HHS Public Access

Author manuscript

Anesth Analg. Author manuscript; available in PMC 2017 July 11.

Published in final edited form as:

Anesth Analg. 2010 July ; 111(1): 191–195. doi:10.1213/ANE.0b013e3181e054ba.

Noninvasive Autoregulation Monitoring with and without Intracranial Pressure in the Naïve Piglet Brain

Ken M. Brady, MD[*], **Jennifer O. Mytar, BS**[*], **Kathleen K. Kibler, BS**[*], **Charles W. Hogue Jr., MD**[*], **Jennifer K. Lee, MD**[*], **Marek Czosnyka, PhD**[†], **Peter Smielewski, PhD**[†], and **R. Blaine Easley, MD**[*]

[*]Department of Anesthesiology and Critical Care Medicine, Johns Hopkins Hospital, Baltimore, Maryland [†]Department of Academic Neurosurgery, Addenbrooke's Hospital, Cambridge, United Kingdom

Abstract

BACKGROUND—Cerebrovascular autoregulation monitoring is often desirable for critically ill patients in whom intracranial pressure (ICP) is not measured directly. Without ICP, arterial blood pressure (ABP) is a substitute for cerebral perfusion pressure (CPP) to gauge the constraint of cerebral blood flow across pressure changes. We compared the use of ABP versus CPP to measure autoregulation in a piglet model of arterial hypotension.

METHODS—Our database of neonatal piglet (5–7 days old) experiments was queried for animals with naïve ICP that were made lethally hypotensive to determine the lower limit of autoregulation (LLA). Twenty-five piglets were identified, each with continuous recordings of ICP, regional cerebral oximetry (rSO_2), and cortical red cell flux (laser Doppler). Autoregulation was assessed with the cerebral oximetry index (COx) in 2 ways: linear correlation between ABP and rSO_2 (COx_{ABP}) and between CPP and rSO_2 (COx_{CPP}). The lower limits of autoregulation were determined from plots of red cell flux versus ABP. Averaged values of COx_{ABP} and COx_{CPP} from 5 mm Hg ABP bins were used to show receiver operating characteristics for the 2 methods.

RESULTS—COx_{ABP} and COx_{CPP} yielded identical receiver operating characteristic curve areas of 0.91 (95% confidence interval [CI], 0.88–0.95) for determining the LLA. However, the thresholds for the 2 methods differed: a threshold COx_{ABP} of 0.5 was 89% sensitive (95% CI, 81%–94%) and 81% specific (95% CI, 73%–88%) for detecting ABP below the LLA. A threshold

Address correspondence and reprint requests to Ken M. Brady, MD, Department of Anesthesiology, Division of Pediatric Anesthesiology and Critical Care Medicine, The Johns Hopkins University School of Medicine, 600 North Wolfe St., Blalock 943, Baltimore, MD 21287. Kbrady5@jhmi.edu.

AUTHOR CONTRIBUTIONS

KMB and RBE helped with study design and conduct, data analysis, and manuscript preparation; JOM, KKK, and JKL helped with data analysis, study conduct, and manuscript preparation; and CWH, MC, and PS helped with data analysis and manuscript preparation.

DISCLOSURE

Under a licensing agreement with Somanetics, Dr. Brady is entitled to a share of fees and royalty received by The Johns Hopkins University on the monitoring technology described in this article. The terms of this arrangement are being managed by The Johns Hopkins University in accordance with its conflict of interest policies. ICM+ software is licensed by the University of Cambridge, Cambridge Enterprise Ltd. Drs. Smielewski and Czosnyka have a financial interest in a part of the licensing fee.

COx_{CPP} of 0.42 gave the same 89% sensitivity (95% CI, 81%–94%) with 77% specificity (95% CI, 69%–84%).

CONCLUSIONS—The use of ABP instead of CPP for autoregulation monitoring in the naïve brain with COx results in a higher threshold value to discriminate ABP above from ABP below the LLA. However, accuracy was similar with the 2 methods. These findings support and refine the use of near-infrared spectroscopy to monitor autoregulation in patients without ICP monitors.

Patients at risk for neurologic injury from anesthesia, cardiopulmonary bypass, sepsis, and even intraoperative head-up positioning require scrupulous monitoring and support of their arterial blood pressure (ABP). Classical teaching assigns a mean ABP of 50 mm Hg to the adult lower limit of autoregulation (LLA) based on Lassen's publication in 1959.[1] The key element of Lassen's curve, the inflection at 50 mm Hg, originated from studies of pregnant women.[2] Despite this limitation, Lassen's curve has long provided the basic tenets of hemodynamic management as to the lowest clinically tolerable ABP.

