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# Functional status is associated with prefrontal cortex activation in gait in subacute stroke patients

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13

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15

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20 **Abstract**

21 The increasing of cerebral oxygenation, more precisely the overactivation of the prefrontal cortex  
22 (PFC), reflects cortical control of gait in stroke disease. Studies about the relationship between brain  
23 activation and the functional status in stroke patients remain scarce. The aim of this study is to  
24 compare brain activation, gait parameters and cognitive performances in single and dual tasks  
25 according to the functional status in subacute stroke patients. Twenty-one subacute stroke patients  
26 were divided in two groups according to Barthel Index ('Low Barthel' and 'High Barthel') and  
27 performed randomly ordered walking, cognitive task (n-back task) and dual tasks (walking + n-back  
28 task). We assessed gait performances (speed, variability) using an electronic walkway system and  
29 cerebral oxygenation ( $\Delta O_2Hb$ ) by functional near infrared spectroscopy. Patients with better  
30 functional status ('High Barthel') showed a lower PFC activation ( $\Delta O_2Hb$ ) and better gait in walking  
31 in single and dual-tasks compared to 'Low Barthel' patients who exhibited decreased gait  
32 performances despite a higher PFC activation, especially in the unaffected side ( $P < 0.001$ ). PFC  
33 overactivation in less functional subacute stroke patients may be due to the loss of stepping  
34 automaticity. Our results suggest that it would be interesting to propose rehabilitation programs  
35 focused on walking, especially for patients with low functional capacity.

### 36 **Introduction**

37 Stroke is associated with gait disorders mainly characterized by a decreased gait speed (Wonsetler  
38 and Bowden, 2017) and greater variability (Balasubramanian et al., 2009). Walking is further  
39 affected by challenging conditions such as simultaneous cognitive and motor tasks, e.g. dual-task  
40 (DT) (Plummer et al., 2013). This increased cognitive demand of walking in DT was underlined by  
41 the key-role of the prefrontal cortex (PFC) (Yogev-Seligmann et al., 2008), whose activation can be  
42 assessed during the walking of stroke patients by the functional near infrared spectroscopy (fNIRS)  
43 (Gramigna et al., 2017). Studies using fNIRS reported a greater brain activity in the PFC during DT  
44 than in single task (ST) in chronic stroke patients, implying that executive functions were especially  
45 involved in this overactivation (Al-Yahya et al., 2016; Hawkins et al., 2018). Recently, we observed  
46 no difference of oxygenated hemoglobin levels ( $\Delta O_2Hb$ ) between motor ST and DT in subacute  
47 stroke patients (Hermand et al. 2019), highlighting a ceiling effect on brain activity observed in DT,  
48 already triggered during walking in ST, and thus the loss of stepping automaticity in these patients.

49 Other recent findings suggest that people with poorer mobility such as elders or neurological patients  
50 exhibits a higher activation of PFC than control groups during walking, reflecting a higher cognitive  
51 demand (Hawkins et al., 2018; Kahya et al., 2019). In stroke, lower mobility was associated with a  
52 higher (and saturated) recruitment of the PFC in walking tasks (Hawkins et al., 2018; Chatterjee et  
53 al., 2019). Hence, the challenge for upcoming studies investigating brain activation during walking in  
54 stroke patients relies on a better understanding of the relationship between the cortical control of gait  
55 and functional independence.

56 The aim of this study is to compare brain activation, gait parameters and cognitive performances in  
57 ST and DT according to the functional status in subacute stroke patients. We hypothesize that the  
58 PFC activation and the decrease of cognitive/gait performances during ST and DT are greater in  
59 stroke patients with a lower functional status.

60

### 61 **Participants and methods**

#### 62 *Participants*

63 Twenty-one subacute stroke patients (table 1) participated in this study, at the Limoges University  
64 Hospital. Inclusion criteria included: acute or subacute stroke (less than 3 months), first stroke (left or  
65 right middle cerebral artery) and being able to walk 10 meters. Exclusion criteria included previous  
66 neurological disease (e.g., Parkinson's disease, dementia). The study was approved by national ethic  
67 committee (CPP 2017-A01883-50).

