

Newly qualified doctors should be made aware that not only will they face the usual challenges expected of being a new doctor, but they will also face challenges unique to working during the COVID-19 pandemic, especially in the event of a second wave. They should be prepared to have to adjust to changes in how they work at short notice based on service-provision needs and should be proactive in prioritising their own wellbeing. ■

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What's in a name?

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Editor – We thank Graham *et al* for their recent article.¹ I have been labelled a number of terms over my medical career: foreign student, international medical graduate, foreign doctor and once a brown doctor. I am from Mauritius, studied in Newcastle and have stayed on to practice medicine. Everyone involved with this has struggled with my forename and surname which is Indian in origin and by default, very early on, my appellation has been shorted to Dr Avi. Ward rounds are written under this appellation, my office sign says Dr Avi Aujayeb and recently I have had complaints addressed as such. I hate being called Dr Avi. My parents and family are proud of me being the first doctor in the family and we are proud of my name. Aujayeb means 'dynamic' and Avinash means 'that cannot be destroyed'.² Over time I have given up changing people's mindsets every 4 months or so. From experience, I know everyone knowing and addressing me as Avi makes me more approachable as people have a name that they can pronounce and this is what is acceptable now, and no one will go back to trying to call me Dr Aujayeb. As such, I would just like to point out, that some of us are forced by country specific cultural issues (I wouldn't go as far to call them institutionalised racism) to use their first names, even if we do not want to. ■

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Idiopathic intracranial hypertension

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Editor – Wakerley and colleagues provide a useful update on idiopathic intracranial hypertension.¹ It can be added that, through its relationship with obesity, it is another increasingly prevalent illness of deprivation and poor public health.² One can only hope that there are adequate neurology services in those parts of the country where the illness is most common.³ ■

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Multiple sclerosis

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Editor – I read with interest the article 'Clinical presentation and diagnosis of multiple sclerosis' by Helen Ford; multiple sclerosis (MS) can present as a 'stroke mimic'.¹

In a patient with MS, diagnosing a stroke can be challenging because early signs of a stroke present themselves as an MS flare-up.

An ischaemic stroke must be treated immediately. This can be done by intravenous injection of recombinant tissue plasminogen activator (rtPA) or mechanical thrombectomy or a combination of both, hence it is important to differentiate between a stroke and MS.

MS flares tend to show up more slowly, usually over hours or days, whereas stroke symptoms are sudden and severe and can occur within a few minutes.

MS patients don't normally have a complete loss of vision with an MS flare. They usually get cloudy vision or loss of colour saturation. Stroke patients, on the other hand, will often have a complete loss of vision or half of vision in both eyes.

Loss of ability to speak or understand are common symptoms of stroke whereas muscle spasms, pain, and bowel and bladder problems are more common in an MS flare-up.

Electric shock sensations associated with certain movements usually occur in patients with MS.

The necessity for rapid thrombolysis in acute ischaemic stroke may lead to the treatment of patients with conditions mimicking stroke eg multiple sclerosis. Intravenous thrombolysis (IVT) does not lead to significant complications in 'stroke mimics' suggesting that the risk for IVT-associated complications in this group is low.²

In some patients, symptoms occurs during sleep or the time from when the patient was last seen to be normal is unknown, limited sequence magnetic resonance imaging (MRI) of the brain can be performed to detect if salvageable penumbra is present (restricted diffusion is present and no change on fluid-attenuated inversion recovery (FLAIR) images).

Sometimes, the aetiology of white matter lesions is not clear. Typically, MS lesions in the brain are perivenular and a small vein

occupies the centre of the MS plaque.³ 7 Tesla MRI of the brain demonstrates the presence of a central vessel in 87% of visible white matter lesions called the 'central vein sign'.⁴ ■

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Adrenal insufficiency and checkpoint inhibitors for cancer

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Editor – Simpson *et al* provide a welcome overview of the importance of the recognition and treatment of adrenal insufficiency.¹ However, there is no mention of checkpoint inhibitors for cancer, which have emerged as an important and common cause of secondary adrenal insufficiency, and rarely primary adrenal insufficiency.² The importance and challenge of prompt recognition has been recognised.³ Checkpoint inhibitors are approved globally for the treatment of multiple different cancer types, including non-small-cell lung carcinoma, melanoma, bladder, kidney, and head and neck cancers. Given that around 10% of patients treated with ipilimumab, and 1–2% of patients treated with PD-1/PD-L1 inhibitors develop adrenocorticotropic hormone (ACTH) deficiency, we propose that all patients with current or recent checkpoint inhibitor use should be included in those considered at risk of an adrenal crisis.

In addition, a third of patients receiving a checkpoint inhibitors will require high dose corticosteroids for management of one or more immune related adverse events and it is vital that oncological practice takes on the need to issue these patients with the new steroid card and appropriate education.^{4,5} ■

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A Bayesian strategy for the asymptomatic healthcare worker

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Editor – The mean corpuscular volume, age, platelets and eosinophils (MAPE) strategy proposed by the Formica *et al* might help to resolve the conundrum of the false negative reverse transcriptase polymerase chain reaction-based test (so-called antigen test) for SARS-CoV-2 and the transiently negative antigen test for that infection (ie COVID-19) in some healthcare workers.^{1–4} The conundrum can only be resolved by a Bayesian diagnostic strategy.

The conundrum is compounded by the phenomenon of the transiently negative antigen test.³ This phenomenon was elucidated by Kucirka *et al* who showed that over a 4-day period before the onset of symptoms, the probability of a false negative antigen test result in an infected person decreases from 100% to 67% on day 4. On the day of symptom onset, the median false negative rate can be as high as 38% (95% confidence interval (CI) 18–65%). This decreases to 20% (95% CI 12–30%) 3 days after symptom onset.³

Accordingly, when routine testing is undertaken among healthcare workers, a complementary strategy would be an evaluation of C-reactive protein (CRP) as well as MAPE. In one retrospective study, a high-sensitivity CRP level equal to or greater than 4 mg/L was present in 95.0%, 52.2%, 74.7% and 86.7% of COVID-19 patients as opposed to 87.2%, 28.8%, 31.3% and 45.2% of controls, respectively. The sensitivity of CRP was improved by using that parameter in combination with eosinopenia. The combination of eosinopenia and elevated CRP yielded a sensitivity of 67.9% and a specificity of 78.2% for the diagnosis of COVID-19 infection. The area under the curve amounted to 0.730.⁵

Given that in a retrospective study of 27 COVID-19 patients, the size of the lung lesions detected by computed tomography (CT) of the chest also showed a correlation with CRP, the detection rate of infected persons with falsely negative antigen tests might, arguably, be further improved by the combined strategy of evaluation of MAPE (which includes eosinopenia), CRP and CT of the chest; the latter utilised only in those subjects with high CRP.⁶ The combination of MAPE, raised CRP, and CT-identifiable lung pathology would powerfully enhance the pre-test probability of COVID-19 infection. The deciding factor for triage of healthcare workers with a negative test result would be the weight of pre-test probability. ■

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