More recently, a review of autoregulation studies raised valid concerns about the applicability of an LLA of 50 mm Hg by documenting a wide interstudy variability in the ABP coordinate of the LLA.[3] It is not reasonable to apply a single hemodynamic management strategy for patients with diverse conditions and physiology. Thus, there has been a paradigm shift toward individualizing care by focusing on decrements in ABP as a percentage of a patient's baseline ABP.[4,5]

Hemodynamic care for patients with traumatic brain injury has been further individualized by continuous monitoring of dynamic cerebrovascular autoregulation.[6,7] Auto-regulation monitoring has had more limited exposure outside of the clinical neuroscience arena, but noninvasive approximations of changes in cerebral blood flow (CBF) with transcranial Doppler and near-infrared spectroscopy can be used for this purpose.[8,9] Such data may allow clinicians to target clinical ABP to a level above the individual's LLA to ensure CBF commensurate with cerebral metabolic demand. Studies of continuous autoregulation monitoring in patients with sepsis have been done using both transcranial Doppler and near-infrared spectroscopic techniques.[10,11] Patients with abnormal autoregulation in the setting of sepsis had a higher incidence of delirium. In a separate study of autoregulation monitoring with transcranial Doppler, patients rewarming from cardiopulmonary bypass had a higher incidence of stroke when they demonstrated abnormal autoregulation compared with patients showing normal autoregulation.[12] Although promising, these studies share an important and unproven assumption: that autoregulation can be monitored without intracranial pressure (ICP) monitoring.

Dynamic monitoring of autoregulation repetitively quantifies the constraint of CBF during changes in cerebral perfusion pressure (CPP). For patients with traumatic brain injury, dynamic monitoring of autoregulation using CPP yielded results that were associated with outcome, but when ABP was used for this monitor, there was no correlation with outcome.[13] This difference was predictable from the phenomenon of "false autoregulation" in some patients with brain injury. In these patients, exaggerated ICP changes are induced by ABP changes, so ABP is not suitable to gauge acute perfusion pressure changes in the injured brain.[14,15] In the absence of intracranial injury, the impact of substituting ABP for CPP is

less clear. If ICP slow-wave amplitudes are negligible in comparison to the magnitude of ABP slow waves, then it is reasonable to hypothesize that ABP alone is adequate to measure cerebrovascular autoregulation. Data to support this assumption are necessary for the deployment of autoregulation monitoring outside of the neurosurgical arena.

We compared the accuracy of autoregulation monitoring using ABP or CPP using the cerebral oximetry index (COx) in a piglet model of arterial hypotension. The COx is a moving, linear correlation coefficient between spontaneous slow waves of CPP and regional cerebral oximetry (rSO_2) measured by near-infrared spectroscopy.[9,16] Positive values of COx indicate rSO_2 values that are passive to changes in ABP, a state of impaired cerebrovascular autoregulation. The COx is well suited for clinical monitoring of autoregulation because rSO_2 can be obtained noninvasively and with minimal effort. We hypothesized that ABP can be used in place of CPP for calculation of the COx without degrading accuracy. We compared the COx obtained with ABP (COx_{ABP}) against the COx obtained with CPP (COx_{CPP}) for agreement, ability to detect the LLA, and threshold optimization.

METHODS

All procedures were approved by The Johns Hopkins University Animal Care and Use Committee and conformed to the standards of animal experimentation of the National Institutes of Health.

Animal Selection

Animals included in the present study were part of prior studies evaluating CBF autoregulation using a standardized protocol of decreasing ABP.[9,16–18] We searched our database of waveform recordings for protocols with piglets aged 5 to 7 days and naïve ICP (excluding protocols with induced intracranial injury or hydrocephalus), and with intact recordings of ICP, ABP, red cell flux in the parietal cortex using laser Doppler, and rSO_2. Data from 25 experiments using this protocol were identified and all were included.