68

#### 69 *Functional status*

70 The Barthel Index (BI) (Mahoney and Barthel, 1965) for each patient was evaluated on test day by a  
71 trained hospital practitioner, on a 0~100 point-scale. Patients were assigned in two groups : ‘slight  
72 dependency’, for a higher BI between 91 and 100 (HiB), and ‘moderate dependency’, for a lower BI  
73 between 61 and 90 (LoB) (Shah et al., 1989).

74

### 75 *Design protocol*

76 The patients performed three randomly ordered tests successively: cognitive single task ( $ST_{cog}$ ),  
77 walking single task ( $ST_{mot}$ ) and a DT. Cognitive tasks for  $ST_{cog}$  and DT followed a 2-back task  
78 (Hermand et al., 2019): the experimenter, facing the patient at a distance of 1 m during  $ST_{cog}$  or  
79 walking 1 m behind him/her during DT, read aloud and clearly a series of 20 fixed random numbers,  
80 between 0 and 10, evenly spaced in a 30-s interval. Responses were recorded with a voice recorder.  
81 The percentage of correct answers was computed for each cognitive condition, as missing or  
82 incorrect answers were accounted for as errors (Hermand et al., 2019). In walking  $ST_{mot}$  and DT,  
83 patients walked through an open space at a comfortable pace for 30s, through a 8-meter GAITRite  
84 walkway (Sparta, USA) which provided speed and stride variability. One practice trial for each ST  
85 and DT task was conducted prior to experimental testing to ensure proper hearing/vision and a good  
86 understanding of each task.

87

### 88 *fNIRS acquisition*

89 Cerebral oxygenation was measured using a fNIRS system (Portalite, Artinis Medical, Netherlands).  
90 Two optodes were placed on symmetrical prefrontal sites Fp1 and Fp2 according to the EEG 10/20  
91 system. Acquisition was made through the Oxysoft software (version 3.0.97.1). Differential  
92 Pathlength Factor was set on 5 as its calculation formula does not apply to patients’ age 50 years and  
93 older (Duncan et al., 1996). In each condition, after a 30 second rest for baseline, patients performed  
94 the 30 second test, before a final 30 second rest phase. A 0.1 Hz low-pass filter was applied to the  
95 fNIRS signal to remove physiological and instrumental noise, and motion artifacts were corrected  
96 using Matlab-based scripts when needed (Fishburn et al., 2019; Hermand et al., 2019). The relative  
97 concentrations in  $O_2Hb$  ( $\Delta O_2Hb$ ,  $\Delta O_2Hb$ -affected and  $\Delta O_2Hb$ -unaffected in the PFC,  $\mu\text{mol.L}^{-1}$ ) in the  
98 test interval (i.e., the last 20 seconds) were then normalized by subtracting to them the mean value of  
99 the last 10 seconds of baseline, immediately before the beginning of the task, ie seated for  $ST_{cog}$  and  
100 standing for  $ST_{mot}$  and DT.

101

### 102 *Statistical analysis*

103 A Shapiro-Wilk test confirmed the non-normal distribution of the  $\Delta O_2Hb$  / gait / cognitive data.  
104 Friedman and Wilcoxon tests were then conducted to compare and assess the respective effects of  
105 functional status (i.e., LoB and HiB) and conditions ( $ST_{cog}$ ,  $ST_{mot}$  and DT) on cerebral activity  
106 ( $\Delta O_2Hb$ ) and gait parameters (speed, gait variability). For all analyses, the statistical significance  
107 level was set at  $\alpha < 0.05$ . The statistical analysis was performed using IBM SPSS® Statistics  
108 version 23 (IBM Corp, Armonk, USA).

109

110 **Results**

111 LoB and HiB groups included stroke patients whose BIs range from 70 to 85 (n=8), and from 95 to  
112 100 (n=13), respectively (Table 1).

