**PURPOSE** In healthy brain tissue virtually all metabolic energy is provided by oxidative metabolism of glucose. In tumor cells, however, this process is markedly shifted towards the anaerobic pathway even if sufficient oxygen is present (`Warburg effect`). Thus, the cerebral metabolic rate of oxygen consumption (CMRO$_2$) can provide valuable information on cell viability and tissue health. By $^{17}$O PET studies, reduced CMRO$_2$ was found in brain tumors [1]. In recent years, MRI of the stable oxygen isotope $^{17}$O has been shown to be able to assess cellular oxygen consumption noninvasively [2]. In this study, we performed an $^{17}$O$_2$-inhalation experiment on a histopathologically verified glioblastoma (WHO grade IV) patient. The CMRO$_2$ was quantitatively evaluated after partial volume correction (PVC) of the data.

**METHODS** Direct $^{17}$O MRI was carried out on a 7 Tesla MR system (Magnetom 7T, Siemens AG, Healthcare Sector, Erlangen, Germany) with a custom-built single tuned birdcage head coil [3]. A 3D data set was acquired every 50 s with an isotropic nominal spatial resolution of 8.5 mm with a density-adapted projection reconstruction pulse sequence (TR/TE = $8.3/0.57$ ms, TR$_{RO}$ = 5.6 ms, 6000 projections, $\theta = 60^\circ$, $T_{AD} = 53$ min) [4]. During $^{17}$O$_2$ inhalation ($t_{start} = 10$ min to $t_{stop} = 19$ min) a total of $4.7 \pm 0.11$ of $70\%$-enriched $^{17}$O gas (Rockland Technimted Ltd., Airmont, NY, USA) were delivered to the patient in small gas pulses ($V = 40$ ml/pulse) via a demand oxygen delivery system (Oxytron3, Weinmann, Hamburg, Germany) [3]. The ‘geometric transfer matrix’ algorithm [5] for PVC was adapted to the MRI methodology and applied to the $^{17}$O data before CMRO$_2$ determination. Therefore, registration and segmentation of high resolution $^1$H data was carried out on $T_1$-weighted images (FMRIB Software Library, FSL) delineating white matter (WM), gray matter (GM), cerebrospinal fluid (CSF), the necrotic part of the tumor (NE), contrast enhancing parts of the tumor (CE) and perifocal edema (PE). The point spread function of the radial sequence, needed for the GTM algorithm, was determined from simulations of the sampled k-space volume incorporating $T_2^*$ relaxation [6]. A three-phase model was used to determine CMRO$_2$ [2]. Quantification of the $H_2^{17}$O concentration was carried out using the baseline signal intensity before gas delivery as an internal reference. Water fractions of the compartments were set to $\lambda_{CSF} = 1$, $\lambda_{GM} = 0.83$, $\lambda_{WM} = 0.71$ [7] and $\lambda_{CE} = 0.71$, $\lambda_{NE} = 0.95$, $\lambda_{PE} = 0.90$ [8], respectively.

**RESULTS AND DISCUSSION** The relative signal increase after $^{17}$O$_2$ inhalation is shown in Figure 1 (w/o PVC). Values range from less than 2% increase in the center of the tumor up to more than 25% in the periphery of the brain. The range of minimal increase is found in the necrotic part of the tumor. The highest signal gain was found in the areas of the gyri with a high gray matter fraction which is in agreement with the higher oxygen consumption of gray matter compared to white matter. Signal time curves before and after PVC are shown in Figure 2. PVC leads to noise amplification especially in the case of CSF. After PVC no increase in CSF is found during $^{17}$O$_2$ delivery.

**CONCLUSION** In this work, we performed the first $^{17}$O$_2$-inhalation experiment on a tumor patient. A partial volume correction algorithm was applied and the CMRO$_2$ of brain and tumor compartments was determined. In agreement with PET results, we found decreased oxygen consumption in the tumor compared to normal brain matter.

**REFERENCES**