Coronavirus pneumonitis can cause profound changes in the demands and function of the heart, which may result in the need for haemodynamic support. COVID-19 is a complex condition where multiple pathological factors can combine in a progressive way at any one time¹:

Serial bedside echocardiography can provide insight of the changes that take place as the heart responds to the inflammatory process driven by the lungs. In this article we discuss an approach to echocardiography that will help the treating physician identify the predominant pathophysiology and facilitate correct therapy.

Safety

Of primary importance when performing bedside echocardiography is the prevention of the spread of infectious disease. The following recommendations are in line with the Intensive Care Society’s guidance on focused ultrasound in intensive care (FUSIC):

(i) A dedicated COVID-19 ultrasound machine, ideally one that can stay in the ICU.
(ii) Barrier protection and FFP3 masks during procedure.
(iii) Single-use sachets of ultrasound gel.
(iv) Removal of gel and debris from the probes after use.
(v) Appropriate disposable cleaning wipes that are viricidal and compatible with ultrasound probes.
(vi) Adequate drying time for cleaning solutions.

Service provision and supervision

A pandemic is a difficult time to deliver additional services because resources are limited and demand is high. A full diagnostic echocardiography is time consuming, exposes staff to risks of infection or carriage, and is often superfluous; this burden is increased by serial observation. The goal is to identify serious pathology in a timely manner without unduly burdening the service. An effective approach is one in which the scanning is performed by a trained intensive care clinician who understands the clinical context and treatment modalities available, is able to monitor the response to treatment, and titrate any interventions initiated. A fundamental understanding of echocardiography physics is necessary, combined with a level of practical experience typically seen after FUSIC Heart or British Society of Echocardiography (BSE) Level 1 certification.² In addition, a service requires supporting infrastructure: referral pathways, supervision, education, and governance processes need to be implemented.

Imaging approach

In line with FUSIC heart and BSE Level 1 methodology, qualitative ‘eye-balling’ is sufficient in most cases and reliable quantitative assessment of right ventricular (RV) function and haemodynamic monitoring is desirable. This requires more advanced skills delivered by practitioners holding an advanced qualification, such as the newly released standard for the UK: FUSIC Haemodynamic protocol.³

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COVID-19 can have subtle effects on the heart. The addition of measurements that can help identify RV pathology and monitor change on serial imaging is important for classifying disease and directing interventions.

The details of intervention protocols are beyond the scope of this article, but the relevant articles are referenced and resources detailing these approaches are widely available.

**Appearance on imaging**

Changes that occur with COVID-19 have been well documented and are consistent: RV dilatation in most patients, with or without impairment; and a normal, impaired, or hyperdynamic left ventricle. 5,6

During the early stages of infection there is a significant inflammatory response that drives pyrexia and vasoconstriction, which typically produce a high cardiac output, low systemic vascular resistance (SVR) state. The left ventricle is hyperdynamic in the face of reduced preload and afterload, circulating catecholamines and inflammatory mediators, and a loss of intravascular tone. In a typical scenario, the right ventricle would display a similar hyperdynamic response as the pulmonary vascular resistance (PVR) is also decreased in systemic sepsis.

However, in patients with COVID-19 pneumonitis, profound hypoxaemia causes hypoxic vasoconstriction, which acts to oppose the vasodilation of the pulmonary circulation, resulting in a normal or increased PVR. In addition, RV perfusion may be decreased as this requires an adequate MAP, which is dependent on the SVR. Excessive circulating catecholamines (stress cardiomyopathy) and inflammatory mediators may also cause RV systolic impairment, more evident because of the afterload effects of preserved or high PVR.

This combination of pathologies may cause dilatation of the right ventricle. Measures of RV systolic function—tricuspid annular plane systolic excursion (TAPSE) and RV S prime (RVS)—which are representative measures of longitudinal RV free wall contraction, may be preserved, but in the context of an overall hyperdynamic circulatory state normal values may actually represent early systolic impairment, particularly if there is a discrepancy between the two ventricles.

As disease severity progresses, the right ventricle is exposed to an increasing adverse environment from a combination of iatrogenic insults and progressive lung disease:

(i) High mean airway pressure used during ventilation to manage hypoxaemia, including airway pressure release ventilation (APRV). In particular, during the compliant phase of the disease, alveolar hyperdistention may cause iatrogenic injury by increasing RV afterload.

