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PROVA ORALE N. 1

Ricerca bibliografica per la ricerca clinica.

PROVA NON ESTRATTA

FRANCESCO DE DONATO

F. DeDato

1/7/2022

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PROVA ORALE N. 2

Iter attuativo di uno studio scientifico: dall'idea alla pubblicazione.

PROVA ESTRATTA

FRANCESCO DEDONATO

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PROVA ORALE N. 3

Caratteristiche qualitative di un lavoro scientifico.

PROVA NON ESTRATTA

FRANCESCO DE DONATO

F. DeDonato 1/7/2022

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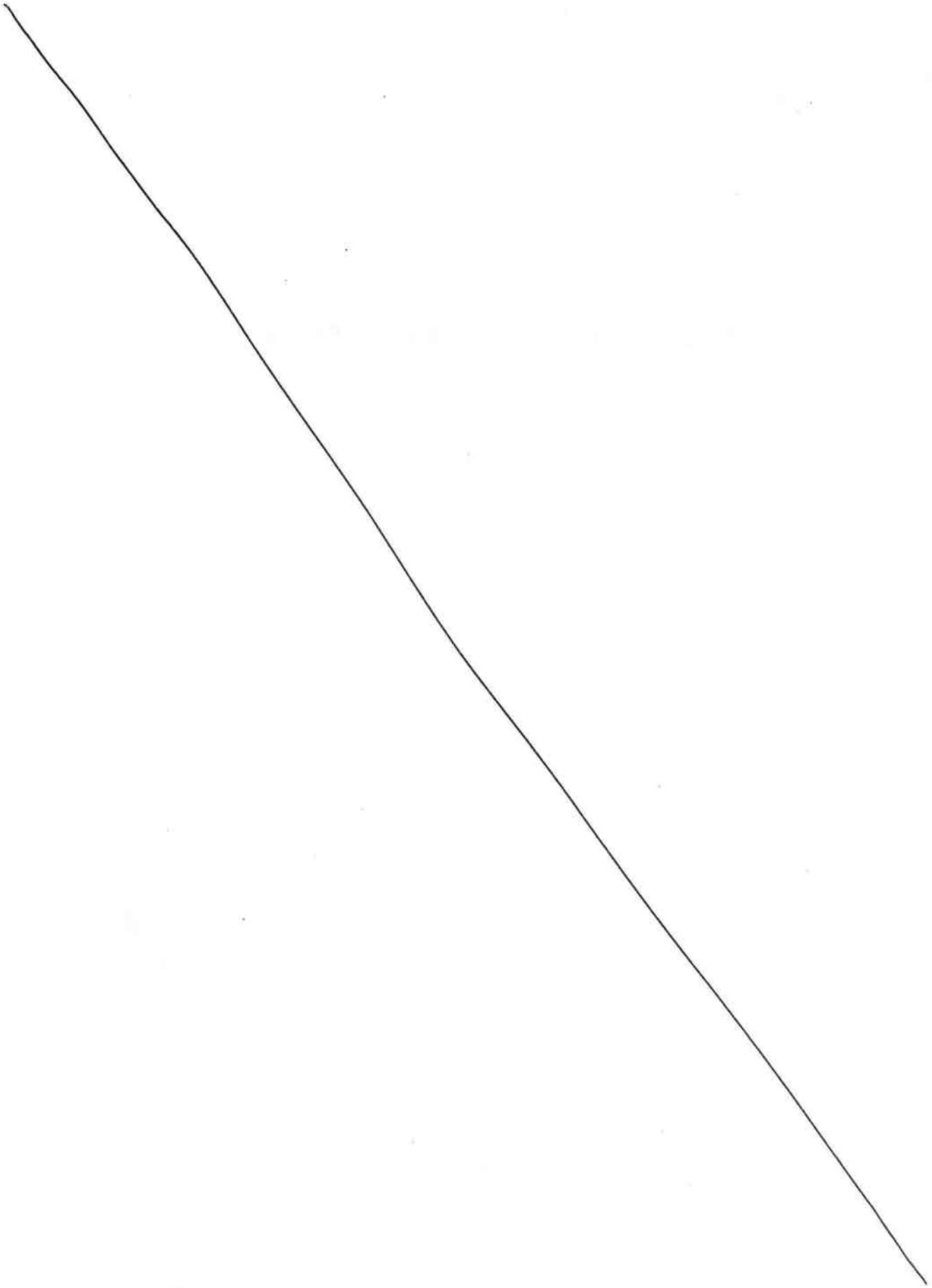
QUESITO DI INFORMATICA

