prospective analysis to be performed to validate our study findings.

In conclusion, machine-learned models using novel DL techniques enable highly accurate automated diagnosis of acute brain infarction. These algorithms have the potential to assist radiologists while improving patient outcomes.

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https://doi.org/10.1016/j.jcmg.2018.03.012

Please note: This work was supported by a generous gift from the Dalio Institute of Cardiovascular Imaging and the Michael Wolk Foundation. Dr. Min serves on the scientific advisory board of Arineta; has ownership in MDDX; and has a research agreement with GE Healthcare. All other authors have reported that they have no relationships relevant to the contents of this paper to disclose. Sherif Nagueh, MD, served as the Guest Editor for this paper. Drs. Beecy and Chang contributed equally to this work and are joint first authors.

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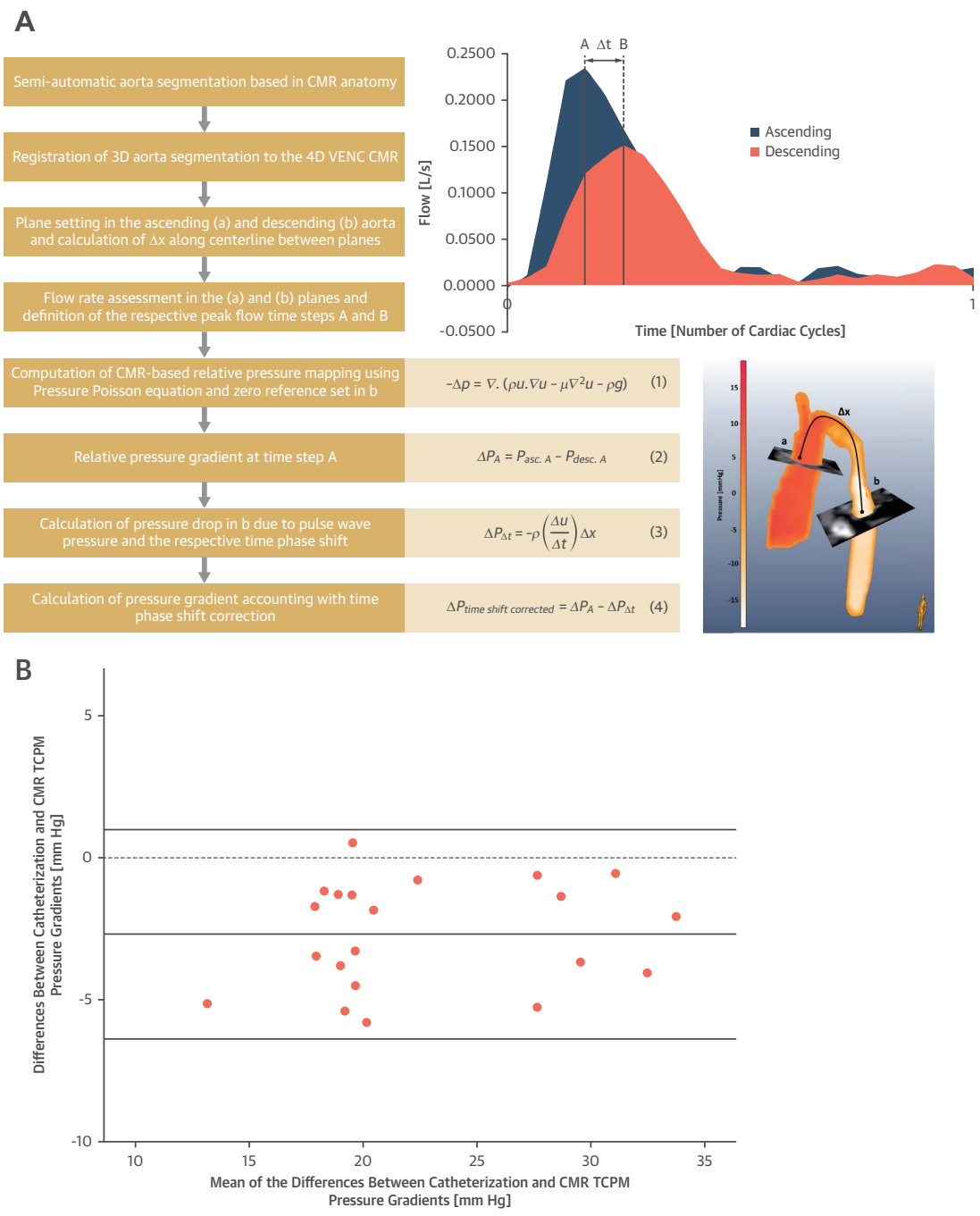
CMR-Based and Time-Shift Corrected Pressure Gradients Provide Good Agreement to Invasive Measurements in Aortic Coarctation

In aortic coarctation (CoA), clinical guidelines recommend treatment in the presence of a relevant pressure gradient (1). Although reliable noninvasive measurement approaches would be crucial, the accuracy of currently available methods is limited and cardiac catheterization has remained a clinical reference standard. Four-dimensional velocity-encoded cardiac magnetic resonance (CMR) was shown to be able to map relative pressures in a vessel (2,3). In contrast to invasive peak-to-peak measurements from heart catheterization, however, pressure mapping neglects the time-phase shift and thus adds bias. We aimed to validate a novel CMR-based time-shift corrected pressure mapping (TCPM) approach against catheterization and to compare this method against current noninvasive diagnostic standards, Doppler echocardiography, and cuff-based pressure differences.

