express CYP27B1 and can convert 25(OH)D$_3$ to 1,25(OH)$_2$D$_3$. Significant amounts of 1,25(OH)$_2$D$_3$ can be produced locally by the involved immune cells during infection. However, VDBP controls T cell responses to vitamin D by sequestering 25(OH)D$_3$ and inhibiting the production of 1,25(OH)$_2$D$_3$ in T cells.\textsuperscript{5}

Based on these findings, we believe that further research should also focus on VDBP in COVID-19 patients.\textsuperscript{6}

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**Supplemental oxygen in COVID-19: a friend or foe?**

**DOI:** 10.7861/clinmed.Let.20.5.3

Editor – We read the article ‘Potential role of endothelial cell surface ectopic redox complexes in COVID-19 disease pathogenesis’ with great interest.\textsuperscript{1} Dr Isabella Panfoli explains the cause of viral damage-induced microvascular inflammation and thrombosis seen in susceptible people with COVID-19. She proposes ectopic expression of electron transport chain (ETC) on the luminal endothelial cell (EC) membrane secondary to viral damage as a cause of luminal oxidative stress priming microvascular thrombosis. She comments that high oxygen input in the presence of impaired ectopic ETC can result in uncontrolled augmented reactive oxygen species (ROS) production that can be prevented by strict fine tuning of oxygen flux during mechanical ventilation. This is concordant with the experimental study by Helmerhorst et al who demonstrated prolonged ventilation with higher oxygen concentrations (hypoxia) induced immune response in pulmonary compartment in mice.\textsuperscript{2} In contrast to these, Goyal et al put forward that hypoxia is itself pro-inflammatory and its timely detection and correction by oxygen supplementation likely improves mortality in COVID-19 patients.\textsuperscript{3} So, titrating fractional inspired oxygen (FiO2) to correct hypoxia but without causing hyperoxia that could result in deleterious ROS production is of paramount importance. But which clinical criteria determines correctly the transition line between harm and therapy by supplemental oxygen? What is the correct timing? Can ROS scavengers (including N-acetyl cysteine, glutathione, alpha-lipoic acid or ascorbic acid), nuclear factor erythroid 2-related factor 2 (nrf-2) agonists, ETC complex I or III inhibitors or angiotensin-II blockers be used to liberally increase FiO2 during mechanical ventilation? Are there other sources of ROS than ECs? It seems that we need further experimental and clinical studies to answer even the optimal dosing of supplemental oxygen in correcting hyperoxia in patients with COVID-19.\textsuperscript{4}

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**Challenges to new doctors during the pandemic**

**DOI:** 10.7861/clinmed.Let.20.5.4

Editor – Thank you for publishing the article ‘FiY101: A guide for newly qualified doctors’.\textsuperscript{1} It offers a practical approach to managing common queries and anxieties for new doctors. As a trainee working through the COVID-19 pandemic from its start, I have observed and experienced a number of challenges to ways in which we work and to our wellbeing that are relevant to newly qualified doctors. I wish to highlight a few of these alongside the helpful advice already given.

The authors rightly mention that foundation rotas are subject to change. During the pandemic, not only have rota patterns changed but a number of doctors have been redeployed to entirely different departments.\textsuperscript{2} Often those redeployed first have been foundation doctors who had to readjust not only to a new rota but also to a new team and have had to cover patients with completely different problems at short notice – as in the case of doctors redeployed from surgical to medical jobs.

New doctors should also bear in mind the need to prioritise booking annual leave early. It is often difficult to coordinate leave with other members of the team, on-call commitments and social events.\textsuperscript{3} There may be a temptation to delay booking leave even the absence of definite social events. However, I would advise, based on my experience, that it would be more prudent to book leave even in the absence of definite social plans as many of us have found that we needed the time away from work simply to rest and recover.

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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.

RUTH PORTHER
Core trainee 2, Nevill Hall Hospital, Abergavenny, UK

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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.

AVINASH AUJAYEB
Consultant in respiratory and acute medicine, Northumbria Healthcare NHS Foundation Trust, Cramlington, UK

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Idiopathic intracranial hypertension

DOI: 10.7861/clinmed.Let.20.5.6

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.

PAUL MORRISH
Consultant in neurology, Gloucestershire, UK

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Multiple sclerosis

DOI: 10.7861/clinmed.Let.20.5.7

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 periventricular and a small vein...