Surgical Preparation

The protocol briefly described below to determine the LLA has been published.[9] All piglets were anesthetized by inhalation of isoflurane and nitrous oxide, and supplemented with infusions of vecuronium (5-mg bolus and 2 mg/h infusion) and fentanyl (25-μg bolus and 25 μg/h infusion) so that the inhaled anesthetic could be reduced to 0.8% isoflurane and 50% nitrous oxide in oxygen. Anesthesia was maintained until the completion of the experiment, at which time the animals were euthanized with IV potassium. All animals had femoral venous and bilateral femoral arterial cannulations. A 5F esophageal balloon catheter (CooperSurgical, Trumbull, CT) was advanced through the remaining femoral vein to the inferior vena cava for decreasing of the ABP. Craniotomies were performed for placement of a left-sided external ventricular drain, a left parietal laser-Doppler probe (Moor Instruments, Devon, UK) that was advanced through a dural incision to contact the parietal cortex, and a left occipital brain-temperature monitor. The right scalp was left intact for placement of a SomaSensor for cerebral oximetry (Somanetics, Troy, MI). Additional left-sided

craniotomies were made in 9 animals that were part of a study of rheoencephalography. These 1-mm craniotomies were used for placement of a pair of silver wires inserted 4 mm into the parietal cortex, from which impedance measurements were taken (data not yet published or included in this study). After monitor placement, all craniotomies were sealed with dental cement, the skin was reapplied to the skull with suture, and the animals were allowed to recover for 1 hour. Piglets were kept on a warming blanket to keep brain and rectal temperature between 38.5°C and 39.5°C. Arterial blood gases were kept in the normal physiologic range: pH 7.35 to 7.45, $PaCO_2$ 35 to 45 mm Hg, and PaO_2 150 to 250 mm Hg.

Determination of the LLA

ABP was gradually decreased by infusion into the balloon catheter in the inferior vena cava over 158 ± 50 minutes (mean ± SD). This method causes a slow drift in the ABP to zero, but preserves spontaneous low-frequency (0.05–0.003 Hz) hemodynamic activity, which provides the signal for autoregulation analysis. The method used to determine the LLA has been described elsewhere.[9] Continuous recordings of red cell flux from the laser Doppler were sampled at 100 Hz and recorded as 10-second time-integrated mean values, plotted as a function of ABP. These plots were dichotomized to give 2 data sets with best fit lines having the lowest combined residual error squared. The resultant best fit lines are solved for their intersection, which gives the ABP at the LLA. For this study, the ABP at LLA was chosen as the gold standard, not CPP at LLA. This is because we sought to evaluate this more clinically relevant question: Can COx detect ABP above and below LLA without an ICP monitor? Showing that CPP above and below LLA can be discriminated has no value for scenarios in which ICP is not monitored.

Continuous Autoregulation Monitoring

ABP, ICP, and rSO_2 were sampled at 100 Hz to a computer with ICM+ software (Cambridge University, Cambridge, UK). These waveforms were then low-pass filtered by recording 10-second time-integrated mean values, which removes pulse- and respiratory-frequency oscillations but preserves slow waves, which have a period between 20 and 300 seconds. COx_{CPP} is calculated by linear (Pearson's) correlation between 30 paired samples of CPP and rSO_2 (a 300-second epoch). COx_{ABP} is calculated in the same manner using ABP instead of CPP. The COx values are updated every 10 seconds in a moving, 300-second window. Values of COx approaching +1 indicate that the rSO_2 is passive to ABP at the frequencies of slow waves, which should be autoregulated. Values of COx approaching 0 indicate that rSO_2 is appropriately constrained in the face of slow hemodynamic oscillations. According to common clinical practice with autoregulation monitoring, all values of COx_{ABP} and COx_{CPP} were sorted according to the ABP at which they were measured and averaged in bins of ABP spanning 5 mm Hg. These bins were normalized to the known LLA of the animal (LLA, LLA + 5, LLA − 5, etc.) for evaluation of the ability of the COx to discriminate ABP above and below the LLA.

Statistical Analysis

Receiver operating characteristics were determined for the COx_{ABP} and COx_{CPP} by sorting the binned, averaged values of COx above and below the LLA, excluding values obtained at the LLA. Furthermore, the same binned, averaged values of COx_{ABP} and COx_{CPP} were

compared with linear correlation (Spearman rank test) using Prism software (GraphPad, San Diego, CA). Bland-Altman analysis, accounting for repeated measures, was performed comparing the same measurements of COx_{ABP} and COx_{CPP} using STATA (version 10.1; Stata Corp., College Station, TX).[19,20]

RESULTS

During the induction of hypotension, the average ICP was 10.0 ± 3.7 mm Hg and the average LLA was ABP of 48 ± 10 mm Hg (mean ± SD). For gross characterization of the ability of the COx_{ABP} and COx_{CPP} to delineate a hypotensive state associated with loss of autoregulation, the summary curves of COx_{ABP} and COx_{CPP} for all 25 animals, normalized to each individual LLA, are shown in Figure 1. Data from these plots above the LLA (122 points) and below the LLA (113 points) were used to determine the sensitivity and specificity of this display method to detect ABP below the LLA.

