understanding of the workforce situation in the state or community in which they work.

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FATIGUE IN THE ACUTE PHASE AFTER FIRST STROKE PREDICTS POORER PHYSICAL HEALTH 18 MONTHS LATER

Tiffany J. Braley, Devin L. Brown, Ronald D. Chervin, Ann Arbor, MI: Lerdal and Gay¹ examined

a key relationship between acute poststroke fatigue and long-term functional outcomes. We wonder though whether sleep disorders, and obstructive sleep apnea (OSA) in particular, could help to explain the reported observations.

Given the high prevalence of OSA in acute stroke,² we were surprised that data on OSA or OSA symptoms were not presented. Previous studies have shown that OSA is a risk factor for poor post-stroke outcomes.³ Sleep disturbances, including OSA, impair health-related quality of life measures such as the Short Form–36.⁴

Furthermore, despite traditional emphasis on sleepiness as a consequence of sleep apnea, many patients with OSA report that problems with fatigue, tiredness, or lack of energy supersede their problems with sleepiness.⁵

Taken together, these findings raise the possibility that poorer physical outcomes in this cohort could be explained or exacerbated by an underlying sleep disorder and that acute poststroke fatigue may represent a consequence of poor sleep. Next steps in the important research that Lerdal et al. have initiated should consider OSA as a potential causal link between post-stroke fatigue and 18-month physical functioning.

Author Response: Annars Lerdal, Oslo; Caryl L. Gay, San Francisco: Braley et al. asked whether sleep disorders, particularly OSA, might explain the association between acute phase fatigue and poorer physical health 18 months after stroke. Although OSA was not specifically assessed in our study, we previously reported that fatigue during the acute phase was not associated with either the Pittsburgh Sleep Quality Index⁶ or actigraph estimates of nighttime sleep disturbance.⁷ Thus, it seems unlikely that the fatigue patients experienced during the acute phase was solely attributable to sleep disturbance. Nonetheless, given the prevalence of OSA in acute stroke, we agree that this possible mechanism warrants further investigation using specific OSA assessments.

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Tiffany J. Braley, Anners Lerdal, Devin L. Brown, et al.

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