

# Central blood pressure estimation in type 1 diabetes

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# Central blood pressure estimation in type 1 diabetes: impact and implications of peripheral calibration method.

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**Objective:** Peripheral blood pressure (BP) waveforms are used for noninvasive central BP estimation. Central BP could assist in cardiovascular risk assessment in patients with type 1 diabetes mellitus (T1DM). However, correct calibration of peripheral BP waveforms is important to accurately estimate central BP. We examined differences in central BP estimated by radial artery tonometry depending on which brachial BP (SBP/DBP vs. MAP/DBP) is used for calibration of the radial waveforms, for the first time in T1DM.

**Methods:** A cross-sectional study in T1DM patients without known cardiovascular disease. Radial artery BP waveforms were acquired using applanation tonometry (*SphygmoCor*) for the estimation of central SBP, central pulse pressure (PP) and central augmentation pressure, using either brachial SBP/DBP or MAP/DBP for the calibration of the radial pressure waveforms.

**Results:** Fifty-four patients (age:  $46 \pm 9.5$  years; T1DM duration:  $27 \pm 8.8$  years) were evaluated. Central BP parameters were significantly higher when brachial MAP/DBP-calibration was used compared with brachial SBP/DBP-calibration ( $7.5 \pm 5.04$ ,  $7.5 \pm 5.04$  and  $1.5 \pm 1.36$  mmHg higher central SBP, central PP and central augmentation pressure, respectively,  $P < 0.001$ ).

**Conclusion:** In patients with T1DM, there are significant differences in central BP values estimated with radial artery tonometry, depending on the method used for calibration of the radial waveforms. Brachial MAP/DBP-calibration resulted in consistently higher central BP as compared to using brachial SBP/DBP, leading to patient re-stratification. Hence, the accuracy of noninvasive estimation of central BP by radial tonometry is dependent on calibration approach, and this problem must be resolved in validation studies using an invasive reference standard to determine which method best estimates true central BP.

**Keywords:** arterial stiffness, calibration, central blood pressure, tonometry, type 1 diabetes

**Abbreviations:** AP, augmentation pressure; FF, form factor; GTF, generalized transfer function; PP, pulse pressure; T1DM, type 1 diabetes; T2DM, type 2 diabetes

## INTRODUCTION

Brachial SBP is insufficient to capture arterial stiffness and BP at the central level. Individuals with the same brachial SBP can have markedly different central SBP so that using brachial SBP alone could lead to under or overestimation of cardiovascular risk [1]. Although general clinical use is not yet recommended, there is an interest in central BP parameters potentially providing information on cardiovascular risk beyond conventional cuff-measured BP. Some studies have reported central haemodynamic parameters to be independent predictors of target organ damage [2] and cardiovascular events [3,4]. Evaluating central SBP might add value in cardiovascular risk assessment in certain populations with a high cardiovascular burden such as type 1 diabetes mellitus (T1DM). In these patients, central BP parameters measured noninvasively were found to be significantly different from nondiabetic controls [5–8] and independently associated with CVD [6,9].

Peripheral arterial waveforms can be used for noninvasive estimation of central BP by means of a mathematical transfer function [10]. However, correct estimation of central BP requires accurate scaling (calibration) of peripheral BP waveforms [10–12], for which different strategies exist. In a lot of studies, radial artery pressure waveforms were calibrated with brachial SBP/DBP, thereby ignoring potential brachial-to-radial SBP amplification [12,13]. Brachial SBP is not equal to radial SBP as shown by Armstrong *et al.* [14], who found a radial SBP of more than 5 mmHg higher than brachial SBP in most participants. Hence, using

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brachial SBP for calibration of radial artery waveforms could result in a substantial underestimation of local radial SBP, which can further propagate into an underestimation of central SBP [13,15].

Using brachial MAP/DBP is likely the preferred choice to calibrate peripheral waveforms, assuming DBP and MAP to be reasonably equal along the brachial-to-radial arterial path [11,13,16]. Previous studies have investigated the impact of calibration approach on central BP in several populations, concluding that central BP estimation is indeed calibration-dependent. MAP/DBP calibration yielded higher estimates of central BP than SBP/DBP calibration, and these differences might lead to different clinical interpretation of results [11,17–19]. However, difficulties exist to accurately estimate MAP noninvasively, as most commercial devices do not provide MAP derived from the peak oscillometric waveform, or the accuracy of the internal algorithms of these automated oscillometric devices is not reported [12,20]. Therefore, approximated values based on a mathematical formula, including SBP, DBP and a form factor are often used [21], with a form factor of 0.4 being advised instead of the 33% rule [17,22].