We prospectively enrolled 21 patients with CoA (11 men, 10 women, mean age 23 ± 15 years) with clinical indication for cardiac catheterization due to relevant pressure gradients based on echocardiography and/or arterial hypertension. Our local ethical committee approved the study, and written informed consent was obtained from the participants or their guardians. CMR was performed before the catheterization procedure with a 5-element cardiac phased-array coil on a Philips Achieva R3.2.2 1.5-T scanner (Philips Medical System, Best, the Netherlands). During routine catheterization, pressure curve and resulting peak-to-peak gradients were recorded on a Schwarzer Haemodynamic Analyzing System (Heilbronn, Germany) in 2 predefined locations of the aorta. Previously described and clinically established 3- and 4-dimensional velocity encoded CMR sequences were used (3).

Figure 1A illustrates the post-processing and computation workflow to assess CMR-based TCPM. Semiautomatic aortic segmentation was performed with ZIBAmira (Zuse Institute, Berlin, Germany). Pressure mapping including antialiasing, registration, and flow analysis was done with MevisFlow (Fraunhofer MEVIS, Bremen, Germany). To generate relative pressure maps, pressure-Poisson equation was solved as detailed previously (2). CoA pressure differences were assessed at peak flow in the ascending and descending aorta and corrected to take into the account time-shift between pressure and volume flow in both vessels. Data were analyzed using SPSS version 21 (IBM Corporation, Armonk, New York). Bland-Altman plot and mean-equivalence paired *t*-test were performed (4). The null hypothesis in this test assumes measurements between methods differ considering a set clinical threshold of 5.0 mm Hg, corresponding to the variability of current clinical reference standards (invasive heart catheterization).

FIGURE 1 TCPM Implementation and Agreement With Catheterization



(A) Diagram illustrating TCPM, pressure mapping with catheter measurement points A and B and respective flow curves. **(B)** Bland-Altman analysis comparing catheterization peak-to-peak and TCPM pressure gradients. 3D = 3-dimensional; 4D = 4-dimensional; CMR = cardiac magnetic resonance; TCPM = time-shift corrected pressure mapping; VENC = velocity encoded.

There was a significant linear correlation between TCPM and invasive peak-to-peak pressure gradients ($R^2 = 0.90$; $p < 0.001$). Bland-Altman plots showed good agreement between TCPM (24.03 ± 5.70 mm Hg) and catheterization (21.34 ± 5.84 mm Hg). Mean differences were -2.69 mm Hg with 95% limits of agreement between -6.38 and 1.00 mm Hg (Figure 1B). Mean-equivalence *t*-test confirms agreement between

methods (p = 0.007). Without time-phase shift, correction mean differences were 6.3 with limits of agreement between -14.44 and 20.73 mm Hg.

Comparison between echocardiography and catheterization resulted in a lower coefficient of determination ($R^2 = 0.04$; p = 0.398) and significantly higher mean differences of 19.25 mm Hg without equivalence (p = 0.832). Blood pressure gradients also resulted in a low coefficient of correlation ($R^2 = 0.28$; p = 0.014) with mean differences of -8.44 mm Hg without equivalence (p = 0.197).

TCPM shows good agreement with heart catheterization. The results also confirm that arm-leg measurements tend to underestimate and echocardiography as well as uncorrected pressure mapping to overestimate pressure gradients.

The reliable use of pressure mapping and novel TCPM will depend on availability, resolution, and quality of CMR data. Patients underwent conscious sedation during catheterization, which is known to affect hemodynamics, whereas there was no sedation during CMR, echocardiography, and cuff measurements. Additionally, CMR and echocardiography were not performed simultaneously to catheterization. Nevertheless, the proposed CMR-based TCPM method provides significant equivalence with invasive heart catheterization in contrast to echocardiography and cuff measurements, in which data show large and relevant deviation.

At centers where 4-dimensional velocity-encoded CMR is available, it can already provide additional guidance before invasive procedures are performed. Beyond CoA, TCPM measurements are robust and carry the promising potential for other stenotic diseases, such as aortic valve disease or pulmonary artery stenosis, in which current noninvasive clinical techniques face similar diagnostic challenges. In CoA, TCPM may help to avoid invasive diagnostic procedures in the future. Clinical validation is the first step to bring modelling approaches to the bedside. Further steps of translation include randomized controlled and multicenter clinical trials in larger cohorts.

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https://doi.org/10.1016/j.jcmg.2018.03.013

Please note: The study was partially funded by the German Research Foundation (DFG, Grant GO1067/6-1-KU1329/10-1, Berlin, Germany) and by the European Commission under the ICT Program (Grant Agreement: 611232, Brussels, Belgium). Mr. Fernandes and Dr. Kuehne have filed a request for the grant of an academic patent of the presented method. All authors have reported that they have no relationships relevant to the contents of this paper to disclose. Mr. Fernandes and Ms. Alves contributed equally to this work and are joint first authors. (Proof of Concept of Model Based Cardiovascular Prediction; NCT02591940).

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Afterload Augmentation Can Reveal Concealed Myocardial Ischemic Memory

Imaging technology capable of detecting myocardial ischemic memory is desired because angina is often temporary. Post-systolic shortening (PSS) is myocardial shortening after aortic valve closure, which is easily measured via speckle-tracking echocardiography and is a known sensitive marker of acute ischemia (1). A relative decrease in regional contraction generates an imbalance of tension between the ischemic and surrounding myocardium, resulting in PSS. Assessment of PSS can be used to detect myocardial ischemic memory because it persists even after transient ischemia (2,3). However, its persistence is not always long enough, thus hampering its clinical application.

An increase of afterload affects myocardial deformation and makes PSS prominent (4). We therefore hypothesized that afterload augmentation could reveal concealed ischemic memory after transient ischemia and evaluated whether brief afterload augmentation allows the reappearance of PSS that had disappeared after transient ischemia.