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DOI: [10.1159/000352045](https://doi.org/10.1159/000352045)**The Capsular Warning Syndrome Reconsidered**

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The term ‘capsular warning syndrome’ (CWS) was coined by Donnan et al. [1] in 1993 to describe the phenomenon of repeated sensory and/or motor symptoms affecting at least two of the face, arm and leg, without cortical signs. Since the original description of this phenomenon, other investigators have described the ‘pontine warning syndrome’ caused by stroke in the paramedian pons, describing fluctuations in gaze palsies, dysarthria and ataxic hemiparesis [2–4]. The following case exemplifies the way that fluctuations can continue even when infarction is already present.

A 79-year-old retired nurse in good health suffered sudden-onset slurred speech and right hemiplegia. When she arrived in the emergency department an hour later she was normal again but her right hemiplegia returned shortly thereafter. She was administered IV tPA (intravenous tissue-type plasminogen activator) but continued to fluctuate between mild and severe right hemiparesis several times per day for 5 days after onset, with episodes of worsening typically lasting 20–30 min without relation to blood pressure. Brain MRI performed 2 days after onset, in the midst of her fluctuations, showed an acute lacunar infarct in the left internal capsule/corona radiata on diffusion-weighted imaging. After the fluctuations ceased she had mild paresis that improved over the next 3 months until it was only subtly apparent.

What is occurring during the fluctuations of CWS? Some have hypothesized that damage to small perforating vessels makes them susceptible to hemodynamic changes resulting in transient flow failure [1]. The presence of infarction on MRI in the midst of fluctuations makes it hard to lend credence to claims that transient motor deficits are due to flow failure.

Perhaps the fluctuations result not from waxing and waning ischemia, but failure to adequately engage alternative networks involved in motor control. The motor system is a network that comprises secondary motor regions and nonmotor areas that can be engaged when corticospinal tract damage occurs [5, 6]. Premotor

and supplementary motor areas have connections to the primary motor cortex but also have direct projections to the spinal cord [7]. Even as early as 48 h poststroke, patients with subcortical infarction engage bilateral prefrontal, ipsilateral posterior parietal and bilateral sensorimotor cortices during the attempted performance of a finger-sequencing task [8]. These brain regions may be part of a compensatory pathway that is recruited by the patients in order to perform the motor task. The fluctuations that are observed in CWS may represent perturbation of this complex network. As in any complicated network consisting of multiple interacting regions, disruption of the system can produce oscillations in function.

CWS, which also may be seen in the pontine corticospinal tract, is a well-characterized syndrome that has been poorly named. It is not restricted to the internal capsule and is not a warning, because infarction has already occurred. The pathophysiology remains poorly understood and requires further systematic investigation. Instead of conceiving of CWS as a transient ischemic event that heralds subsequent infarction, it may be that a stuttering pattern of decline is an intrinsic feature of the motor system.

References

- 1 Donnan GA, O'Malley HM, Quang L, Hurley S, Bladin PF: The capsular warning syndrome: pathogenesis and clinical features. *Neurology* 1993; 43:957–962.
- 2 Huang HW, He SW, Tan SQ, Su LL: Patient with pontine warning syndrome and bilateral posterior internuclear ophthalmoplegia: case report. *BMC Neurol* 2010;10:55.
- 3 Muengtawepong S, Singh NN, Cruz-Flores S: Pontine warning syndrome: case series and review of literature. *J Stroke Cerebrovasc Dis* 2010;19:353–356.
- 4 Saposnik G, Noel de Tilly L, Caplan LR: Pontine warning syndrome. *Arch Neurol* 2008;65:1375–1377.
- 5 Grefkes C, Fink GR: Reorganization of cerebral networks after stroke: new insights from neuroimaging with connectivity approaches. *Brain* 2011;134:1264–1276.
- 6 Held V, Szabo K, Bazner H, Hennerici MG: Chronic small vessel disease affects clinical outcome in patients with acute striatocapsular stroke. *Cerebrovasc Dis* 2012;33:86–91.
- 7 Dum RP, Strick PL: Motor areas in the frontal lobe of the primate. *Physiol Behav* 2002;77:677–682.
- 8 Marshall RS, Perera GM, Lazar RM, Krakauer JW, Constantine RC, DeLaPaz RL: Evolution of cortical activation during recovery from corticospinal tract infarction. *Stroke* 2000;31:656–661.