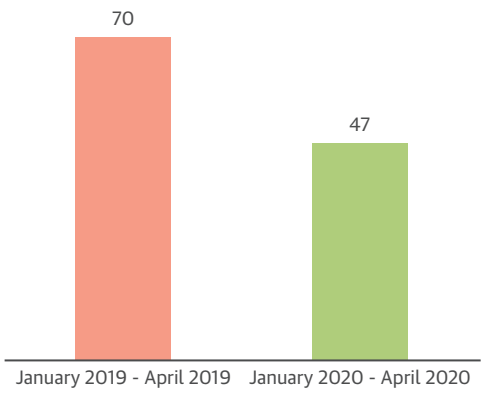
Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

FIGURE 1 Infective Endocarditis Number During COVID-19 Pandemic Versus the Same Time Frame in 2019



Bar chart displaying the decrease in the number of patients diagnosed with infective endocarditis during COVID-19 (coronavirus disease-2019) pandemic period compared with the same time frame in 2019.

Alternative techniques, such as computed tomography (CT), have been proposed (3). CT allows rapid scanning, and it is noninvasive, reducing the time and exposure of patients and personnel (4). It can quickly assess valvular and perivalvular involvement, extracardiac complications, and coronary artery anatomy. Moreover, it may be useful for the evaluation of concomitant pulmonary disease (3). In our centers, CT was performed in 32 (68%) patients with IE during the pandemic and in 52 (74%) patients during the same time frame in 2019, respectively.

IE is a deadly disease that requires a rigorous diagnostic and management approach. Current guidelines recommend early surgery in patients with complicated IE (3). When surgery is indicated but not performed, the mortality is around 50% (2).

Despite the fact that COVID-19 pandemic is the new priority for the health care systems worldwide, patients with IE may be at higher risk than before. Disregarding any SARS-CoV-2 coinfection, the patient with IE should be oriented toward an appropriate treatment pathway, based on detailed clinical evaluation and alternative diagnostic methods, decreasing this unacceptable mortality rate.

Bernard Cosyns, MD, PhD
Andreea Motoc, MD*
Florent Arregle, MD
Gilbert Habib, MD, PhD

*Department of Cardiology
University Hospital of Brussels (Centrum voor Hart- en Vaat ziekten, Universitair Ziekenhuis Brussel)
Laerbeeklaan 101
1090, Jette
Brussels
Belgium
E-mail: andreea.motoc@gmail.com

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The Predictive Role of Left and Right Ventricular Speckle-Tracking Echocardiography in COVID-19

A recent study shows that left ventricular ejection fraction (LVEF) is preserved in most patients with coronavirus disease-2019 (COVID-19) infection, but LV diastolic and right ventricular (RV) function are impaired (1). We assessed left and right myocardial systolic function by speckle-tracking echocardiography (STE) in 100 consecutive patients with COVID-19 and analyzed their prognostic value on survival and need for intubation.

All patients had a diagnosis of COVID-19 confirmed by a polymerase chain reaction assay for severe acute respiratory syndrome-coronavirus-2 and underwent STE examination within 24 h of admission. Demographic, clinical, and laboratory data were systematically recorded. Patients were risk stratified according to their COVID-19 Modified Early Warning

TABLE 1 Impact of Clinical and Echocardiographic Characteristics on Mortality and Clinical Event Rate

