Monitoring of cerebral oxygenation and cognitive function in elective carotid endarterectomy



Kuzkov V. V., Obraztsov M. Y., Lenkin P. I., Klyagin A. A., Sokolova M. M., Kirov M. Y.

Department of Anesthesiology and Intensive Care Medicine, Northern State Medical University, Arkhangelsk, Russian Federation

Vsevolod V. Kuzkov, M.D., Ph.D E-mail: v_kuzkov@mail.ru

Background & Goal

Carotid endarterectomy (CA) can be associated with intraoperative cerebral ischemia and injury resulting in postoperative derangement of cognition. In parallel, an acute restoration of blood flow after the revascularisation may be followed by the hyperperfusion state. The role of surgical arterial bypass to prevent cerebral ischemia during the clamping remains disputable.

The goal of this study was to investigate the effect of transient arterial bypass of repaired carotid artery on cerebral oxygenation and cognitive function after elective CA.

Methods

Forty-five adult patients were subjected to elective CA and enrolled into the ongoing single-center prospective study. The patients were randomized into two groups: with temporary carotid artery bypass (the Bypass group, n = 24) and without the bypass (the Control group, n = 21). Interventions were performed in the settings of total intravenous anesthesia (propofol, fentanyl, atracurium). All patents were monitored using cerebral tissue oxymetry (SctO₂, Fore-Sight Cerebral Oximeter, Casmed, USA) over the intact side (SctO_{2INT}) and potentially ischemic operated side (SctO_{2OPER}) separately. Invasive mean arterial blood pressure (MAP), SpO₂, EtCO₂ and arterial blood gases were registered. All variables were measured at multiple stages intraoperatively as well as at 3 and 6 hrs after the surgery.

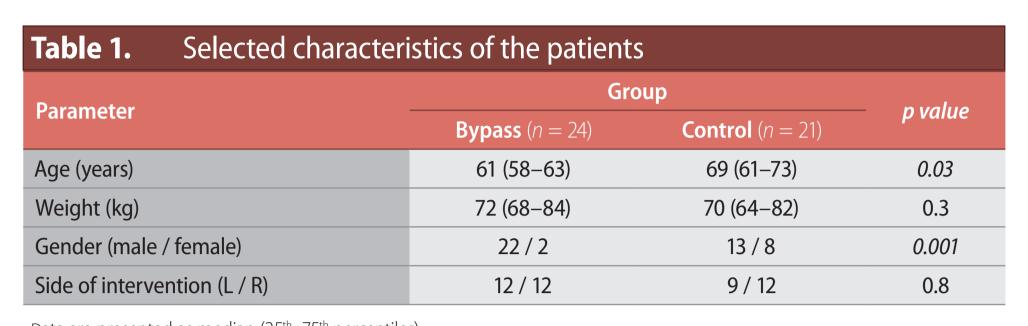
Intraoperatively, if SctO₂ values reduced below 55%, the temporal bypass was set in the control group and MAP and FiO₂ increased in the Bypass group.

All patients were tested for cognitive function using three versions of Montreal Cognitive Assessment Score (MoCA) by 12 hrs before the intervention and postoperatively at 6–8 and 24 hrs.

The data are presented as median (25th–75th percentiles) and analyzed for intergroup differences using Mann–Whitney *U*-test. Intragroup changes of parameters were assessed using Wilcoxon's test. The correlations were presented using Spearman's *rho* correlation coefficient. *P* value below 0.05 was assumed as statistically significant.