Programmi per la gestione di Database.



Postoperative change in baseline parameters (heart rate, mean arterial pressure, hematocrit, and $PaCO_2$) can potentially affect Vmca, THRR, and CO_2 reactivity. Although the difference in these variables between the impaired and intact THRR groups did not reach statistical significance (Tables 1, 2), it did approach significance for heart rate and mean arterial pressure ($P = 0.06$), and hematocrit ($P = 0.07$). However, the differences were not clinically significant either, thereby making it unlikely that these differences would have affected the results of postoperative change in Vmca, THRR, and CO_2 reactivity.

Effect of Tumors on CO_2 Reactivity

Decreased CO_2 reactivity is seen in a number of conditions,[26–28] and suggests poor outcome in comatose patients.[29] Although Palvolgyi[8] reported the loss of CO_2 reactivity in patients with tumors, Paulson et al[9] observed only a "dissociated vasoparalysis" that is, loss of autoregulation with preserved CO_2 reactivity. Dissociated vasoparalysis has been described as a sign of tissue acidosis and has been thought of as a stage in gradual transition from normal vasomotor function to complete vasomotor paralysis.[9] Our data indicate preservation of CO_2 reactivity in all patients with supratentorial tumors with dissociated vasoparalysis occurring in 7 (20%) patients. We did not assess the cerebrovascular response to increased CO_2 tensions because of the risk of increasing intracranial pressure.

Limitations

Major limitations of this study include the facts that autoregulation was studied only once after tumor decompression and that we do not have long-term outcome data. Hence, we cannot comment on whether autoregulation improved 24 hours after surgery in cases where it was impaired preoperatively, nor can we comment on the long-term implications of impaired autoregulation in these patients. Moreover, our sample size is relatively small. Despite these limitations, this study brings up the important fact that cerebral autoregulation might be compromised in patients presenting for surgery for supratentorial tumor, potentially subjecting them to increased risk of secondary results in perioperative period.

Summary

Preoperative cerebral autoregulation was impaired in a significant number of patients with large supratentorial tumor size and midline shift more than 5 mm and was associated with postoperative impaired cerebral autoregulation during the first 24 hours after surgery. These findings define the risk factors for impairment of cerebral autoregulation in neurosurgical patients undergoing tumor decompression and may help to guide perioperative optimal blood pressure control and in selection of anesthetic agents in these patients.