	Outcome					
	Death (n = 23)	p Value	Intubation (n = 14)	p Value	Combined (n = 30)	p Value
Age	1.04 (1.02-1.06)	0.001	1.00 (0.98-1.03)	0.70	1.03 (1.01-1.05)	0.001
Male	1.02 (0.36-3.20)	0.90	1.40 (0.46-5.00)	0.60	1.06 (0.47-2.60)	0.90
Modified Early Warning Score	1.30 (1.17-1.50)	<0.001	1.35 (1.16-1.60)	<0.001	1.40 (1.26-1.56)	<0.001
Temperature	1.18 (0.65-2.04)	0.50	1.89 (1.03-3.30)	0.04	1.80 (1.18-2.70)	0.005
O_2 saturation	0.85 (0.78-0.92)	<0.001	0.87 (0.79-0.97)	0.01	0.85 (0.79-0.91)	<0.001
Heart rate	1.00 (0.97-1.03)	0.60	1.04 (1.00-1.07)	0.02	1.03 (1.00-1.05)	0.05
Systolic blood pressure	0.98 (0.96-1.01)	0.40	0.98 (0.95-1.01)	0.20	0.99 (0.98-1.02)	0.90
Diastolic blood pressure	0.97 (0.95-1.00)	0.20	0.99 (0.96-1.03)	0.70	0.99 (0.97-1.02)	0.90
C-reactive protein	1.00 (0.99-1.01)	0.10	1.01 (1.00-1.02)	<0.001	1.01 (1.00-1.01)	0.002
D-dimers	1.07 (0.91-1.18)	0.30	1.18 (1.06-1.30)	0.005	1.12 (0.99-1.22)	0.06
Troponin I	1.00 (0.99-1.00)	0.80	1.00 (1.00-1.01)	0.03	1.00 (0.99-1.00)	0.20
B-type natriuretic peptide	1.00 (1.00-1.02)	0.02	1.00 (0.99-1.01)	0.90	1.00 (0.99-1.01)	0.08
LV assessment						
Ejection fraction	0.93 (0.86-1.04)	0.20	1.00 (0.89-1.18)	0.90	0.96 (0.90-1.06)	0.40
LV end-diastolic diameter	0.97 (0.94-1.01)	0.20	0.98 (0.95-1.03)	0.50	0.98 (0.95-1.01)	0.20
LV end-systolic diameter	0.97 (0.92-1.03)	0.40	0.99 (0.93-1.07)	0.80	0.97 (0.93-1.02)	0.30
E/A	0.28 (0.02-2.00)	0.20	0.02 (0.001-0.30)	0.004	0.44 (0.10-1.60)	0.20
E/e′ average	1.04 (0.96-1.10)	0.20	0.94 (0.78-1.05)	0.40	1.04 (0.98-1.09)	0.10
Peak LV global longitudinal strain	0.84 (0.73-0.96)	0.01	0.88 (0.75-1.03)	0.10	0.83 (0.74-0.93)	0.001
End-systolic LV global longitudinal strain	0.87 (0.76-1.00)	0.05	0.88 (0.75-1.03)	0.10	0.83 (0.74-0.93)	0.001
Peak LV free wall longitudinal strain	0.90 (0.79-1.01)	0.09	0.86 (0.74-0.99)	0.04	0.87 (0.79-0.96)	0.007
End-systolic LV free wall longitudinal strain	0.90 (0.80-1.01)	0.08	0.83 (0.74-0.98)	0.03	0.87 (0.79-0.96)	0.005
Peak LV septal wall longitudinal strain	0.89 (0.78-1.02)	0.10	0.94 (0.81-1.08)	0.40	0.86 (0.77-0.95)	0.003