Receiver operating characteristic curves for both indices had the same area under the curve of 0.91 (95% confidence interval [CI] of 0.88–0.95 was also the same), shown in Figure 2. However, the 2 indices have different threshold values at which sensitivity and specificity are optimized. Table 1 shows candidate threshold values for both COx_{ABP} and COx_{CPP} with resultant sensitivity and specificity for detecting the LLA with the binning method in this study. Using a similar sensitivity cutoff of 89%, the COx_{ABP} threshold is 0.5 with specificity of 82% (95% CI, 74%–88%) and a likelihood ratio of 4.69, whereas the COx_{CPP} threshold is lower at 0.42 with specificity of 77% (95% CI, 69%–84%) and a likelihood ratio of 3.86.

Figure 3 shows the scatter of COx values above and below the LLA for each index, with the candidate threshold values of 0.5 for COx_{ABP} and 0.42 for COx_{CPP} superimposed. The COx_{ABP} and COx_{CPP} were compared by linear correlation and the Bland-Altman method (Fig. 4) using all 259 data points obtained from the described binning and averaging method, without excluding points obtained at the LLA. Spearman correlation coefficient between the 2 indices was highly significant ($r = 0.96$, $P < 0.0001$). Bias with the Bland-Altman analysis was −0.08 and the 95% limits of agreement were −0.22 to 0.18.

DISCUSSION

Autoregulation monitoring has gained traction in the neurosurgical arena, where it can be used to delineate optimal perfusion pressures in patients with head trauma.[7,21,22] However, outside of the neurocritical care unit, there is a paucity of experience with the modality despite an abundance of patients at high risk of sustaining neurologic injury. For the non-neurosurgical patient at risk of brain injury, the ICP monitor is often contraindicated.

Monitoring autoregulation with near-infrared spectroscopy and an arterial catheter is practical, involves minimal risk, and is technically simple, but in the absence of an ICP monitor, requires the assumption that slow waves of ABP are accurate surrogates for low-frequency perfusion pressure changes in the brain. This study examined this assumption and showed, in a piglet model with naïve ICP, that COx_{ABP} has an accuracy that is highly comparable to COx_{CPP}. Furthermore, these data indicate that the use of ABP-derived COx

results in a slightly higher threshold COx value for discriminating the LLA compared with CPP-derived COx in the same animals.

Interpretation of these results is tempered by the fact that the data were obtained in a controlled laboratory setting with fixed blood gas values and a graded induced drift in ABP that allowed a complete assessment of each animal's autoregulation curve. Although this is the best way to test the 2 methods against a standard determination of the LLA, and against each other, the laboratory environment does not mimic the relatively chaotic clinical scenario of patients at risk of cerebral hypoperfusion. Furthermore, all of the data were obtained in infant swine with immature and uninjured brains. This limitation affects application of the presented data to adult populations. The COx has previously been monitored in adult patients with sepsis and adult patients undergoing cardiopulmonary bypass. Although the data presented herein are from neonatal swine, it is helpful to interpret the findings from those studies, which used ABP to estimate perfusion pressure changes in the brain.[11,12]

The neonatal swine model is most relevant to the pediatric population. In children, traumatic brain injury is a leading cause of death, infants born at 28 weeks of gestation can expect a 20% incidence of developmental delay, and congenital heart surgery is associated with a 30% to 70% incidence of new ischemic lesions.[23–25] The method described herein is imminently translatable to these populations.

We do not assert that autoregulation monitoring will eliminate the burden of neurologic injury in susceptible patient populations. However, these data present a viable metric that can be deployed without an ICP monitor to discriminate ABP that yields impaired cerebrovascular autoregulation from ABP that yields intact cerebrovascular autoregulation. It is reasonable to posit that knowing a patient's LLA will lead to an improvement in care.

Acknowledgments

Supported by the Hartwell Foundation and the Foundation for Anesthesia Education and Research (both to KMB).