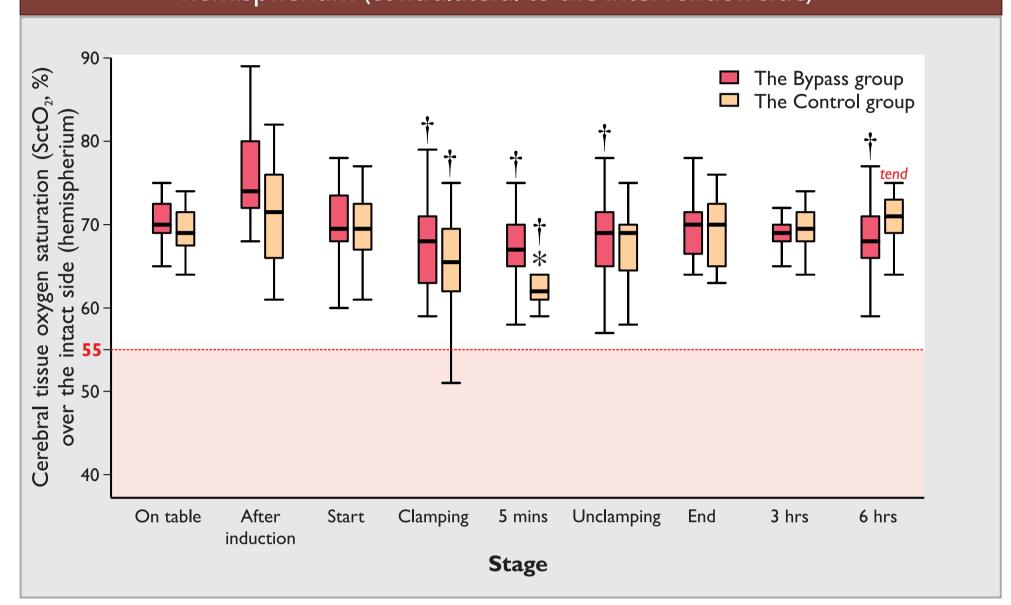
Results & Discussion

We found no intergroup differences in weight, and side of intervention (Table 1), while the patients in the Control group were moderately older (p = 0.03, Table 1).



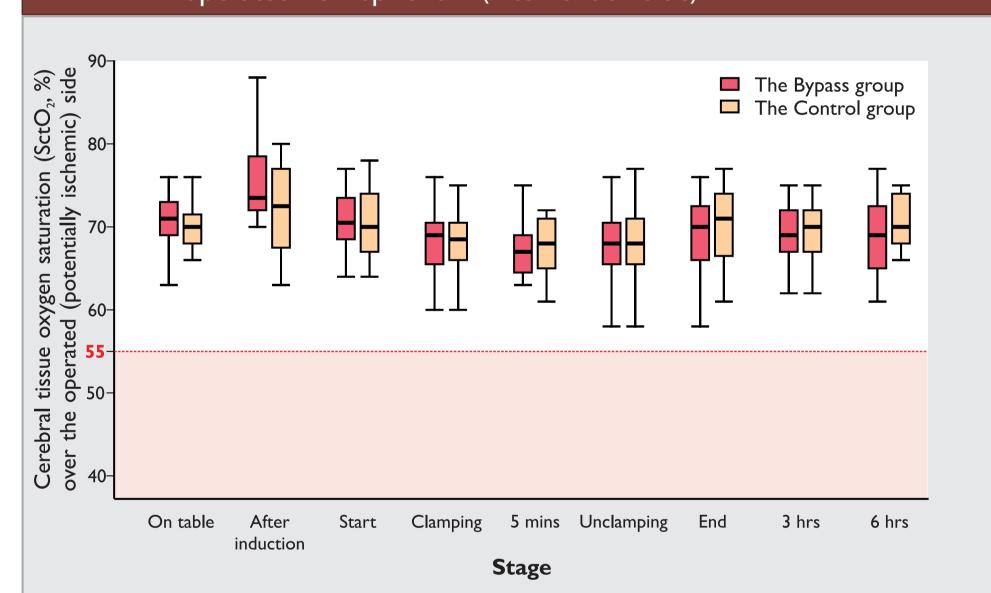
Data are presented as median (25^{th} – 75^{th} percentiles). * — p < 0.05 in Mann–Whitney U-test between the groups.

Figure 1. Changes in cerebral tissue oxygen saturation (SctO₂) over the intact hemispherium (contralateral to the intervention side)



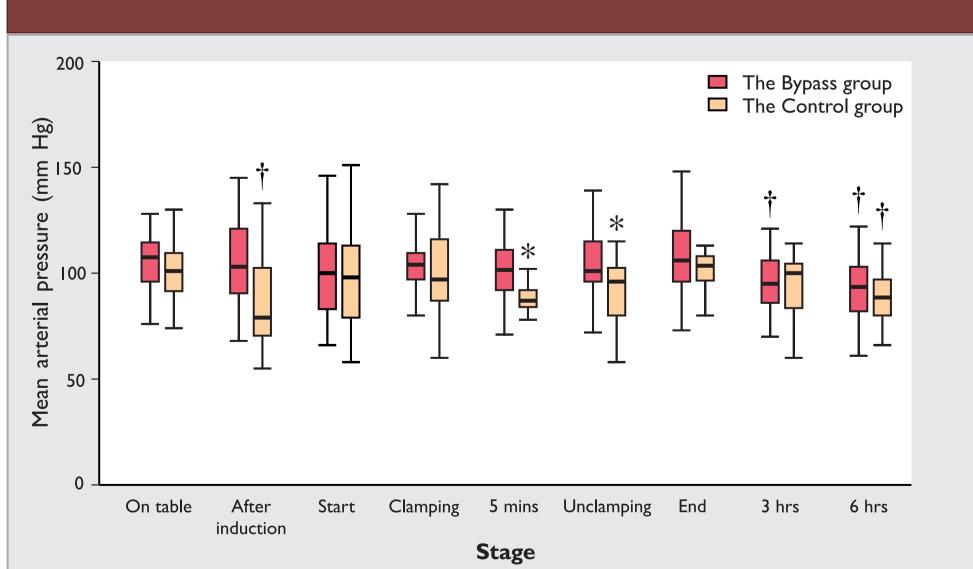
Data are presented as a box plot (median; 25^{th} – 75^{th} percentiles). * — p < 0.05 between groups (Mann–Whitney U-test); † — p < 0.05 within group compared with "Start" stage.