Although central BP can be prematurely increased in patients with T1DM and its accurate estimation could be of clinical importance, to our knowledge, there has never been a study to determine whether different calibration approaches could influence central BP estimation in this population. Therefore, this study aimed for the first time in patients with T1DM, to examine differences in noninvasively estimated central BP parameters by radial artery tonometry, depending on whether brachial SBP/DBP or MAP/DBP was used for calibration of radial pressure waveforms.

## MATERIALS AND METHODS

### Study design and participants

In this cross-sectional study, patients with T1DM attending the outpatient clinic were asked to participate as part of a larger cardiovascular screening programme, aimed to define cardiovascular risk and refine cardiovascular risk evaluation in T1DM, in a study investigating the role of arterial stiffness in cardiovascular risk estimation. Patients were recruited at the Endocrinology and Cardiology department (Ghent University Hospital, Belgium) between April 2019 and February 2021. Inclusion criteria were age more than 18 years, minimal T1DM duration of 10 years and absence of known CVD (i.e. no history of angina pectoris, acute coronary syndrome, stroke, symptomatic peripheral artery disease or any cardiovascular procedure). The rationale for including this particular target population was its relevance in primary CVD prevention, in whom the possible consequences of chronic glycemic exposure on the arterial wall and on the development of subclinical CVD, can be examined. The study was approved by the Ethics Committee of Ghent University Hospital and all patients provided written informed consent.

### Central blood pressure parameters

Radial artery BP waveforms were acquired using applanation tonometry with the SphygmoCor device (AtCor Medical, Sydney, Australia) according to the consensus

guidelines [23]. All patients were evaluated at the same time of day (0800 h) to minimize influence of diurnal variation in blood vessel tone, after 8-h overnight fasting and having abstained from vasoactive medication, caffeine, tea, polyphenol-rich foods, alcohol, nicotine and strenuous exercise in the 24 h prior to testing. Glycemia was monitored and tonometry was only performed if blood glucose was between 70 and 250 mg/dl (3.9–13.9 mmol/l). Measurements were performed in a quiet room after 10 min of supine rest, with patients not allowed to speak nor sleep, and all measurements were performed at the right side. First, brachial cuff SBP and DBP were measured three times by a validated oscillometric device (Omron IntelliSense 705IT; Omron Healthcare Europe B.V., Hoofddorp, The Netherlands) [24], with 2 min in between, and the mean value of the three measurements was used. As oscillometric MAP is not provided by the BP device, brachial MAP was calculated with a fixed form factor of 0.40 ( $\text{MAP} = \text{brachial DBP} + 0.40 \times \text{brachial PP}$ ) as previously recommended [17,22]. Next, at least three radial artery tonometric measurements were performed. Waveforms were processed with pulse wave analysis (PWA) and only those having an operator index more than 90% were accepted as valid, according to the quality criteria embedded in the device. A generalized transfer function (GTF) was used [10,25] to estimate the following central BP parameters: central SBP, central PP and central augmentation pressure. The mean value of the three measurements was used for analysis. Central BP parameters were calculated twice, first after radial artery BP waveforms being calibrated with the standard SphygmoCor procedure, that is, using brachial SBP/DBP [13], and a second time using brachial MAP/DBP calibration.

### Statistical analysis

Data were analysed with IBM SPSS Statistics 27.0 (IBM Corp., Armonk, New York, USA). Data were checked for normality with the Shapiro-Wilk test as well as visually by Q-Q plots and histograms, and shown as mean  $\pm$  SD or median [P<sub>25</sub>-P<sub>75</sub>] depending on the distribution. Paired *t*-tests were used to compare estimated central BP parameters between the two calibration methods. The *z*-test for two proportions was used to compare differences in the number of patients classified with central hypertension by the two calibration methods. Level of significance for all tests was *P* value less than 0.05.

## RESULTS

### Patient characteristics

Fifty-four patients with T1DM ( $n = 54$ ; 32 men, 22 women) were evaluated, aged  $46 \pm 9.5$  years (range: 26–68 years), with a T1DM disease duration of  $27 \pm 8.8$  years (range: 11–59 years), HbA1c of  $7.8 \pm 0.83\%$  and BMI value of  $25.4 \pm 3.88 \text{ kg/m}^2$ .