113

114 *Brain activation (Fig. 1)*

115 There was an overall BI effect on  $\Delta O_2Hb$  ( $P=0.0022$ ),  $\Delta O_2Hb$ -unaffected ( $P=0.0009$ ) and  $\Delta O_2Hb$ -  
116 affected ( $P=0.040$ ). More precisely, LoB patients exhibited a higher activation than HiB in  $ST_{mot}$  for  
117  $\Delta O_2Hb$  ( $3.13 \pm 1.67$  vs.  $1.48 \pm 1.67 \mu mol.L^{-1}$ ,  $P=0.025$ , fig. 1A) and for  $\Delta O_2Hb$ -unaffected ( $1.70 \pm$   
118  $0.85$  vs.  $0.63 \pm 0.92 \mu mol.L^{-1}$ ,  $P=0.011$ , fig. 1B), and in DT for  $\Delta O_2Hb$ -unaffected ( $2.18 \pm 0.93$  vs.  
119  $1.06 \pm 1.87 \mu mol.L^{-1}$ ,  $P=0.036$ , fig. 1B). No difference was observed for  $\Delta O_2Hb$ -affected in both LoB  
120 and HiB patients (fig. 1C).

121

122

123 -----Insert Figure 1-----

124

125 *Gait and cognitive performances (Table 1)*

126 An overall BI effect was observed on both speed and gait variability. More precisely, speed was  
127 higher and gait variability was lower in HiB patients than in LoB in  $ST_{mot}$  ( $P=0.0017$  and  $P=0.0016$ ,  
128 respectively) and in DT ( $P=0.0018$  and  $P=0.0013$ , respectively).

129 There was no BI effect on cognitive performance across all conditions.

130

131 -----Insert Table 1-----

132

133 *Effects of DT (Fig. 1 and table 1)*

134 No difference between ST and DT on PFC oxygenation was observed for the whole population (HiB  
135 and LoB patients pooled together), and for the HiB group (separately). In LoB patients,  $\Delta O_2Hb$  and

136  $\Delta O_2Hb$ -unaffected were lower in  $ST_{cog}$  than in DT ( $P<0.05$  and  $P<0.01$ , respectively). In LoB  
137 patients,  $\Delta O_2Hb$ -unaffected was lower in  $ST_{cog}$  than in  $ST_{mot}$  ( $P=0.028$ , fig. 1B).

138 No difference was observed between  $ST_{mot}$  and DT on gait parameters (except a trend for gait  
139 variability,  $P=0.085$ ) for all population. However, gait variability was higher in DT than in  $ST_{mot}$  for  
140 HiB patients only ( $P=0,039$ ).

141 There was an overall effect of DT on cognitive performances ( $P<0.05$ ), but this negative impact was  
142 only observed for HiB patients ( $P<0.05$ ) and not for LoB patients ( $P>0.05$ ).

143

144 **Discussion**

145 First, this study shows that patients with a better functional status (HiB) showed a lower PFC  
146 activation and better gait parameters in ST and DT compared to LoB patients, who exhibited  
147 decreased gait performances despite a higher PFC activation, especially in the unaffected side (Fig.  
148 2).

149

150 -----Insert Figure 2-----

151

152 LoB patients required additional attentional resources for walking: this is in accordance with our  
153 previous work in which  $ST_{mot}$  and cognitive-motor DT induced a PFC overactivation (vs.  $ST_{cog}$ ) in  
154 subacute stroke patients (Hermand et al., 2019). We had highlighted the existence of a ‘ceiling’  
155 phenomenon in brain oxygenation induced by walking: a brain overactivation in stroke patients could  
156 be triggered by  $ST_{mot}$  and could not be further augmented by an additional cognitive load in DT  
157 (Hermand et al., 2019). The present study evidences a similar phenomenon in LoB patients, but not  
158 in HiB, which illustrates a greater reliance on cortical control of gait in patients with poor mobility.

159 Moreover, it is interesting to note that the functional status is associated with a sided overactivation  
160 for LoB patients during  $ST_{mot}$  and DT (vs.  $ST_{cog}$ ): the unaffected side was more activated (fig.1B), as  
161 a compensatory mechanism for the affected PFC (fig. 1C), as previously observed during cognitive  
162 tasks (working memory) for unaffected PFC compensating for various ipsilesional damages areas  
163 (Mihara et al., 2012). Despite a higher PFC activation, LoB gait performances remained lower than  
164 HiB, which confirms that a higher BI is associated with a lower gait variability (Balasubramanian et  
165 al., 2009). According to our data, we could assume that, for LoB patients, an increase of a central  $O_2$   
166 availability in the unaffected PFC, under a cognitive or a physiological stress, would not be enough  
167 to compensate for the affected PFC, potentially because the maximal cognitive capabilities might  
168 already be reached ; as a consequence, performance in gait/cognitive tasks remain low, in ST and DT.