REFERENCES

1. Czosynka M, Smeilewski P, Kirkpatrick P, et al. Monitoring of cerebral autoregulation in head-injured patient. *Stroke*. 1996;27:1829–1834.
2. Smeilewski P, Czosynka M, Kirkpatrick P, et al. Evaluation of the transient hyperemic response test in head-injured patients. *J Neurosurg*. 1997;86:773–778.
3. Dernbach PD, Little JR, Jones SC, et al. Altered cerebral autoregulation and carbon dioxide reactivity after aneurysmal SAH. *Neurosurgery*. 1988;22:822–826.
4. Lovick AJ, Pickard JD, Goddard BA. Prediction of late ischaemic complications after cerebral aneurysmal surgery: use of mobile microcomputer system for measurement of pre, intra and post operative cerebral blood flow. *Acta Neurochir (Wein)*. 1982;63:37–49.
5. Pickard JD, Mathieson JD, Patterson J, et al. Prediction of late ischaemic complications after cerebral aneurysm surgery by intraoperative measurement of cerebral blood flow. *J Neurosurg*. 1980;53:305–308.
6. Muizelaar JP, Lutz HA III, Becker DP. Effect of mannitol on ICP and CBF and correlation with pressure autoregulation in severely head-injured patients. *J Neurosurg*. 1984;61:700–706.
7. Schmieder K, Schregel W, Harders A, et al. Dynamic cerebral autoregulation in patients undergoing surgery for intracranial tumors. *Eur J Ultrasound*. 2000;12:1–7.
8. Palvolgyi R. Regional cerebral blood flow in patients with intracranial tumors. *J Neurosurg*. 1969;31:149–163.
9. Paulson OB, Olesen J, Christensen MS. Restoration of autoregulation of cerebral blood flow by hypocapnea. *Neurology*. 1972;22:286–293.
10. Girling KJ, Cavill G, Mahajan RP. The effects of nitrous oxide and oxygen on transient hyperemic response in human volunteers. *Anesth Analg*. 1999;89:175–180.
11. Bedforth NM, Girling KJ, Harrison JM, et al. The effects of sevoflurane and nitrous oxide on middle cerebral artery blood flow velocity and transient hyperemic response. *Anesth Analg*. 1999;89:170–174.
12. Tibble RK, Girling KJ, Mahajan RP. A comparison of the transient hyperemic response test and the static autoregulation test to assess graded impairment in cerebral autoregulation during propofol, desflurane, and nitrous oxide anesthesia. *Anesth Analg*. 2001;93:171–176.
13. Smeilewski P, Czosynka M, Kirkpatrick P, et al. Assessment of cerebral autoregulation using carotid artery compression. *Stroke*. 1996;27:2197–2203.
14. Aaslid R, Lindegaard KF, Sorteberg W, et al. Cerebral autoregulation dynamics in humans. *Stroke*. 1989;20:45–52.
15. Moppett IK, Mahajan RP. Transcranial Doppler ultrasonography in anaesthesia and intensive care. *Br J Anaesth*. 2004;93:710–724.
16. Mahajan RP, Cavill G, Simpson EJ. Reliability of the transient hyperemic response test in detecting changes in cerebral autoregulation induced by the graded variations in end-tidal carbon dioxide. *Anesth Analg*. 1998;87:843–849.
17. Gupta MM, Bithal PK, Dash HH, et al. Effects of stellate ganglion block on cerebral haemodynamics as assessed by transcranial Doppler ultrasonography. *Br J Anaesth*. 2005;95:669–673.
18. Mahajan RP, Cavill G, Simpson EJ, et al. Transient hyperaemic response: a quantitative assessment. In: Klingelhofer J, Bartels E, Ringelstein EB, eds. *New Trends in Cerebral Haemodynamics and Neurosonology*. Amsterdam: Elsevier Science BV; 1997:618–623.
19. De Salles AA, Manchola I. CO_2 reactivity in arteriovenous malformations of the brain: a transcranial Doppler ultrasound study. *J Neurosurg*. 1994;80:624–630.
20. Giller CA. A bedside test for cerebral autoregulation using transcranial Doppler ultrasound. *Acta Neurochir (Wien)*. 1991;108:7–14.
21. Smeilewski P, Czosynka M, Iyer V, et al. Computorised transient hyperaemic response test: a method for the assessment of cerebral autoregulation. *Ultrasound Med Biol*. 1995;21:599–611.
22. Nakayama Y, Tanaka A, Kumate S, et al. Cerebral blood flow in normal brain tissue of patients with intracranial tumors. *Neuorol Med Chir (Tokyo)*. 1996;36:709–714.
23. Smith DR, Jacobson J, Kobrine AI, et al. Regional cerebral blood flow with intracranial mass lesions. Part II: Autoregulation in localized mass lesions. *Surg Neurol*. 1977;7:238–240.