Surge capacity and updated admission criteria: response of the NHS-commissioned national respiratory extracorporeal membrane oxygenation network to the COVID-19 pandemic

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Editor—Since 2011, NHS England has commissioned a national multicentre service to provide extracorporeal membrane oxygenation (ECMO) for adult patients with potentially reversible respiratory failure refractory to conventional management. We recently reported survival to ICU discharge of 74% for 1205 patients treated during the first 6 yr of the network service.[1] The 2009 H1N1 influenza pandemic showed the effectiveness of referral and transfer to an ECMO centre in adults with severe acute respiratory failure suggested by the CESAR trial,[2,3] and was a driver of the creation of the national ECMO network in England. Since its inception, clinicians have anticipated and planned for the potential future need for surge capacity related to pandemic respiratory illness. In response to the coronavirus disease 2019 (COVID-19) pandemic, the number of ECMO beds available was increased from 15 to 100, and centres were designated to formally cover referrals from Wales, Scotland, and Northern Ireland.[4]

At peak surge activity (April 22, 2020), 87 of these 100 beds were occupied, and as of May 31, 2020, 216 patients have received ECMO for COVID-19-related respiratory failure. In April 2020, 898 patients were referred to the service, 18% of whom were accepted and admitted to an ECMO centre. For comparison, referrals in April 2018 and 2019 totalled 98 and 82, respectively.

Clinicians in the ECMO service agree that the current evidence base does not allow strict criteria to determine who might benefit from ECMO, and encourage the referral of all patients with acute respiratory failure from a reversible noncardiac cause without refractory multiorgan failure to their regional ECMO centre. The decision to proceed to ECMO is ultimately based on clinical judgment made by a multidisciplinary group of experienced clinicians, often on the basis of initial information received over the phone or an electronic form.

At the start of the response to COVID-19 and in the absence of previous disease-specific data to guide decision-making in patients with COVID-19, the ECMO service agreed to be more explicit with the referral criteria (Table 1) to assist clinicians in identifying potential patients.[4] Although the scoring systems listed have not been validated in some patient populations (e.g. Clinical Frailty Score[5] in patients <65 yr old), and clearly not validated in COVID-19 viral pneumonitis, the aim of the criteria is to standardise and triage patients on referral. In addition, this strategy ensures that clinicians should involve at least one other ECMO centre in deciding if it is appropriate to proceed with a patient with a lower probability of benefit (e.g. lower Respiratory ECMO Survival Prediction [RESP] score[6]).

We would emphasise that these criteria cannot be extrapolated to other conditions. For example, the UK dataset shows that some non-COVID conditions are very likely to benefit from ECMO support (asthma and trauma are associated with survival of 95% and 84%, respectively).[1] Therefore, clinicians should continue to refer those patients they believe may

Table 1 Advisory inclusion and exclusion criteria for respiratory ECMO support during the COVID-19 pandemic.

Inclusion criteria:
• Potentially reversible severe respiratory failure
• Murray lung injury score[7] ≥3
• Failed trial of ventilation in prone positioning ≥6 h (unless contraindicated)
• Failed high PEEP ventilation strategy ≥6 h (unless contraindicated)
• Clinical Frailty Scale[5] ≤3
• If RESP score[6] ≤3 ECMO should be considered only after agreement across at least two centres
Exclusion criteria:
• Refractory multiorgan failure

COVID-19, coronavirus disease 2019; ECMO, extracorporeal membrane oxygenation; PEEP, positive end-expiratory pressure; RESP, respiratory extracorporeal membrane oxygenation survival prediction.

benefit from ECMO, understanding that these modified referral criteria are not absolute but part of a dynamic shared decision-making process between the referring team and the ECMO service.

References

1. Warren A, Chiu Y, Villar S, et al. Outcomes of the NHS ECMO national extracorporeal membrane oxygenation service for adults with respiratory failure: a multicentre observational cohort study. *BJA* 2020. in press
2. Noah MA, Peek GJ, Finney SJ, et al. Referral to an extracorporeal membrane oxygenation center and mortality among patients with severe 2009 influenza A(H1N1). *JAMA* 2011; **306**: 1659–68
3. Peek GJ, Mugford M, Tiruvoipati R, et al. Efficacy and economic assessment of conventional ventilatory support versus extracorporeal membrane oxygenation for severe adult respiratory failure (CESAR): a multicentre randomised controlled trial. *Lancet* 2009; **374**: 1351–63
4. Available from: https://www.england.nhs.uk/coronavirus/wp-content/uploads/sites/52/2020/04/C0156-Extra-Corporeal-Membrane-Oxygenation-ECMO-Adult-Speciality-Guide-1.pdf. [Accessed 3 June 2020]
5. Rockwood K, Song X, MacKnight C, et al. A global clinical measure of fitness and frailty in elderly people. *CMAJ* 2005; **173**: 489–95
6. Schmidt M, Bailey M, Sheldrake J, et al. Predicting survival after extracorporeal membrane oxygenation for severe acute respiratory failure. The Respiratory Extracorporeal Membrane Oxygenation Survival Prediction (RESP) score. *Am J Respir Crit Care Med* 2014; **189**: 1374–82
7. Murray JF, Matthay MA, Luce JM, Flick MR. An expanded definition of the adult respiratory distress syndrome. *Am Rev Respir Dis* 1988; **138**: 720–3

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Potential pathophysiology of COVID-19 in patients with obesity. Comment on *Br J Anaesth* 2020; 125: e262–e263

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Editor—We read with great interest the correspondence of Memtsoudis and colleagues,[1] which reported an over-prevalence of obesity amongst both critically ill coronavirus disease 2019 (COVID-19) patients with respiratory failure admitted to the ICU and severely ill patients not admitted to the ICU. The authors suggested that obstructive sleep apnoea-associated baseline inflammation and generation of intrathoracic shear forces could explain the co-occurrence of obesity and severe-to-critical COVID-19 disease. We commend the authors for their novel contribution to the evolving literature on COVID-19 and its potential risk factors and pathophysiology. We wish to further propose several pathophysiological pathways that may explain the disproportionate incidence of adverse outcomes amongst obese COVID-19 patients.

Preliminary studies suggest a strong association between obesity and COVID-19 mortality and morbidity. An unpublished observational cohort study of 16 749 COVID-19 patients in the UK found that obese patients were 37% more likely to die in-hospital than non-obese patients after adjusting for co-morbidities and patient characteristics (preprint data available from https://www.medrxiv.org/content/10.1101/2020.04.23.20076042v1). Amongst a cohort of 383 COVID-19 patients in Shenzhen, China, patients identified as obese had a 142% higher risk of developing severe pneumonia compared with non-obese patients.[2] The Intensive Care National Audit & Research Centre report on COVID-19 in critical care showed that the distribution of BMI of COVID-19 patients matched that of the general population, suggesting that obesity is likely not linked to severe COVID-19 infection requiring an ICU admission.[3] However, the report does support evidence of an increased risk of death amongst obese patients admitted to the ICU. Obesity should at least be viewed as a potential risk factor for severe COVID-19 manifestations or worse outcomes, and clinicians and scientists should gain a better understanding of the possible mechanistic role obesity plays in the pathogenesis of COVID-19.[4,5]

Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) enters human cells by binding to the angiotensin-converting enzyme 2 (ACE-2) receptor.[6] This receptor is

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