169 The overactivation of the unaffected PFC could illustrate this disequilibrium between the affected  
170 and the unaffected sides, and the subsequent reassignment of cerebral tasks to the unaffected side, in  
171 whole or in part (Leone et al., 2017). We can assume that LoB patients may exhibit a primary  
172 recruitment of unaffected PFC to compensate for the deficient side, less available to voluntary gait  
173 control. HiB patients which have better performance in gait and/or cognitive parameters would  
174 have interhemispheric activation balance in PFC and a limited need of additional cerebral O<sub>2</sub>  
175 availability, as observed in normal older subjects (Hawkins et al., 2018; Mori et al., 2018). This also  
176 could involve another potential mechanism during recovery, relying on the interaction between PFC  
177 and other brain areas involved into stepping automaticity, such as premotor and primary motor  
178 cortices, which could enhance compensatory mechanisms in HiB patients, as observed in older  
179 normal subjects (Beurskens et al., 2014). However, in our study, we were not able to measure the  
180 activation of other brain areas, and fNIRS technology only offer a limited depth penetration that does  
181 not allow us to assess the activation of deeper cerebral structures. Lastly, the functional status did not  
182 impact cognitive capacities across all conditions, which comes in accordance with several studies  
183 (Plummer et al., 2013).

184 Second, there were no difference of PFC oxygenation between gait conditions (ST<sub>mot</sub> and DT), but  
185 we observed better gait performances (i.e., gait variability) in ST<sub>mot</sub> compared to DT only for HiB.  
186 This highlights the key-role of functional status on the cognitive-motor interference: HiB patients  
187 with better recovered gait and/or cognition are those who decrease their performance in DT.  
188 Compared to LoB patients in which DT does not impact the already low gait/cognitive performances,  
189 this decrease in HiB patients could be then associated to a “normal” behaviour (Yogev-Seligmann et  
190 al., 2008; Plummer et al., 2013; Mori et al., 2018) and hence may reflect better recovery of walking  
191 capabilities. This discrepancy between LoB and HiB patients in subacute phase could lead to further  
192 reflection on personalized rehabilitation modalities according to their functional status: LoB patients,  
193 more prone to fall risks (Sheikh and Hosseini, 2020), could benefit from rehabilitation strategies  
194 designed to improve stepping automaticity whereas HiB patients may focus on increasing the  
195 complexity of cognitive tasks.

196  
197 In conclusion, our study highlights a PFC overactivation in the unaffected side for less functional  
198 stroke patients, triggered in walking conditions (ST<sub>mot</sub>), potentially setting an upper limit which may  
199 not be exceeded in DT (Hermand et al., 2019). This would likely be due to the loss of stepping  
200 automaticity in ST (i.e., higher-level control of gait), and then is not observed in more autonomous  
201 stroke patients. This overactivation in patients with poor mobility may mean that basic motor task

## **Stroke, functional status & cerebral oxygenation**

202 requires most of their attention resources, despite low motor performance in ST and DT. Also, the  
203 functional status (BI) could be a valuable indicator to assess both motor and cerebral recovery in  
204 stroke patients. Future studies might need to include more subacute stroke patients with various  
205 functional status, evaluated with the Fugl-Meyer Assessment (de Oliveira et al., 2006) or by a 10-  
206 meter gait test (Pellicer et al., 2017), and controlled sociodemographic factors. Finally, the evolution  
207 of brain activation during a follow-up of a stroke patients' cohort during rehabilitation would be  
208 interesting to investigate from acute to chronic phase of stroke.