### Central blood pressure parameters

All three estimated central BP parameters (central SBP, PP and augmentation pressure) were significantly higher when brachial MAP/DBP was used compared with brachial SBP/DBP calibration, as summarized in Table 1 and Fig. 1 (a-c). Brachial cuff SBP was  $127 \pm 11.9 \text{ mmHg}$ , which was

**TABLE 1. Brachial and radial SBP and central blood pressure parameters estimated from differently calibrated radial artery pressure waveforms**

Parameter	MAP/DBP calibration [1]	SBP/DBP calibration [2]	Difference between two methods ([1,2])
Brachial SBP (mmHg)	127 ± 11.9	127 ± 11.9	NA
Radial SBP (mmHg)	137 ± 15.0 <sup>a</sup>	127 ± 11.9 <sup>b</sup>	10.1 ± 7.13 ( <i>P</i> < 0.001)
Central SBP (mmHg)	122.5 ± 13.41	115.0 ± 12.32	7.5 ± 5.04 ( <i>P</i> < 0.001)
Central PP (mmHg)	47.0 ± 11.51	39.4 ± 9.30	7.5 ± 5.04 ( <i>P</i> < 0.001)
Central AP (mmHg)	10.6 ± 6.96	9.2 ± 6.28	1.5 ± 1.36 ( <i>P</i> < 0.001)

Data presented as mean ± standard deviation. Brachial SBP measured with oscillometric cuff method (Omron); radial artery pressure waveform measured with tonometry (Sphygmocor). NA, not applicable.

<sup>a</sup>Radial SBP estimated by calibrating radial artery pressure waveform considering brachial and radial MAP and DBP identical.

<sup>b</sup>Radial SBP and DBP considered identical to brachial SBP and DBP as this information is entered in the Sphygmocor device as being radial pressure.

significantly lower than radial tonometry SBP (137 ± 15.0 mmHg) estimated using brachial MAP/DBP calibration, resulting in a derived brachial-to-radial SBP amplification of 10.1 ± 7.13 mmHg (*P* < 0.001) when using the latter method.

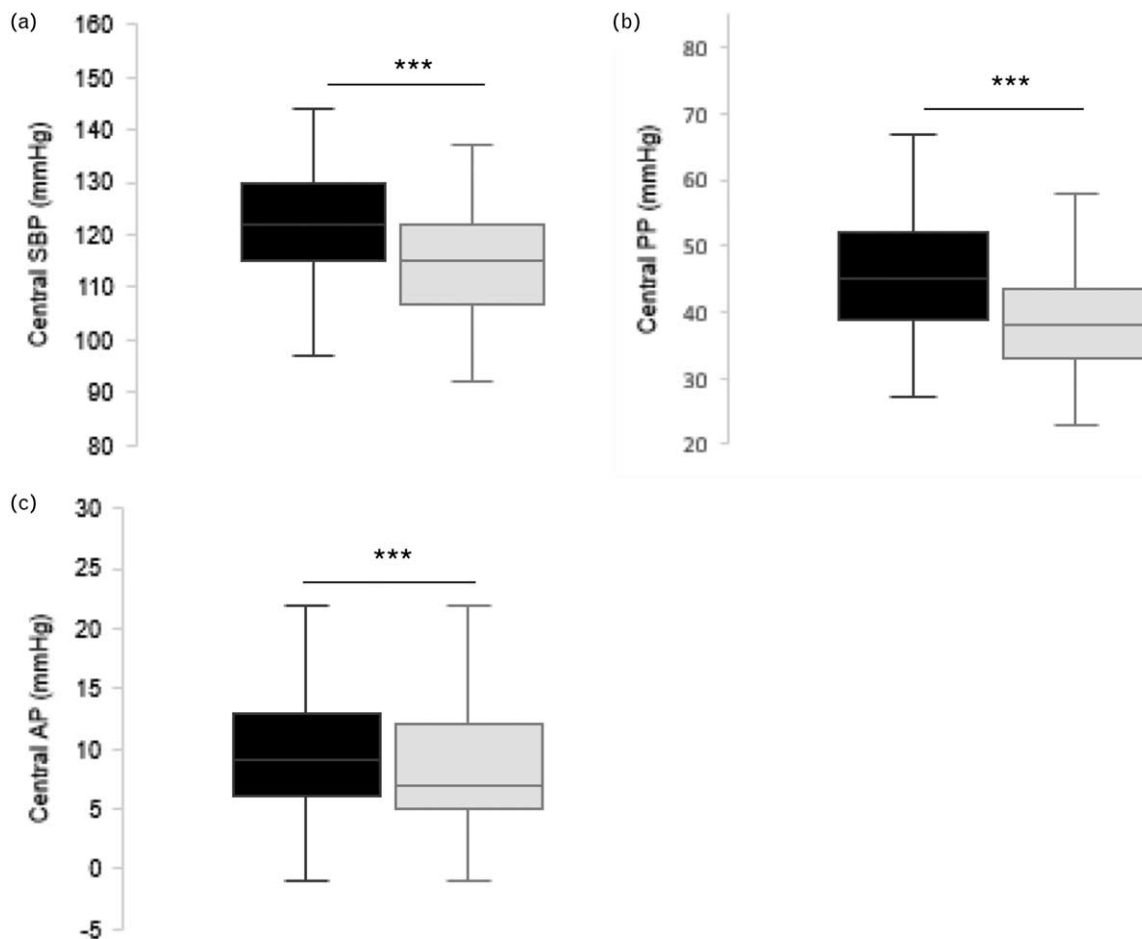
### Patient stratification based on central SBP