RV assessment						
Right atrial pressure	1.03 (0.87-1.16)	0.60	1.06 (0.88-1.20)	0.50	1.03 (0.91-1.13)	0.50
RV end-diastolic area	1.02 (0.91-1.14)	0.70	1.04 (0.92-1.19)	0.50	0.97 (0.89-1.06)	0.60
RV end-systolic area	1.05 (0.90-1.19)	0.50	1.10 (0.94-1.20)	0.20	1.02 (0.90-1.15)	0.60
RV fractional area change	0.96 (0.90-1.03)	0.30	0.96 (0.89-1.04)	0.30	0.96 (0.92-1.02)	0.20
Tricuspid annular plane systolic excursion	0.17 (0.07-0.45)	0.005	0.45 (0.15-1.44)	0.20	0.32 (0.15-0.73)	0.008
RV S′	0.82 (0.72-0.96)	0.02	0.86 (0.74-1.04)	0.10	0.81 (0.71-0.93)	0.003
Tei index	6.10 (1.19-24.00)	0.03	0.93 (0.04-7.20)	0.90	4.00 (1.08-12.2)	0.04
Peak RV 4-chamber longitudinal strain	0.89 (0.76-1.03)	0.10	0.85 (0.71-1.00)	0.05	0.85 (0.75-0.96)	0.008
End-systolic RV 4-chamber longitudinal strain	0.88 (0.74-1.02)	0.09	0.86 (0.72-1.01)	0.07	0.85 (0.74-0.95)	0.007
Peak RV free wall longitudinal strain	0.91 (0.81-1.02)	0.10	0.88 (0.78-1.00)	0.06	0.83 (0.75-0.93)	0.0006
End-systolic RV free wall longitudinal strain	0.87 (0.77-0.98)	0.03	0.85 (0.71-1.00)	0.05	0.85 (0.75-0.96)	0.008
Peak RV septal wall longitudinal strain	0.94 (0.81-1.08)	0.40	0.86 (0.73-1.01)	0.07	0.91 (0.82-1.02)	0.10
End-systolic RV septal wall longitudinal strain	0.93 (0.81-1.07)	0.40	0.90 (0.78-1.04)	0.10	0.92 (0.84-1.02)	0.10
Peak RV apical septal segment longitudinal strain	0.93 (0.83-1.05)	0.30	0.95 (0.84-1.09)	0.50	0.95 (0.87-1.05)	0.40
Peak RV mid septal segment longitudinal strain	0.89 (0.79-1.00)	0.05	0.97 (0.85-1.09)	0.60	0.91 (0.83-0.99)	0.04
Peak RV basal septal segment longitudinal strain	1.02 (0.94-1.11)	0.60	0.90 (0.83-1.02)	0.10	0.96 (0.90-1.030	0.30
Peak RV apical free wall segment longitudinal strain	0.97 (0.90-1.05)	0.50	0.90 (0.82-0.98)	0.01	0.95 (0.89-1.00)	0.09
Peak RV mid free wall segment longitudinal strain	0.93 (0.83-1.05)	0.30	0.95 (0.84-1.09)	0.50	0.95 (0.87-1.05)	0.40
Peak RV basal free wall segment longitudinal strain	1.02 (0.94-1.11)	0.50	0.92 (0.83-1.01)	0.10	0.96 (0.90-1.03)	0.30

