Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

thorax, which make the pressure necessary to maintain a steady pulse noticeably lower.[5] Dilated/aneurysmal major vessels may have the potential risk of rupture during CPR in children, especially emaciated children, as in this case.

Conflict of Interest

None.

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Hemodynamic Monitoring Options in COVID-19

To the Editor:

Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), an RNA betacoronavirus, is a novel respiratory pathogen whose ominous progression has been implicated in disease that ranges from respiratory failure and acute respiratory distress syndrome (ARDS) to multi-organ dysfunction.[1,2] Among the most susceptible populations at risk for severe infection are patients with cardiovascular comorbidities, such as hypertension, diabetes mellitus, coronary artery disease, and cardiomyopathy, who have a higher representation among nonsurvivors.[2,3] Although pulmonary pathophysiologic processes undoubtedly underlie the perilous coronavirus disease 2019 (COVID-19) course, the emergence of cardiac injury later in the disease could have major implications for outcomes. Markers of myocardial injury, such as troponin and N-terminal pro-brain natriuretic peptide (NT-proBNP), have been shown to increase rapidly in nonsurvivors.[2,3] Although cardiovascular disease appears to predispose some patients to COVID-19–related myocardial injury, acute myopericarditis in the absence of prior disease has also been described, even without signs of interstitial pneumonia.[4] ARDS mechanical ventilation with high positive end-expiratory pressure (PEEP), along with the presence of intravascular thrombosis in the setting of sepsis-induced coagulopathy, could increase susceptibility to pulmonary hypertension, right heart dysfunction, and end-organ (eg, kidney) hypoperfusion.[5] The risk of cardiovascular and renal injury in conjunction with the importance of conservative fluid management as a key tenet of ARDS treatment justifies the need for judicious monitoring of intravascular volume status in the COVID-19 cohort.[6] We reflect on the clinical utility of select popular hemodynamic monitoring devices and suggest that the pulmonary artery catheter (PAC) shows empirical promise as a validated tool that maximizes monitoring capabilities and minimizes expenditure of hospital resources.

It is difficult to estimate intravascular volume with current standard- of- care monitors, which could delay the timely identification of end-organ hypoperfusion and increase the risk of morbidity and mortality. Pulmonary artery catheters (PACs) can measure right atrial or ventricular pressures, pulmonary arterial pressures, and continuous mixed venous oxygen saturation (SvO_2), in addition to having the capabilities to estimate systemic vascular resistance (SVR) and pulmonary vascular resistance (PVR), as well as cardiac output (CO) (Table 1).[7] Together, these parameters could provide valuable information about volume status and cardiac function. Patients afflicted with pulmonary hypertension or right- heart dysfunction secondary to coagulopathy could benefit from PAC-based management of discordant right and left ventricular failure under the guidance of PVR, right ventricular ejection fraction (RVEF), and SvO_2.[5,8] High PVR, low RVEF, and low SvO_2, for instance, might suggest a need for inotropes. Furthermore, accurate estimations of stroke volume could also help avert pre-renal kidney injury. However, we recognize that PA catheterization is not without its limitations and risks. High intrathoracic pressures in the setting of a high PEEP strategy to improve oxygenation in ARDS could lead to overestimation of left ventricular volumes based on PA diastolic pressures. High PEEP could also theoretically increase the risk of PA perforation, one of the most feared complications of PAC use. In addition, the effectiveness of PAC-derived data in significantly altering clinical outcomes still remains equivocal.[9] Nevertheless, we propose that the PAC maximizes resource use in the COVID-19 ICU arena by providing continuous assessments of intravascular volume and cardiac function while limiting staff exposure and personal protective equipment (PPE) usage.

The past 3 decades have seen the emergence of alternative devices to PACs, which are thought to provide similar hemodynamic information in a minimally invasive manner. Minimally invasive technologies can use principles of transpulmonary

Table 1
Technologies Available for Hemodynamic Monitoring in COVID-19 Patients

Monitoring Device	Measurement Method	Parameters	Major Advantages	Major Disadvantages
PAC	Thermodilution	Measurements: right atrial and ventricular pressures, PAP, PCWP, SvO_2 (continuously with specialized PACs) Calculations: CO, SVR, PVR, RVEF	Provides numerous variables to gauge volume status and cardiac function; minimizes expenditure of hospital resources; reliable in ARDS management	High PEEP could lead to overestimation of LV volume by increasing PADP; invasive monitor with risk of PA perforation; equivocal effectiveness in altering clinical outcomes
PiCCO	TPTD, pulse wave analysis	CO, SV, SVV, PPV Volumetric assessments: GEDV, EVLW, PVPI	Continuous, accurate CO relative to PAC; provides volumetric measures of preload (GEDV) and pulmonary edema (EVLW, PVPI); associated with favorable ARDS outcomes	Invasive monitor that requires CVC and arterial catheter*
LiDCO	Transpulmonary lithium dye dilution, pulse wave analysis	CO, SV, SVV, PPV	Continuous, accurate CO relative to PAC; requires arterial catheter without the need for CVC	Unreliable with use of muscle relaxants; not yet examined in ARDS management*
FloTrac	Pulse wave analysis	CO, SV, SVV, PPV	Easy-to-use, operator-independent system	Accuracy of CO remains equivocal, especially in the setting of low SVR; SVV is poorly predictive of volume responsiveness*
NICOM	Thoracic bioreactance	CO, SV, SVV	Continuous, accurate CO that correlates with fluid responsiveness, irrespective of hemodynamic instability or arrhythmias; noninvasive device that uses electrodes	Not yet examined in ARDS management
Esophageal Doppler	Doppler ultrasound in the esophagus at 45° relative to the descending aorta	CO, SV	Accurate assessment of CO and fluid responsiveness; provides invaluable information about preload, afterload, and contractility	Expertise required because improper positioning of the esophageal probe can underestimate CO
Echocardiography (transthoracic, transesophageal)	2D and 3D imaging; pulsed wave Doppler	CO; dynamic parameters of volume responsiveness, ie, respiratory variations in venocaval size, as well as changes in ventricular size, LVOT, VTI, and LV filling pressure	Detects numerous pathophysiological states, such as wall motion abnormalities, LV diastolic dysfunction, and pericardial effusions	No continuous monitoring; expertise required