References

1. Lassen NA. Cerebral blood flow and oxygen consumption in man. Physiol Rev. 1959; 39:183–238. [PubMed: 13645234]
2. McCall ML. Cerebral circulation and metabolism in toxemia of pregnancy; observations on the effects of veratrum viride and apresoline (1-hydrazinophthalazine). Am J Obstet Gynecol. 1953; 66:1015–30. [PubMed: 13104502]
3. Drummond JC. The lower limit of autoregulation: time to revise our thinking? Anesthesiology. 1997; 86:1431–3. [PubMed: 9197320]
4. Strandgaard S. Autoregulation of cerebral blood flow in hypertensive patients. The modifying influence of prolonged antihypertensive treatment on the tolerance to acute, drug-induced hypotension. Circulation. 1976; 53:720–7. [PubMed: 815061]
5. Finnerty FA Jr, Witkin L, Fazekas JF. Cerebral hemodynamics during cerebral ischemia induced by acute hypotension. J Clin Invest. 1954; 33:1227–32. [PubMed: 13192186]
6. Czosnyka M, Smielewski P, Piechnik S, Schmidt EA, Seeley H, al-Rawi P, Matta BF, Kirkpatrick PJ, Pickard JD. Continuous assessment of cerebral autoregulation—clinical verification of the method in head injured patients. Acta Neurochir Suppl. 2000; 76:483–4. [PubMed: 11450074]

7. Steiner LA, Czosnyka M, Piechnik SK, Smielewski P, Chatfield D, Menon DK, Pickard JD. Continuous monitoring of cerebrovascular pressure reactivity allows determination of optimal cerebral perfusion pressure in patients with traumatic brain injury. Crit Care Med. 2002; 30:733–8. [PubMed: 11940737]
8. Czosnyka M, Smielewski P, Kirkpatrick P, Menon DK, Pickard JD. Monitoring of cerebral autoregulation in head-injured patients. Stroke. 1996; 27:1829–34. [PubMed: 8841340]
9. Brady KM, Lee JK, Kibler KK, Smielewski P, Czosnyka M, Easley RB, Koehler RC, Shaffner DH. Continuous time-domain analysis of cerebrovascular autoregulation using near-infrared spectroscopy. Stroke. 2007; 38:2818–25. [PubMed: 17761921]
10. Pfister D, Siegemund M, Dell-Kuster S, Smielewski P, Ruegg S, Strebel SP, Marsch SC, Pargger H, Steiner LA. Cerebral perfusion in sepsis-associated delirium. Crit Care. 2008; 12:R63. [PubMed: 18457586]
11. Steiner LA, Pfister D, Strebel SP, Radolovich D, Smielewski P, Czosnyka M. Near-infrared spectroscopy can monitor dynamic cerebral autoregulation in adults. Neurocrit Care. 2009; 10:122–8. [PubMed: 18807218]
12. Joshi B, Brady K, Lee J, Easley B, Panigrahi R, Smielewski P, Czosnyka M, Hogue CW Jr. Impaired autoregulation of cerebral blood flow during rewarming from hypothermic cardiopulmonary bypass and its potential association with stroke. Anesth Analg. 2010; 110:321–8. [PubMed: 20008083]
13. Lewis PM, Smielewski P, Pickard JD, Czosnyka M. Dynamic cerebral autoregulation: should intracranial pressure be taken into account? Acta Neurochir (Wien). 2007; 149:549–55. [PubMed: 17476455]
14. Wagner EM, Traystman RJ. Hydrostatic determinants of cerebral perfusion. Crit Care Med. 1986; 14:484–90. [PubMed: 3084172]
15. Sahuquillo J, Amoros S, Santos A, Poca MA, Valenzuela H, Baguena M, Garnacho A. False autoregulation (pseudoauto-regulation) in patients with severe head injury. Its importance in CPP management. Acta Neurochir Suppl. 2000; 76:485–90. [PubMed: 11450075]
16. Brady KM, Lee JK, Kibler KK, Easley RB, Koehler RC, Shaffner DH. Continuous measurement of autoregulation by spontaneous fluctuations in cerebral perfusion pressure: comparison of 3 methods. Stroke. 2008; 39:2531–7. [PubMed: 18669896]
17. Lee JK, Kibler KK, Benni PB, Easley RB, Czosnyka M, Smielewski P, Koehler RC, Shaffner DH, Brady KM. Cerebro-vascular reactivity measured by near-infrared spectroscopy. Stroke. 2009; 40:1820–6. [PubMed: 19286593]
18. Brady KM, Lee JK, Kibler KK, Easley RB, Koehler RC, Czosnyka M, Smielewski P, Shaffner DH. The lower limit of cerebral blood flow autoregulation is increased with elevated intracranial pressure. Anesth Analg. 2009; 108:1278–83. [PubMed: 19299800]
19. Bland JM, Altman DG. Measuring agreement in method comparison studies. Stat Methods Med Res. 1999; 8:135–60. [PubMed: 10501650]
20. Carstensen B, Simpson J, Gurrin LC. Statistical models for assessing agreement in method comparison studies with replicate measurements. Int J Biostat. 2008; 4:1–26.
21. Brady KM, Shaffner DH, Lee JK, Easley RB, Smielewski P, Czosnyka M, Jallo GI, Guerguerian AM. Continuous monitoring of cerebrovascular pressure reactivity after traumatic brain injury in children. Pediatrics. 2009; 124:e1205–12. [PubMed: 19948619]
22. Bratton SL, Chestnut RM, Ghajar J, McConnell Hammond FF, Harris OA, Hartl R, Manley GT, Nemecek A, Newell DW, Rosenthal G, Schouten J, Shutter L, Timmons SD, Ullman JS, Videtta W, Wilberger JE, Wright DW. Brain Trauma Foundation; American Association of Neurological Surgeons; Congress of Neurological Surgeons; Joint Section on Neurotrauma and Critical Care, AANS/CNS. Guidelines for the management of severe traumatic brain injury. IX. Cerebral perfusion thresholds. J Neurotrauma. 2007; 24(Suppl 1):S59–64. [PubMed: 17511547]
23. Adelson PD, Kochanek PM. Head injury in children. J Child Neurol. 1998; 13:2–15. [PubMed: 9477242]
24. Msall ME. The panorama of cerebral palsy after very and extremely preterm birth: evidence and challenges. Clin Perinatol. 2006; 33:269–84. [PubMed: 16765724]