209

210 **References**

- 211 Al-Yahya, E., Johansen-Berg, H., Kischka, U., Zarei, M., Cockburn, J., and Dawes, H. (2016).  
 212 Prefrontal Cortex Activation While Walking Under Dual-Task Conditions in Stroke: A  
 213 Multimodal Imaging Study. *Neurorehabil Neural Repair* 30, 591–599.  
 214 doi:10.1177/1545968315613864.
- 215 Balasubramanian, C. K., Neptune, R. R., and Kautz, S. A. (2009). Variability in spatiotemporal step  
 216 characteristics and its relationship to walking performance post-stroke. *Gait & Posture* 29,  
 217 408–414. doi:10.1016/j.gaitpost.2008.10.061.
- 218 Beurskens, R., Helmich, I., Rein, R., and Bock, O. (2014). Age-related changes in prefrontal activity  
 219 during walking in dual-task situations: A fNIRS study. *International Journal of*  
 220 *Psychophysiology* 92, 122–128. doi:10.1016/j.ijpsycho.2014.03.005.
- 221 Chatterjee, S. A., Fox, E. J., Daly, J. J., Rose, D. K., Wu, S. S., Christou, E. A., et al. (2019).  
 222 Interpreting Prefrontal Recruitment During Walking After Stroke: Influence of Individual  
 223 Differences in Mobility and Cognitive Function. *Front. Hum. Neurosci.* 13, 194.  
 224 doi:10.3389/fnhum.2019.00194.
- 225 de Oliveira, R., Cacho, E. W. A., and Borges, G. (2006). Post-stroke motor and functional  
 226 evaluations: a clinical correlation using Fugl-Meyer assessment scale, Berg balance scale and  
 227 Barthel index. *Arq Neuropsiquiatr* 64, 731–735. doi:10.1590/s0004-282x2006000500006.
- 228 Duncan, A., Meek, J. H., Clemence, M., Elwell, C. E., Fallon, P., Tyszczuk, L., et al. (1996).  
 229 Measurement of cranial optical path length as a function of age using phase resolved near  
 230 infrared spectroscopy. *Pediatr. Res.* 39, 889–894. doi:10.1203/00006450-199605000-00025.
- 231 Fishburn, F. A., Ludlum, R. S., Vaidya, C. J., and Medvedev, A. V. (2019). Temporal Derivative  
 232 Distribution Repair (TDDR): A motion correction method for fNIRS. *NeuroImage* 184, 171–  
 233 179. doi:10.1016/j.neuroimage.2018.09.025.
- 234 Gramigna, V., Pellegrino, G., Cerasa, A., Cutini, S., Vasta, R., Olivadese, G., et al. (2017). Near-  
 235 Infrared Spectroscopy in Gait Disorders: Is It Time to Begin? *Neurorehabil Neural Repair* 31,  
 236 402–412. doi:10.1177/1545968317693304.
- 237 Hawkins, K. A., Fox, E. J., Daly, J. J., Rose, D. K., Christou, E. A., McGuirk, T. E., et al. (2018).  
 238 Prefrontal over-activation during walking in people with mobility deficits: Interpretation and  
 239 functional implications. *Human Movement Science* 59, 46–55.  
 240 doi:10.1016/j.humov.2018.03.010.
- 241 Hermand, E., Tapie, B., Dupuy, O., Fraser, S., Compagnat, M., Salle, J. Y., et al. (2019). Prefrontal  
 242 Cortex Activation During Dual Task With Increasing Cognitive Load in Subacute Stroke  
 243 Patients: A Pilot Study. *Front. Aging Neurosci.* 11, 160. doi:10.3389/fnagi.2019.00160.
- 244 Kahya, M., Moon, S., Ranchet, M., Vukas, R. R., Lyons, K. E., Pahwa, R., et al. (2019). Brain  
 245 activity during dual task gait and balance in aging and age-related neurodegenerative  
 246 conditions: A systematic review. *Experimental Gerontology* 128, 110756.  
 247 doi:10.1016/j.exger.2019.110756.