Abbreviations: CO, cardiac output; CVC, central venous catheter; EVLW, extravascular lung water; GEDV, global end-diastolic volume; LiDCO, lithium dilution cardiac output; LV, left ventricle; LVOT, left ventricular outflow tract; NICOM, noninvasive cardiac output monitoring; PAC, pulmonary artery catheter; PADP, pulmonary artery diastolic pressure; PAP, pulmonary arterial pressure; PCWP, pulmonary capillary wedge pressure; PEEP, positive end-expiratory pressure; PiCCO, pulse index contour cardiac output; PPV, pulse pressure variation; PVPI, pulmonary vascular permeability index; PVR, pulmonary vascular resistance; RVEF, right ventricular ejection fraction; SvO_2, mixed venous oxygen saturation; SV, stroke volume; SVV, stroke volume variation; SVR, systemic vascular resistance; TPTD, transpulmonary thermodilution; VTI, velocity-time integral.

* Pulse contour analysis may be less accurate in the setting of arrhythmias, valve pathology, intracardiac shunts, and extracorporeal circulation.

thermodilution or indicator dye dilution in addition to arterial waveform variation analysis from arterial catheters. These monitors gauge cardiac function through estimations of cardiac output; they can also evaluate intravascular volume status and likelihood of response to a fluid challenge through stroke- volume variation (SVV) and pulse- pressure variation (PPV).[10] Examples include the pulse index contour cardiac output (PiCCO), lithium dilution cardiac output (LiDCO), and FloTrac devices (Table 1). The PiCCO system is unique in its ability to provide volumetric assessments of preload (global end-diastolic volume) and pulmonary edema (extravascular lung water, pulmonary vascular permeability index).[11] PiCCO guidance may be associated with a shorter duration of respiratory support and faster recovery of PaO_2-to-FiO_2 ratios in ARDS patients, perhaps because optimization of extravascular lung water serves as a positive prognostic factor.[12,13] The LiDCO system provides comparable CO measurements to PACs, but unlike the PiCCO, it has not been examined in ARDS patients managed with a

high PEEP strategy; furthermore, the LiDCO is not reliable in the setting of muscle relaxation, given that quaternary ammonium ion-based agents may interfere with the lithium sensor.[10,14] The FloTrac monitor has also gained popularity as an easy-to-use, operator-independent system, but the accuracy of FloTrac-derived CO measurements remains equivocal.[15] Furthermore, unlike the PiCCO and LiDCO monitors, the FloTrac system may not be reliable in septic patients with low SVR, nor could FloTrac-derived SVV be used for fluid optimization, as it is poorly predictive of volume responsiveness.[10]

Noninvasive monitors rely on quantification of thoracic impedance variability, ultrasound imaging, and measurement of velocity-time integrals from Doppler signals to provide information about CO (Table 1). The noninvasive cardiac output monitoring (NICOM) device analyzes phase shifts in the voltage of electrical impulses passed between electrodes on either side of the body; it provides accurate CO values in mechanically ventilated patients that correlate with fluid responsiveness, irrespective of hemodynamic instability or arrhythmias, although it has yet to be studied in the context of ARDS management.[10] Ultrasound-based technologies, such as esophageal Doppler and echocardiography, have mass appeal, as they are also reliable in their real-time assessment of CO and fluid responsiveness.[10] Transthoracic and transesophageal echocardiography provide key information on ventricular function and dynamic parameters of volume responsiveness, such as respiratory variations in venocaval size, and changes in ventricular size, left ventricular outflow tract velocity-time integral, and left ventricular filling pressure.[10,16] However, these monitors are limited by their intermittent nature and the need for expertise in proper ultrasound beam alignment relative to the structures of interest or direction of blood flow. In comparison to PAC use, the application of echocardiography to gauge intravascular fluid status would likely result in increased traffic of healthcare workers to and from the rooms of COVID-19 patients, along with more frequent use of PPE.

The armamentarium of hemodynamic monitoring devices should be exploited to augment therapeutic tactics for conservative fluid management in a population that is vulnerable to sustaining multi-organ injury. The PAC is a promising option that maximizes monitoring capabilities while limiting exposure of healthcare workers and depletion of highly coveted PPE. The PiCCO may be a viable less-invasive alternative as it has been studied in trials of ARDS patients previously, while other monitors still have questionable accuracy and/or applicability in ARDS management with a high PEEP strategy. The COVID-19 landscape should serve as a launching pad for randomized controlled trials to evaluate whether device-driven management promotes beneficial patient outcomes.

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Conflict of Interest

The authors confirm that they have no conflicts of interest to disclose.

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