Values are hazard ratio (95% confidence interval).
E/A = E wave velocity divided by A wave velocity; E/e′ = E wave velocity divided by E prime velocity; LV = left ventricular; RV = right ventricular.

Score (1). Routine computed tomography was not done due to the risk of contamination of the computed tomography area. All patients who experienced clinical deterioration (need for intubation or hemodynamic deterioration) underwent repeated STE. Median time between consecutive measurements was 3.5 days (interquartile range: 3 to 5 days). To reduce exposure and contamination, STE was assessed off-line. Nonadjusted and adjusted Cox proportional hazards models for mortality or combined event (death or new need for intubation) hazard ratios (HRs) were calculated for STE parameters. Analysis for survival was obtained for all patients. Analyses for the combined event were done,

excluding 9 patients who were mechanically ventilated before baseline STE. The ethics committee of the Tel Aviv Medical Center approved the study (Institutional Review Board number 0196-20-TLV) and voided the requirement of informed consent for the echocardiographic assessment.

A total of 100 patients with COVID-19 infection (age 64.3 ± 20.7 years, 64% male) underwent routine echocardiographic evaluation, and subsequent off-line STE evaluation was feasible in 93 (93%), 83 (83%), and 78 (78%) patients for the LV, RV, or both ventricles, respectively. At the time of baseline STE, 61, 26, and 13 patients had mild, moderate, or severe disease, respectively. The latter had high troponin I (763 ng/l), B-type natriuretic peptide (BNP) (75 pg/ml), and C-reactive protein (162.3 mg/l). Although only 11% of patients had EF ≤50%, abnormal LV (based on peak LV global longitudinal strain [GLS] ≤16.6) and RV free wall longitudinal strain (RVFWLS) (≤20.0) were observed in 42% and 38%, respectively. In 35 of 78 (45%) of patients assessed for both ventricles, all strain parameters were in the normal range. The lowest RV strain values were for the mid and apical septal segments ($p < 0.0001$ for trend). Patients with poorer clinical grade levels had worse LVGLS, LVFWLS, RVGLS, and RVFWLS ($p < 0.05$ for all). Of note, septal strain parameters and routine echocardiographic parameters of the LV were not different between the groups. All strain measurements had good intraobserver and interobserver reproducibility. The peak LVGLS intraclass correlation was 0.89 and 0.86, respectively, and for RVFWLS was 0.90 and 0.88, respectively.

Second echocardiography was required in 19% of patients. In these patients, the most common pattern was RV STE deterioration, mostly in the mid segments with apical sparing ($p < 0.05$). At the end of follow-up (27 [18, 40] days) 23 patients died and 14 patients needed intubation, or both. The impact of clinical and STE characteristics on mortality and clinical event rate is summarized in **Table 1**. Survival was reduced with abnormal LVGLS (74 ± 7% vs. 92 ± 8% at 30-day follow-up; $p = 0.05$). Survival was reduced with abnormal RVFWLS (76 ± 7% vs. 92 ± 5% at 30-day follow-up; $p = 0.03$). LVGLS was associated with mortality (HR 0.8; $p = 0.003$] and combined events (HR: 0.80; $p = 0.005$) when adjusted for EF, or if adjusted for tricuspid annular plane systolic excursion and BNP (HR for mortality: 0.56; 95% confidence interval: 0.19 to 0.95; $p = 0.02$; HR for combined event: 0.70; 95% confidence interval: 0.48 to 0.94; $p = 0.01$). RVFWLS was associated with combined events (HR: 0.84; $p = 0.008$) after adjustment for RV S′ or age and Modified Early Warning Score (HR: 0.90; $p = 0.05$) but not if adjusted for tricuspid annular plane systolic excursion and BNP.

A recent report (2) showed that RV strain predicts mortality in patients with COVID-19 infection. However, RV assessment was limited to RVFWLS, and LV strain analyses and repeated exams were not performed. We are the first to show the segmental nature of RV dysfunction, with patterns typical for pulmonary embolism or other types of acute cor pulmonale (3-5). Furthermore, we are the first to evaluate LV strain in patients with COVID-19 infection. We show that abnormal LV longitudinal strain is more common than reduced EF, and that LV STE is superior to LVEF for predicting adverse outcome in patients with COVID 19 infection.

In conclusion, with COVID-19 infection, LV and RV STE are abnormal in ~40% of patients. Poorer clinical grade and clinical deterioration are mostly associated with worsening RV segmental STE, in pattern suggestive of acute cor pulmonale. LV and RV STE are strong predictors of mortality and need for intubation in patients with COVID-19 infection.

Ehud Rothschild, BSc†
Guy Baruch, BSc†
Yishay Szekely, MD
Yael Lichter, MD
Alon Kaplan, BSc
Philippe Taieb, MD
Michal Laufer-Perl, MD
Gil Beer, MD
Livia Kapusta, MD, PhD‡
Yan Topilsky, MD‡*

*Division of Cardiovascular Diseases and Internal Medicine
Tel Aviv Medical Center
6 Weizmann Street
Tel Aviv 6423906
Israel
E-mail: topilskyyan@gmail.com

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†Drs. Rothschild and Baruch contributed equally to this work and are co-first authors. ‡Drs. Kapusta and Topilsky contributed equally to this work and are co-last authors. The authors have reported that they have no relationships relevant to the contents of this paper to disclose.
The authors attest they are in compliance with human studies committees and animal welfare regulations of the authors' institutions and Food and Drug Administration guidelines, including patient consent where appropriate. For more information, visit the *JACC: Cardiovascular Imaging* author instructions page.