- 248 Leone, C., Feys, P., Moumdjian, L., D'Amico, E., Zappia, M., and Patti, F. (2017). Cognitive-motor  
 249 dual-task interference: A systematic review of neural correlates. *Neuroscience &*  
 250 *Biobehavioral Reviews* 75, 348–360. doi:10.1016/j.neubiorev.2017.01.010.
- 251 Mahoney, F. I., and Barthel, D. W. (1965). Functional evaluation: the Barthel index. *Md State Med J*  
 252 14, 61–65.
- 253 Mihara, M., Miyai, I., Hattori, N., Hatakenaka, M., Yagura, H., Kawano, T., et al. (2012). Cortical  
 254 control of postural balance in patients with hemiplegic stroke: *NeuroReport* 23, 314–319.  
 255 doi:10.1097/WNR.0b013e328351757b.
- 256 Mori, T., Takeuchi, N., and Izumi, S.-I. (2018). Prefrontal cortex activation during a dual task in  
 257 patients with stroke. *Gait Posture* 59, 193–198. doi:10.1016/j.gaitpost.2017.09.032.
- 258 Pellicer, M. G., Lusa, A. C., Casanovas, J. M., Ferrer, B.-C. S., Pellicer, M. G., Lusa, A. C., et al.  
 259 (2017). Effectiveness of a multimodal exercise rehabilitation program on walking capacity  
 260 and functionality after a stroke. *J Exerc Rehabil* 13, 666–675. doi:10.12965/jer.1735056.528.
- 261 Plummer, P., Eskes, G., Wallace, S., Giuffrida, C., Fraas, M., Campbell, G., et al. (2013). Cognitive-  
 262 Motor Interference During Functional Mobility After Stroke: State of the Science and  
 263 Implications for Future Research. *Archives of Physical Medicine and Rehabilitation* 94, 2565-  
 264 2574.e6. doi:10.1016/j.apmr.2013.08.002.
- 265 Shah, S., Vanclay, F., and Cooper, B. (1989). Improving the sensitivity of the Barthel Index for  
 266 stroke rehabilitation. *Journal of Clinical Epidemiology* 42, 703–709. doi:10.1016/0895-  
 267 4356(89)90065-6.
- 268 Sheikh, M., and Hosseini, H. A. (2020). Investigating the relationship between spatiotemporal gait  
 269 variability and falls self-efficacy in individuals with chronic stroke. *Physiotherapy Theory*  
 270 *and Practice*, 1–9. doi:10.1080/09593985.2020.1771799.
- 271 Wonsetler, E. C., and Bowden, M. G. (2017). A systematic review of mechanisms of gait speed  
 272 change post-stroke. Part 1: spatiotemporal parameters and asymmetry ratios. *Topics in Stroke*  
 273 *Rehabilitation* 24, 435–446. doi:10.1080/10749357.2017.1285746.
- 274 Yogev-Seligmann, G., Hausdorff, J. M., and Giladi, N. (2008). The role of executive function and  
 275 attention in gait. *Mov Disord.* 23, 329–342. doi:10.1002/mds.21720.
- 276
- 277

278 **Captions**

279 **Figure 1**

280 PFC oxygenation values (A:  $\Delta O_2Hb$ ; B:  $\Delta O_2Hb$ -unaffected; C:  $\Delta O_2Hb$ -affected) for LoB (black dots)  
281 and HiB (white dots) patients in three tests:  $ST_{cog}$ ,  $ST_{mot}$  and DT. (mean  $\pm$  SD)