Figure 2. Changes in cerebral tissue oxygen saturation (SctO₂) over the operated hemispherium (intervention side)

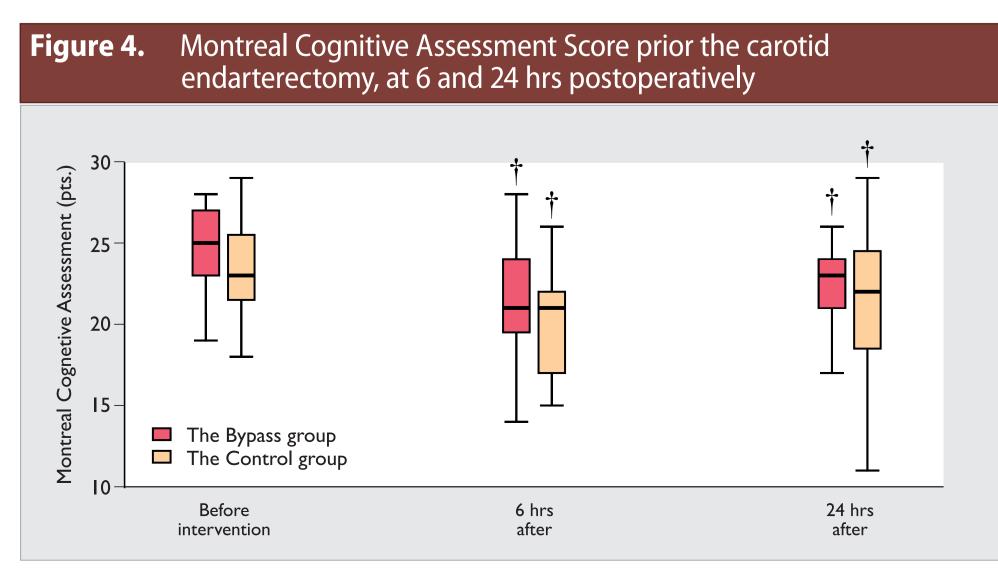


Data are presented as a box plot (median; 25th-75th percentiles).

Figure 3. Changes in mean arterial pressure in the perioperative period



Data are presented as a box plot (median; 25^{th} – 75^{th} percentiles). * — p < 0.05 between groups (Mann–Whitney U-test); † — p < 0.05 within group compared with "on table" stage.



Data are presented as a box plot (median; 25^{th} – 75^{th} percentiles). t — p < 0.05 within group compared with value before intervention

In both groups, $SctO_{2INT}$ decreased after the clamping, 5 minutes thereafter, and at 6 hrs after intervention compared with the start of surgery (p < 0.05). In the Bypass group, $SctO_{2INT}$ was significantly higher at 5 minutes after carotid unclamping: 67 (65–70)% vs. 62 (61–66)% but tended to be lower at 6 hrs after surgery (p < 0.2) as compared to the control group (Figure 1). These changes may result from a relative hyperperfusion of operated side in the Control group, since these patients were not preconditioned with intraoperative bypass-related hyperperfusion.

Importantly, there were no intergroup differences in $SctO_{2OPER}$ (Figure 2). We found an association between respective $SctO_{2INT}$ and $SctO_{2OPER}$ values (rho = 0.32-0.79; p < 0.05), except for 3 hrs postoperatively. The lack of correlation between $SctO_{2INT}$ and $SctO_{2OPER}$ values at this time point might be explained by early postoperative hyperperfusion state.

In addition, $SctO_2$ correlated with respective MAP values after induction, as well as at 30 and 45 minutes after clamping (rho = 0.34-0.38; p < 0.05).

In the control group, MAP was significantly lower at 5 minutes after clamping and after unclamping (p < 0.05, Figure 3). This phenomenon may be related to the acute emergence of the bypass-related arterial hyperperfusion in the Bypass group resulting in compensatory vasoconstriction. In the Control group, MAP significantly decreased after the induction and at 6 hrs. In the Bypass group, MAP was lower at 3 and 6 hrs following the CA compared with the initial values (Figure 3).

The assessment of cognitive function did not reveal any differences between the groups (Figure 4). The MoCA scores reduced significantly in both groups postoperatively by 6-8 hrs after intervention and by 24 hrs. We found strong association between MoCA values before and after the interventions (rho = 0.65-0.73; p < 0.0001).

Conclusions

When SctO₂ was maintained above 55%, the arterial bypass during elective CA did not improve cognitive function despite transiently higher SctO₂ over the intact hemisphere.

The decrease of SctO₂ over the intact side might result from the compensatory stealing by the ischemic (operated) side. The values of SctO₂ demonstrate associations between intact and operative sides and correlation with mean arterial pressure.