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Myocardial Impairment and Acute Respiratory Distress Syndrome in Hospitalized Patients With COVID-19

The ECHOVID-19 Study

Acute respiratory distress syndrome (ARDS) is the primary complication observed in coronavirus disease-2019 (COVID-19)-related deaths (1). Additionally, studies have found cardiac biomarkers to be increased in a significant proportion of patients, emphasizing COVID-19-related cardiac injury (2,3).

We aimed to assess the prevalence and value of assessing myocardial impairment using echocardiography and cardiac biomarkers in hospitalized patients with COVID-19.

The Echocardiographic COVID-19 (ECHOVID-19) study is a prospective multi-center cohort study of hospitalized patients with laboratory-confirmed COVID-19 at 8 hospitals of eastern Denmark (March 30 to June 1, 2020). Inclusion criteria are as follows: laboratory-confirmed Severe acute respiratory syndrome-coronavirus-2 (SARS-CoV-2) and ≥18 years. All patients independent of their health status underwent echocardiography (including two-dimensional-speckle tracking) according to a predetermined protocol and cardiac biomarkers were obtained. Investigators were blinded to the health status of the patients prior to inclusion. The endpoint was ARDS defined according to the Berlin Definition (4). All participants gave written informed consent and the study was performed in accordance with the 2nd Declaration of Helsinki and approved by the regional ethics committee. The study is registered at Clinicaltrials.gov (NCT04377035). Echocardiographic examinations were performed using the portable Vivid IQ Ultrasound System and analyzed off-line using EchoPAC version 203 (GE Healthcare). Elevated troponins were defined as troponins greater than the 99th percentile upper limit reference. A clinical cutoff value of ≥300 ng/l for N-terminal pro-B-type natriuretic peptide was used.

A total of 174 patients were included (mean age: 68 ± 15 years, 55% males). Of the included patients, 14% had prevalent heart disease. Median time from hospital admission to echocardiography was 4 days (interquartile range: 2 to 8). During follow-up (median: 16 days [interquartile range: 6 to 24]), 27 (16%) patients developed ARDS. Patients developing ARDS were older, more frequently men, smokers, hypertensive, and had prevalent heart disease, elevated N-terminal pro-B-type natriuretic peptide, troponin, and C-reactive protein. In addition, they had more impaired systolic function assessed using echocardiography (lower left ventricular ejection fraction [LVEF]: 51% vs. 59%, and global longitudinal strain (GLS): 13.7% vs. 16.9%). Among the 129 patients who had all biochemical and echocardiographic parameters available, 79.1% had myocardial impairment (decreased systolic function as assessed using echocardiography and/or elevated cardiac biomarkers). In this group, 20 developed ARDS, whereas only 2 in the nonmyocardial impairment group developed ARDS.

In Cox regression analysis adjusted for age, sex, body mass index, C-reactive protein, time to echocardiography, hypertension, diabetes, hyperlipidemia, and smoking, both LVEF and GLS were associated with ARDS (**Figure 1A and 1B**). Cardiac biomarkers and systolic parameters were assessed separately. These results did not change when restricting our analysis to patients without prevalent heart disease from the fully adjusted model. Cardiac biomarkers were not significantly associated with ARDS in multivariable models. Patients with preserved systolic function and cardiac biomarkers within the normal range had a low risk of ARDS (specificity: 28%; sensitivity: 96%; positive predictive value: 23%; negative predictive value: 97%).

In this multi-center study, we found that myocardial impairment is a common finding in hospitalized patients with COVID-19 and that it is associated with a higher risk of developing ARDS. We do not propose that impaired systolic function due to COVID-19 infection is a causal agent of ARDS development. Although we found that a combination of cardiac biomarkers and echocardiographic measures could rule out patients at high risk of developing ARDS, the results were based on a low number of events, which are reflected in the low specificity and positive predictive value. A limitation to the study is that patients presenting with myocardial impairment in the study may already have had unacknowledged myocardial impairment prior to infection. Therefore, we do not claim that the COVID-19 infection directly impairs myocardial function because this would require control for previous myocardial performance.