282 LoB patients: Condition vs.  $ST_{cog}$  (\*:  $P < 0.05$ ; \*\*:  $P < 0.01$ )

283 HiB patients: Condition vs.  $ST_{cog}$  (#:  $P < 0.05$ )

284 LoB vs. HiB (overall), +:  $P < 0.05$

285 LoB vs. HiB, \$:  $P < 0.05$ ; \$\$:  $P < 0.01$

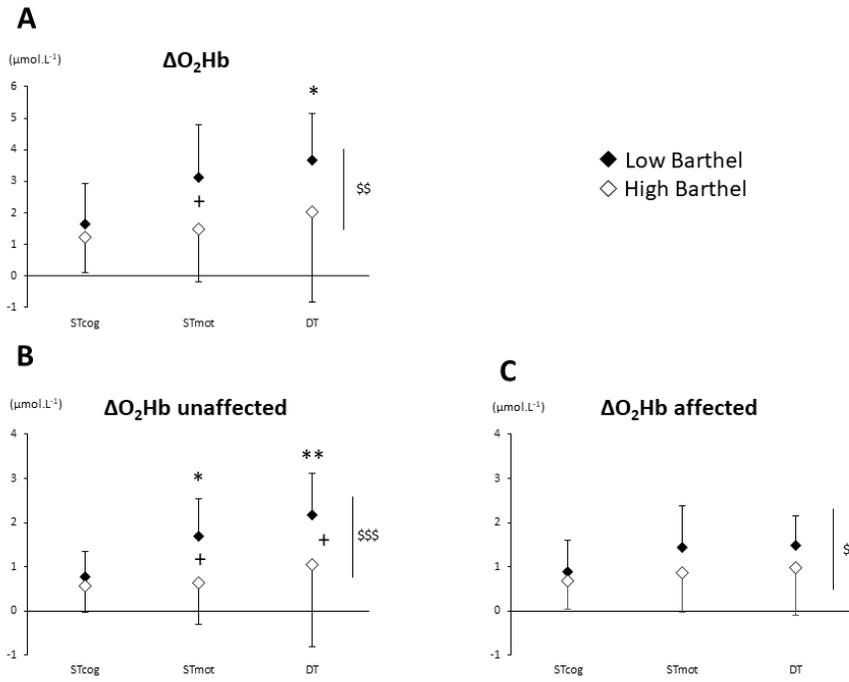
286

287 **Figure 2**

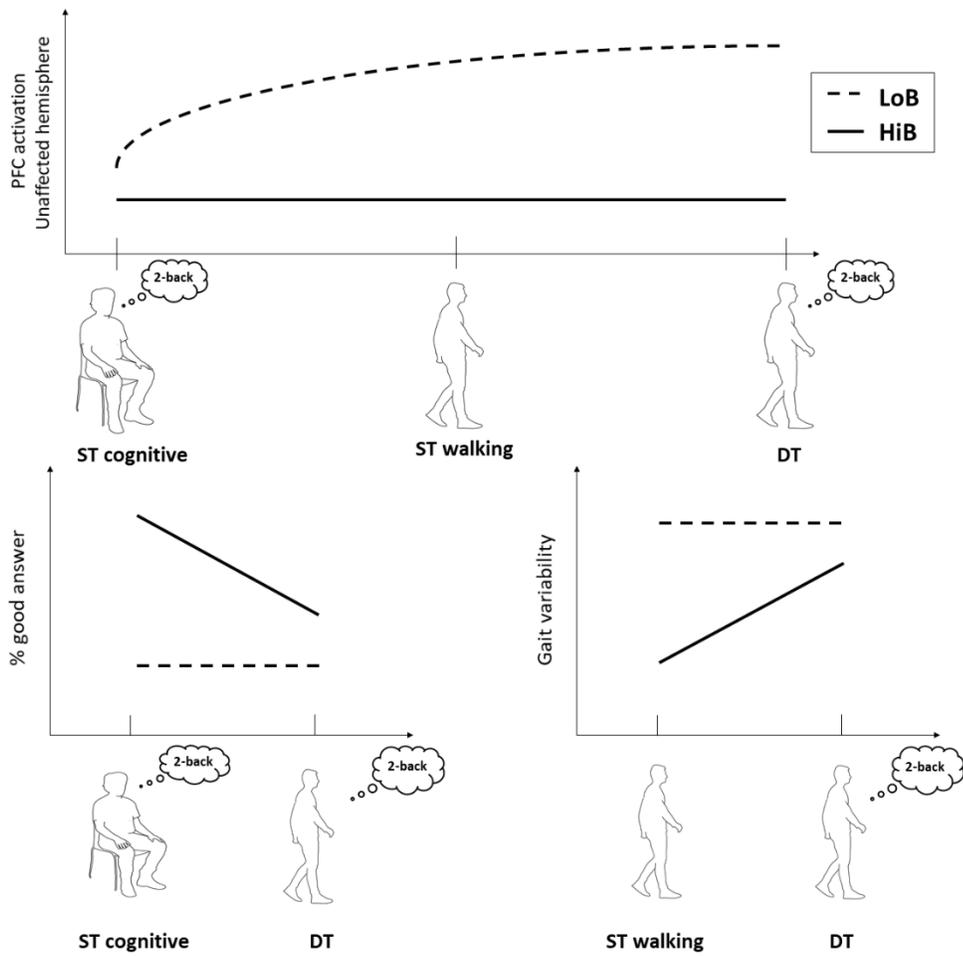
288 Conceptual framework illustrating cognitive-motor performances and cerebral oxygenation in  
289 walking tasks according to functional status.