25. McKenzie ED, Andropoulos DB, DiBardino D, Fraser CD Jr. Congenital heart surgery 2005: the brain: it's the heart of the matter. Am J Surg. 2005; 190:289–94. [PubMed: 16023448]

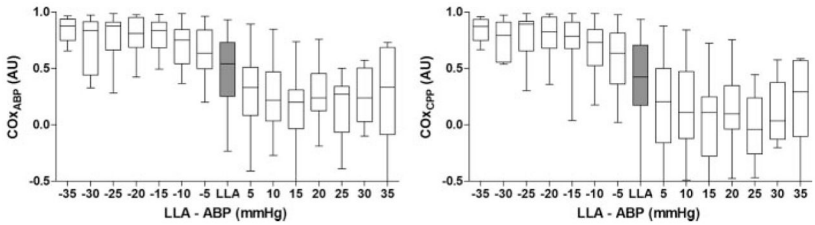

Figure 1.

Cerebral oximetry index arterial blood pressure (COx_{ABP}) and cerebral oximetry index cerebral perfusion pressure (COx_{CPP}) for all 25 animals normalized to the lower limit of autoregulation (LLA). Box whisker plots show range, interquartile percentages, and median values for COx averaged in 5 mm Hg bins for each animal. Shaded boxes show values obtained at the LLA. Binning and averaging dynamic autoregulation metrics is a common display method and these figures allow a visual inspection of the ability of the 2 methods to discriminate adequate from inadequate arterial blood pressure.

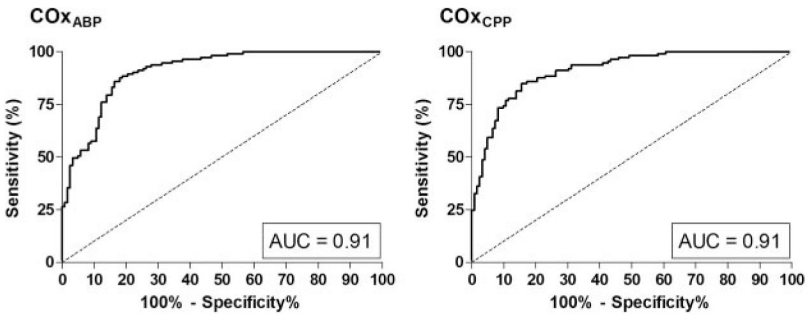

Figure 2.

Receiver operating characteristics for the cerebral oximetry index obtained without intracranial pressure monitoring (COx_{ABP} [left]) and with intracranial pressure monitoring (COx_{CPP} [right]) showing the similar ability of both COx methods to delineate arterial blood pressure (ABP) above and below the lower limit of autoregulation (LLA). AUC = area under the curve.

