

Tics, TikTok and COVID-19

Rob J Forsyth 

In grappling with COVID-19, the world has been relearning many lessons—from second waves to the importance of mask wearing—from that neglected tutor, the great 1918 (‘Spanish’) influenza pandemic. For neurologists, 1918 produced an additional unforgettable lesson in postinfectious neuropathology as an outbreak of encephalitis lethargica left cohorts of institutionalised adults living with severe Parkinsonism into the 1970s and immortalised in Oliver Sacks’ *Awakenings*.

Mindful of this story, systematic efforts to capture and report neuropsychiatric sequelae of COVID-19 were put in place at the outset of the pandemic and, in contrast to 1918, where a single syndrome predominated, a complex picture including intracerebral haemorrhage, infarction and inflammation is emerging.¹

Our understanding of the links between inflammation, the brain and behaviour has a long and controversial history, particularly in relation to movement disorders. A causal role for H1N1 influenza in the *Awakenings* tragedy is widely assumed, although in fact the evidence is somewhat circumstantial. In the 21st century, we have the hotly debated, proposed phenomenon of paediatric autoimmune neuropsychiatric syndrome (PANS) whose advocates insist on an autoimmune aetiology for many phenomena (particularly obsessive-compulsive disorder and tics) more conventionally viewed as arising from a non-inflammatory, polygenic predisposition.² Such debates have a lineage back to the well-attested mass outbreaks of ‘dancing mania’ of the 14th–17th centuries, where religious fervour

was probably more important than post-infectious processes.

Against this background, Heyman *et al*³ highlight their experience of a marked increase in referrals of young people with severe acute exacerbations of tics during the COVID-19 pandemic, an experience I and anecdotally others share. They strongly favour a functional aetiology for these cases, driven by the introvertible mental health toll on young people of the pandemic and lockdown. If we are to avoid the arid mind versus body, psychology versus cytokines debates that have bedevilled this and related fields for so long, proponents on each side need to generate truly falsifiable hypotheses amenable to experimental testing. In this the ‘functional’ proponents have arguably had more success recently. For too long, functional diagnoses were seen as diagnoses of exclusion, a last resort when no ‘medical’ explanation could be found. Such negative framings are rightly criticised by advocates of inflammatory processes as potential sinkholes of medical ignorance. Equally importantly *negative framings do not generate testable hypotheses*. Increasingly, however, we see functional diagnosis as a process of positive recognition of characteristic features (such as the role of attention in maintenance of signs).⁴ In contrast, the PANS hypothesis is now arguably more lacking in truly falsifiable, testable, experimental predictions precisely because it in turn has been framed negatively as a diagnosis of exclusion.

A key question for Heyman and others to address is ‘why, specifically, tics?’ The rather complex picture of post-COVID-19 neurology¹ contrasts with the homogeneity of the encephalitis lethargica phenomenon. One might suggest that phenomenological homogeneity supports a biological process (consider Sydenham

chorea) but as with the dancing mania phenomenon of the Middle Ages, ‘social contagion’ provides another mechanism, and the role of social media highlighted by Heyman *et al* suggests some directly testable hypotheses that could be the focus of further research in this latest twist in the long story of inflammation and the mind.

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