290

291



292 Figure 1



293 Figure 2

294  
295  
296

297 **Table 1.**298 Clinical characteristics of patients and mean values ( $\pm$  SD) of gait parameters and cognitive  
299 performance in ST and DT (mean  $\pm$  SD).300 LoB vs. HiB: \*\*,  $P < 0.01$ 301 ST<sub>cog</sub> vs DT or ST<sub>mot</sub> vs. DT: +,  $P < 0.05$ 

		LoB group (‘moderate dependency’) (n=8)	HiB group (‘slight dependency’) (n=13)	Overall (n=21)
<b>Clinical characteristics</b>				
Gender (Male/Female)		4/4	10/3	14/7
Age (years)		70.6 $\pm$ 10.5 [57;87]	66.6 $\pm$ 10.4 [56;86]	68.1 $\pm$ 9.4 [56;87]
Height (cm)		167.6 $\pm$ 7.5 [155;181]	168.5 $\pm$ 10.2 [150;183]	168.1 $\pm$ 8.9 [150;183]
Weight (kg)		71.9 $\pm$ 12.8 [52;91]	76.4 $\pm$ 14.8 [60;100]	74.7 $\pm$ 13.1 [52;100]
Barthel Index (/100)		76.3 $\pm$ 6.4 [70;85]	98.1 $\pm$ 2.5*** [95;100]	89.8 $\pm$ 11.5 [70;100]
Walking assistance (one crutch / rollator)		3 / 2	0	--
Days post-stroke		54.5 $\pm$ 39.3 [11;99]	68.1 $\pm$ 28.2 [16;93]	62.9 $\pm$ 30.9 [11;99]
Stroke subtype (Ischemic/Hemorrhagic)		6 / 2	11 / 2	17 / 4
Affected hemisphere (Left/Right)		4 / 4	8 / 5	12 / 9
Level of education		3.6 $\pm$ 1.3 [3;7]	3.5 $\pm$ 1.2 [3;5]	3.5 $\pm$ 1.2 [3;7]
<b>Gait parameters</b>				
<i>Speed (cm.s<sup>-1</sup>)</i>	ST <sub>mot</sub>	40.2 $\pm$ 14.8 [16.1;60]	88.5 $\pm$ 26.5 ** [26.3;131.9]	71.6 $\pm$ 32.7 [16.1;131.9]
	DT	32.4 $\pm$ 14.0 [16.1;56.5]	73.2 $\pm$ 29.6** [21.1;133]	57.7 $\pm$ 31.7 [16.1;133]
<i>Gait variability (n.u.)</i>	ST <sub>mot</sub>	18.2 $\pm$ 14.9 [7.5;46.8]	4.8 $\pm$ 2.5 ** [1.4;10.7]	9.0 $\pm$ 10.3 [1.4;46.8]
	DT	26.3 $\pm$ 16.4 [6.7;46.8]	7.1 $\pm$ 3.6 +** [3.8;14.4]	13.8 $\pm$ 13.5 +
<b>Cognitive performance</b>				
<i>% good answers</i>	ST <sub>cog</sub>	21.4 $\pm$ 11.3 [11.1;44.4]	28.7 $\pm$ 8.1 [16.7;44.4]	26.0 $\pm$ 9.8 [11.1;44.4]
	DT	16.0 $\pm$ 8.6 [5.6;27.8]	16.7 $\pm$ 14.4 + [0;38.9]	16.4 $\pm$ 12.2 +

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