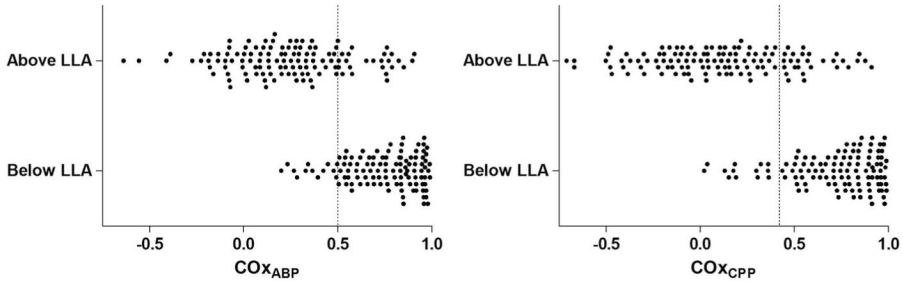

Figure 3.

Scatter plots of the cerebral oximetry index obtained without intracranial pressure monitoring (COx_{ABP} [left]) and with intracranial pressure monitoring (COx_{CPP} [right]) showing data taken above and below the lower limit of autoregulation (LLA) with superimposed candidate threshold values of 0.5 for COx_{ABP} and 0.42 for COx_{CPP}.

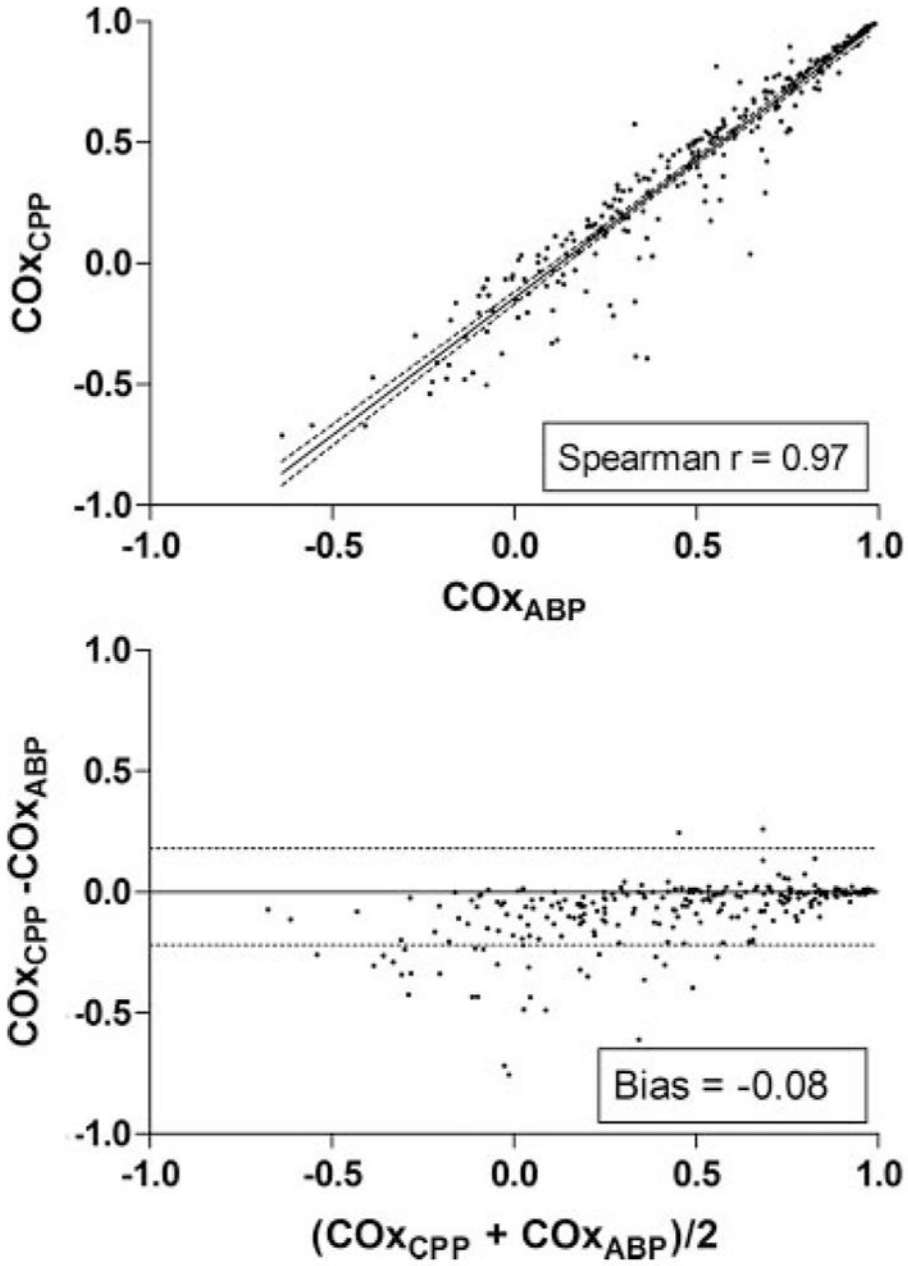

Figure 4.