(ii) Increased consolidation with reduced lung compliance (requiring increased ventilation pressures to achieve adequate tidal volumes).

(iii) Refractory hypoxia, which increases pulmonary vasoconstriction and increases the RV afterload.

(iv) Reduced CO₂ clearance causing worsening pulmonary vasoconstriction.

(v) Superimposed pulmonary infection and systemic infections, which alter the delicate pressure–flow relationships in systems with no physiological reserve.

(vi) Micro- and macropulmonary thromboembolic disease.

(vii) Direct effects on the RV myocardium include ischaemia from low coronary perfusion (secondary to systemic hypotension) and cardiomyopathy caused by stress and/or circulating inflammatory mediators.

(viii) Synergistic effects of existing pulmonary disease such as chronic obstructive pulmonary disease or obesity. 7

As RV distention progresses, venous congestion may cause multiorgan dysfunction and failure. In addition to restrictive fluid therapy protocols and a hyperinflammatory state, the risk of thrombosis increases significantly. The inferior vena cava (IVC) may be dilated with limited variability as a result of high pulmonary and RV pressures, or may show a degree of variability. Significant variability is seen in preload-responsive states when RV function is normal, but can also be seen in non-responsive states when the RV is failing. In established RV failure, features of congestion worsen with decreased or absent IVC variability, with the potential for increasing hypotension, reduced organ perfusion and organ dysfunction.

Possible therapeutic options to optimise the right side of the circulation include: giving cautious monitored boluses of fluid removal; lower ventilatory pressures; prone positioning (which may allow lower ventilatory pressures to be used as lung bases recruit); increased ventilatory pressures to recruit dependent lung and lower PVR; vasoconstrictor therapy to optimise RV perfusion; inotropic drugs to augment RV contractility; pulmonary vasodilator therapy (oral, inhaled, or i.v.); and thrombolysis. Identification of predominating pathophysiology allows correct therapeutic options to be initiated, monitored, and titrated. 7

For the most part, the left ventricle is spared from direct damage, although cases of COVID-19-induced myocarditis are not uncommon. 2 However, there are numerous causes of indirect effects.

Inadequate left ventricular (LV) filling may occur because of: inadequate LV preload (from RV dysfunction, hypovolaemia, pulmonary emboli, high ventilatory pressures); reduced diastolic filling time as a result of tachycardia; or compression by septal bowing from a dilated RV.

Low LV output can occur because of ventricular interdependence or high afterload states caused by excessive vasoconstrictor therapy to manage hypotension resulting from hypovolaemia. Impaired contractility may be caused by any combination of: circulating catecholamines (stress cardiomyopathy); inflammatory mediators; ischaemic injury (global as a result of imbalance between demand and supply, or territorial as a result of existing coronary artery disease); or high-dose sedative agents that exert negative inotropic effects.

Pleural and pericardial effusions may be observed and intervention should be guided by their size and potential lung or cardiac dysfunction, balanced against the risks of drain insertion. Lung ultrasound may be simultaneously performed when assessing for effusions.

**Echocardiography-directed management strategies**

Echocardiography in this setting should be focused and used repeatedly as a haemodynamic monitor. However, understanding the disease process and clinical context is vital to make management decisions based on the findings from the scan. The primary goal is to provide therapy that is successful at managing ventilatory failure whilst preserving RV function as much as possible. This decision tree (Fig. 1) suggests an approach to integration between ultrasound imaging and decision management in ICU patients with COVID-19.

Of note, BSE guidelines for impairment of the RV are as follows:
**Conclusions**

COVID-19 causes profound changes to the right ventricle through multiple different mechanisms. Focused echocardiography is a quick and useful bedside investigation that provides invaluable insight into the multiple overlapping pathologies that can exist. This enables the physician to provide directed and individualised care to support the heart whilst the lung damage is given appropriate time to recover.

**Declaration of interests**

The authors declare that they have no conflicts of interest.

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**Echocardiography for patients with COVID-19**

**Fig 1** Suggested clinical decision tree using echocardiography for patients with COVID-19. ACS, acute coronary syndrome; Angio, angiography; CM, cardiomyopathy; ECMO, extracorporeal membrane oxygenation; IVC, inferior vena cava; NG, nasogastric; norad, noradrenaline (norepinephrine); PASP, pulmonary artery systolic pressure; resp, respiratory; SV, stroke volume; SVV, stroke volume variation.