

Attenuation of cerebral venous contrast in susceptibility weighted imaging (SWI) in pediatric patients under general anesthesia.

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Introduction: Susceptibility weighted imaging (SWI) is known to depict the cerebral vascular system in great detail [1]. It seems to be a promising tool in children for early detection of cerebrovascular pathologies and better clarification of diagnosis [2]. However, young pediatric patients are oftentimes anesthetized during the MRI exam which can lead to a considerable attenuation of the contrast of cerebral veins in SWI [3]. It is known that the deepness of the anesthesia raises the blood CO₂ level which in turn increases the cerebral blood flow (CBF) [4]. Furthermore, it was shown in healthy adults that an increased level of CO₂ in the breathing gas causes a loss of venous contrast in SWI [5]. Purpose of this study was to investigate how anesthesia impacts the venous contrast in SWI by analyzing physiological parameters, which are a measure of the depths of anesthesia, with respect to the observed venous contrast.

Materials and Methods: 108 SWI exams were analyzed: The venous contrast of all exams was classified into two groups one with weak and the other showing a strong contrast between veins and parenchyma. The exams were acquired from 19 patients (11f, 8m; age 3-17y) who are enrolled in an IRB approved clinical study at our institution. The patients suffer from diffuse pontine glioma and were treated with local radiation therapy for 6 weeks. While on study, the patients were also treated with Vandetanib an anti-angiogenic drug. SWI is part of an extensive and advanced MRI protocol to monitor treatment and assess early response of the tumor to therapy. Physiological parameters such as pulse, blood pressure (BP), respiration rate (RR) and end-tidal CO₂ were monitored and recorded by the anesthesiologist during the exams. Anesthesia was maintained by infusion of Propofol (150-300mcg/kg/min). Furthermore, the patient's age and red blood cell count (RBC) at the day of the MR-exam were also recorded.

Results: Figure 1 show examples of SWI data sets which were classified into the two groups of different venous contrasts. Fluctuation of venous contrast has also been observed in the same subjects at different exams. Differences between physiological measures were found in the systolic and diastolic BP with moderate significance ($0.05 < p < 0.001$) and the end-tidal CO₂ with high significance ($p < 0.001$) (Tab.1). The positive mean difference of the BP denotes that the group with the higher venous contrast exhibits higher BP values as compared to the low contrast group. The negative mean difference of etCO₂ denotes that lower etCO₂ values are measured in the group of high venous contrast and vice versa. A box plot of the distributions of the etCO₂ measures is shown in Fig. 2.

Discussion and Conclusion: Our results show the venous contrast in SWI varies with variations in BP and etCO₂. Both parameters are indicators for the deepness of anesthesia, where low BP and high etCO₂ indicates deep anesthesia and high BP and low etCO₂ indicates weak anesthesia. Due to the correlation between deepness of anesthesia and CBF [4] the venous contrast in SWI is most likely modified by changes of the CBF which are in turn induced by anesthesia. The non-significant differences in the age of the two groups may let us conclude that age related differences in physiology have no impact on the venous contrast. Also changes in the RBC which might be caused by the extensive cancer therapy are not responsible for the loss of venous contrast in SWI. These results warrant that any diagnostic argument which is based on venous contrast in SWI needs to take into consideration the actual physiological parameters (at least BP and etCO₂) during the acquisition of the SWI data if the patient was under general anesthesia. However, we feel confident that this effect does not degrade the detection of hemorrhages in anesthetized patients as shown by Tong et al. [6] since the hemorrhagic contrast is caused by extravascular deoxyhemoglobin and methemoglobin rather than by the presence of intravenous deoxyhemoglobin.

References

- [1] Reichenbach JR and Haacke EM. NMR Biomed. 2001;14:453-67.
- [2] Tong KA, et al. AJNR Am J Neuroradiol. 2008;29:9-17.
- [3] Kesavadas C, et al. AJNR Am J Neuroradiol. 2008;29:e71
- [4] Fox J, et al. Anesthesiology. 1992;77:453-6.
- [5] Sedlacik J, et al. Neuroimage. 2008 Jul 18. 2008 Oct 15;43:36-43
- [6] Tong KA, et al. Radiology. 2003;227:332-9

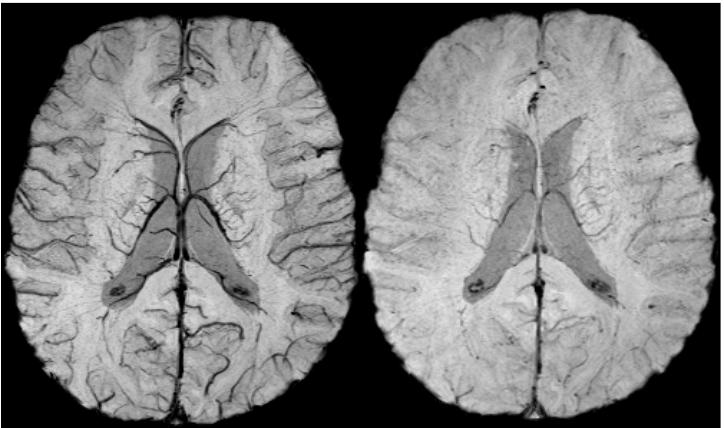


Fig. 1: Minimal intensity projection of 16 mm thick 3D SWI data of a 7 year old patient at two different MRI exams. The patient was under general anesthesia. Images were generated with identical parameters.

Tab. 1: Mean difference of physiological parameters between the two groups of high and low contrast. P-values were derived from Student's t-test (two tailed, two samples, equal variances).

	Age	RBC	Pulse	BP sys/dia	RR	etCO ₂
mean diff.:	0.58 y	0.04 mm ³	-1.27 min ⁻¹	7.13/9.02 mmHg	-0.57 min ⁻¹	-6.80 mmHg
p-value:	0.51	0.62	0.68	0.033/ 0.001	0.64	1E-7

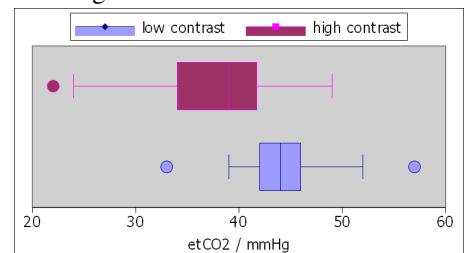


Fig. 2: Box plot of end tidal CO₂ values of the two groups with low or high contrast.