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.

Conclusion These results suggest that type I PKA is important for cardiac growth and is involved in the control of cardiac automatism and calcium homeostasis. Ongoing experiments will determine to which extent the R368X mutation of RIα affects basal and cAMP-stimulated PKA activity in the heart.
Disclosure of interest The authors declare that they have no competing interest.

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Partial cardiac pericyte depletion induces heart failure with reduced ejection fraction

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Introduction While the critical role of pericytes in maintaining vascular integrity has been extensively demonstrated in the brain and in the retina, very little is known about their role in the heart.
Objective To investigate the cardiac structural and functional consequences of partial pericyte depletion (about 60%) in adult mice.
Method To deplete pericytes in adult mice, Pdgfrb-Cre/ERT2; $Rosa^{DTA}$ mice were administered with tamoxifen at 8 weeks of age and again at 10 weeks of age for 5 consecutive days. Control mice were $Rosa^{DTA}$ mice chosen among their littermates and administered with tamoxifen following the same protocol. Cardiac function was assessed via echocardiography and left ventricle (LV) catheterization at 14 weeks of age. Immediately after, mice were sacrificed for histological analyses.
Results Mice depleted with pericytes had a reduced LV ejection fraction (43,9 ± 0,7% vs. 77,6 ± 3,2 in control mice) and an increased end-diastolic pressure (11,12 ± 0,8282 mmHg vs. 5,730 ± 0,9524 in control mice) demonstrating systolic dysfunction. Accordingly, mice depleted with pericytes presented a decreased LV contractility (dP/dtm_{ax}) (3500 ± 387 mmHg/s vs. 5086 ± 157 in control mice) and an increased LV relaxation time (dP/dtm_{in}) (−2664 ± 360,3 mmHg/s vs. −4557 ± 192,9 in control mice). Morphologically, the heart of mice depleted with pericytes did not show hypertrophy, however, it presented signs of fibrosis with increased perivascular type I collagen levels and inflammation with an increased CD45 + cell infiltration and endothelial ICAM-1 expression.
Conclusion Cardiac pericyte depletion induces heart failure with reduced ejection fraction demonstrating that cardiac pericytes are critical for cardiac function. This result suggests that pericyte dysfunction could contribute to the onset of heart failure.
Disclosure of interest The authors declare that they have no competing interest.

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Cardiac performance in patients hospitalized with COVID-19: A 6-month follow-up study

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Introduction Myocardial injury is frequently observed in patients hospitalized with Coronavirus disease 2019 (COVID-19) pneumonia. Different cardiac abnormalities have been reported during the acute COVID-19 phase but there is limited information on late cardiac sequelae in patients who have recovered from acute COVID-19 illness.
Objective To document the presence and quantify the extent of myocardial functional alterations in patients hospitalized 6 months earlier for COVID-19 infection.
Methods and results We conducted a prospective echocardiographic evaluation of 48 patients (58 ± 13 years, 69% male) hospitalized 6 ± 1 month earlier for a laboratory-confirmed and symptomatic COVID-19. Thirty-two (66.6%) had pre-existing cardiovascular risks factors (hypertension, T2DM or dyslipidemia) and three patients (6.2%) had a known prior myocardial infarction. Sixteen patients (33.3%) experienced myocardial injury during the index COVID-19 hospitalization as identified by a rise in cardiac troponin levels. Six months later, 60.4% of patients still reported clinical symptoms including exercise dyspnea for 56%. Echocardiographic measurements under resting conditions were not different between patients with vs. without myocardial injury during the acute COVID-19 phase. In contrast, low-level exercise (25 W for 3 minutes) induced a significant increase in the average E/e' ratio (10.1 ± 4.3 vs. 7.3 ± 11.5, $P = 0.01$) and the systolic pulmonary artery pressure (33.4 ± 7.8 vs. 25.6 ± 5.3 mmHg, $P = 0.02$) in patients with myocardial injury during the acute COVID-19 phase. Sensitivity analyses showed that these alterations of left ventricular diastolic markers were observed regardless of cardiovascular disorders indicating SARS-CoV-2 infection as a primary cause.
Conclusions Six months after the acute COVID-19 phase, significant cardiac diastolic abnormalities are observed in patients who experienced myocardial injury but not in patients without cardiac involvement.
Disclosure of interest The authors declare that they have no competing interest.

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Fast Track hERG phenotyping to evaluate the pathogenicity of KCNH2 genetic variants

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Introduction Mutations in KCNH2 (coding for hERG) cause long or short QT syndromes (LQTS or SQTS), predisposing to life-threatening arrhythmias. More than 1000 variations in hERG sequences have been described, most of them are to be characterized.
Objective The objective is to standardize and accelerate the entire process necessary to phenotype hERG variants. An in silico evaluation was also included to characterize the structural impact of the variants.
Methods We selected 12 variants from patients with LQTS, and 1 with SQTS. We optimized the protocol to efficiently introduce mutations in hERG cDNA despite GC-rich sequences, using the Gibson assembly strategy. A pH-sensitive fluorescent tag was fused to hERG for fast-track evaluation of hERG cell trafficking. An optimized patch-clamp protocol of 35 sec was developed to evaluate hERG