45% of the cases had cerebral oximetric evidence of cerebral hyperperfusion, but no patient developed CHS during follow up. This difference can be explained by the limited time of ntra-operative cerebral ischemia, due to routine shunting.

References

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