The proportion of patients with an estimated central SBP at least 130 mmHg (i.e. 'central hypertension') was significantly different between the calibration methods, with four vs. 13 patients using the SBP/DBP vs. MAP/DBP calibration method, respectively. A potentially 'misclassified group'

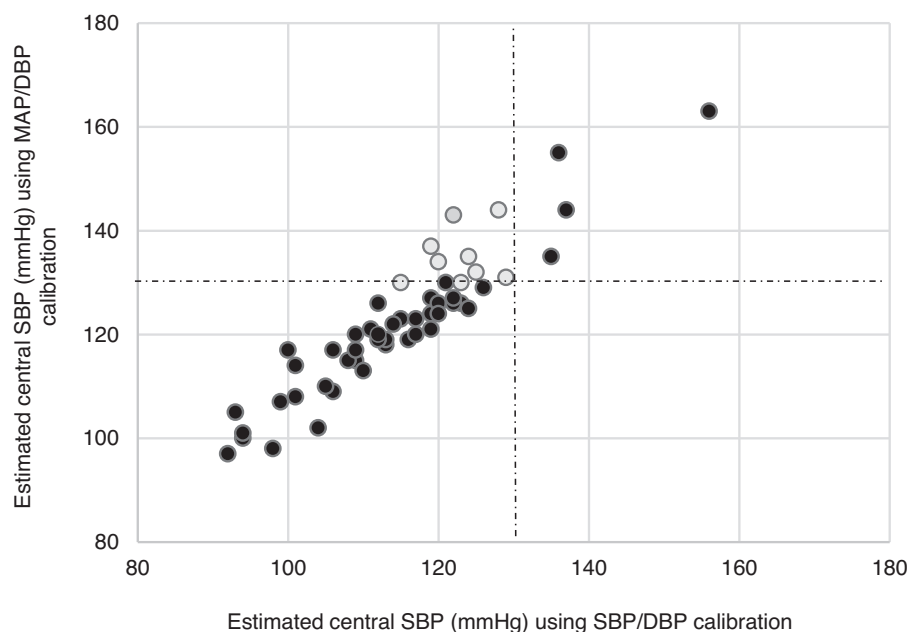
based on the SBP/DBP calibration method (*n* = 9, or 17% of included patients) is exemplified in Fig. 2.

### DISCUSSION

The present study aimed, for the first time in T1DM, to examine differences and the magnitude thereof in noninvasively estimated central BP parameters by radial artery tonometry depending on whether brachial SBP/DBP or MAP/DBP was used for calibration of the radial pressure waveforms. Our data show that also in patients with T1DM, there are significant differences in estimated central BP between the two calibration methods, with brachial



**FIGURE 1** Box and Whiskers plots for estimated central SBP (a), central pulse pressure (b) and central augmentation pressure (c) for the two calibration methods used. (Black: MAP/DBP calibration; Grey: SBP/DBP calibration; \*\*\**P* < 0.001, Whiskers indicate minimum and maximum values).



**FIGURE 2** Scatter plot of estimated central SBP for the two calibration methods. Horizontal and vertical 130 mmHg lines result in four quadrants, with the upper-left quadrant ( $n=9$ , as depicted in grey) showing a potentially misclassified group, being patients with a central SBP  $\geq 130$  mmHg when using brachial MAP/DBP for calibration of radial waveforms, however  $< 130$  mmHg using brachial SBP/DBP-calibration.