Comparison of the cerebral oximetry index obtained with-out intracranial pressure monitoring (COx_{ABP}) and with intracranial pressure monitoring (COx_{CPP}) using linear regression (top panel) and the Bland-Altman method (bottom panel).

Table 1

Sensitivity and Specificity for Threshold Values of COx_{ABP} and COx_{CPP} Ranging from 0.3 to 0.6 with Resultant Sensitivity, Specificity, and Likelihood Ratios

Threshold value	Sensitivity (95% CI)	Specificity (95% CI)	LR
COx_{ABP}			
0.30	96.46 (91.18%–99.03%)	59.02 (49.75%–67.83%)	2.35
0.32	96.46 (91.18%–99.03%)	62.3 (53.07%–70.91%)	2.56
0.34	95.58 (89.98%–98.55%)	64.75 (55.59%–73.18%)	2.71
0.36	94.69 (88.80%–98.03%)	67.21 (58.13%–75.44%)	2.89
0.38	93.81 (87.65%–97.47%)	70.49 (61.56%–78.40%)	3.18
0.40	92.92 (86.53%–96.89%)	72.13 (63.29%–79.87%)	3.33
0.42	92.92 (86.53%–96.89%)	73.77 (65.03%–81.32%)	3.54
0.44	92.04 (85.42%–96.29%)	74.59 (65.91%–82.04%)	3.62
0.46	91.15 (84.33%–95.67%)	75.41 (66.79%–82.75%)	3.71
0.48	90.27 (83.25%–95.04%)	76.23 (67.68%–83.47%)	3.8
0.50	88.5 (81.13%–93.73%)	81.15 (73.07%–87.66%)	4.69
0.52	85.84 (78.03%–91.68%)	81.97 (73.98%–88.34%)	4.76
0.54	84.96 (77.01%–90.99%)	83.61 (75.82%–89.69%)	5.18
0.56	79.65 (71.04%–86.64%)	84.43 (76.75%–90.36%)	5.11
0.58	76.11 (67.17%–83.63%)	86.89 (79.58%–92.31%)	5.8
0.60	74.34 (65.26%–82.09%)	87.7 (80.53%–92.95%)	6.05
COx_{CPP}			
0.30	92.92 (86.53%–96.89%)	68.85 (59.84%–76.93%)	2.98
0.32	91.15 (84.33%–95.67%)	69.67 (60.70%–77.67%)	3.01
0.34	91.15 (84.33%–95.67%)	71.31 (62.42%–79.14%)	3.18
0.36	91.15 (84.33%–95.67%)	73.77 (65.03%–81.32%)	3.48
0.37	88.5 (81.13%–93.73%)	74.59 (65.91%–82.04%)	3.48
0.39	88.5 (81.13%–93.73%)	75.41 (66.79%–82.75%)	3.6
0.40	88.5 (81.13%–93.73%)	76.23 (67.68%–83.47%)	3.72
0.42	88.5 (81.13%–93.73%)	77.05 (68.57%–84.18%)	3.86
0.44	87.61 (80.09%–93.06%)	77.05 (68.57%–84.18%)	3.82
0.46	85.84 (78.03%–91.68%)	79.51 (71.25%–86.28%)	4.19
0.48	85.84 (78.03%–91.68%)	82.79 (74.90%–89.02%)	4.99
0.50	84.96 (77.01%–90.99%)	84.43 (76.75%–90.36%)	5.46
0.52	81.42 (73.01%–88.11%)	84.43 (76.75%–90.36%)	5.23
0.54	78.76 (70.07%–85.89%)	86.07 (78.63%–91.67%)	5.65
0.56	76.11 (67.17%–83.63%)	89.34 (82.47%–94.20%)	7.14
0.58	73.45 (64.32%–81.32%)	90.16 (83.45%–94.81%)	7.47
0.60	72.57 (63.37%–80.54%)	91.8 (85.44%–96.00%)	8.85

COx_{ABP} = cerebral oximetry index arterial blood pressure; COx_{CPP} = cerebral oximetry index cerebral perfusion pressure; CI = confidence interval; LR = likelihood ratio.