MAP/DBP calibration resulting in consistently higher central BP as compared to brachial SBP/DBP calibration, which may lead to patient re-stratification.

### Importance of peripheral waveform calibration in central blood pressure estimation

The meta-analysis of Papaioannou *et al.* [16] showed that using brachial MAP/DBP-calibration was superior compared with SBP/DBP-calibration for estimating aortic SBP. This was confirmed by the invasive study of Picone *et al.* [11], which moreover reported that the level of SBP amplification (aortic-to-brachial and/or brachial-to-radial) had a major impact on the estimated aortic SBP. Findings from a study including more than 1800 healthy individuals demonstrated that estimated SBP amplification over the brachial-to-radial path contributed substantially to the total SBP amplification between aorta and radial artery [26].

Our study now demonstrates that also in patients with T1DM, using brachial SBP/DBP-calibrated radial tonometry results in greater underestimation of central SBP compared with brachial MAP/DBP-calibration. The two methods yielded substantially different results, as we found a 7.8 mmHg higher estimated central SBP when using brachial MAP/DBP vs. SBP/DBP calibration, somewhat higher than previous studies in which MAP/DBP calibration led to higher central SBP estimates of 5.95 [16], 6.2 [11] and 2.5 mmHg [17], as compared to SBP/DBP calibration.

The SphygmoCor procedure uses a radial-to-aorta transfer function with calibration using brachial instead of radial pressure to estimate central BP [13]. However, this approach ignores brachial-to-radial SBP amplification, as the device assumes the entered values to be radial pressures, despite that these entered values are, in fact, *brachial* pressures. Alternative calibration of radial waveforms with radial instead of brachial pressures increased accuracy of

estimated central SBP, reducing the error between estimated and true aortic SBP from 7.1 to 3.0 mmHg [27]. As MAP and DBP are relatively constant along the large artery tree [21,28,29], hence also between brachial and radial artery [13], calibration of radial waveforms with brachial MAP/DBP should produce more accurate central BP estimations [10,12,30]. Another issue of *cuff* brachial SBP for waveform calibration is that this pressure underestimates intra-arterial brachial SBP, and when calibrating with this cuff-SBP (type I device), the derived central SBP is significantly below the true intra-arterial central SBP. Hence, even when using brachial tonometry and calibration with brachial cuff SBP, true brachial SBP is underestimated and therefore also true central SBP [16].

In conclusion, BP amplification and pulse wave calibration has implications for accurate BP measurement [31], and there is a need for a revised approach to calibrate with MAP/DBP (type II devices), or apply more sophisticated algorithms, to derive a central SBP more representative of true aortic SBP [12].

### Brachial-to-radial SBP amplification: main influential factor?

Although brachial-to-radial SBP amplification was considered a fictional phenomenon [32] – that is SBP amplification exclusively taking place between aorta and brachial artery, it has been shown that brachial-to-radial amplification does exist [13,14,26,30,33]. A brachial-to-radial amplification of 3.4 mmHg was found in the study by Asklepious *et al.* [17], and a more recent invasive study showed that radial SBP was on average 5.5 mmHg higher than brachial SBP, however with a large individual variation [14]. Our current findings suggest that brachial-to-radial SBP amplification is also present in patients with T1DM, with a rather large amplification of 10.1 mmHg as derived from estimated

radial tonometry SBP using brachial MAP/DBP calibration. Similar findings were reported by two noninvasive studies (amplifications between 9 and 14 mmHg) in healthy individuals and in T2DM, respectively [15,34]. Brachial-to-radial amplification depends on an individual's haemodynamic and (patho)physiological characteristics [26], seems to be higher in men [15,26] and may increase with age [15], as opposed to a decrease with age for SBP amplification between the aorta and brachial artery [35]. Closer consideration of SBP amplification or *individual* waveform characteristics that differ according to the individual level of amplification may improve accuracy of estimated aortic SBP [11].

### Mean arterial pressure: important to assess, difficult to estimate

Even when using brachial MAP/DBP calibration in central SBP estimation, there are concerns about how to best calculate MAP and which form factor to use [21,31]. In our study, a form factor of 0.40 was used, as this has been shown to yield an approximation of true intra-arterial MAP, the latter being underestimated using the one-third rule for MAP [22,36]. Especially when using brachial MAP to calibrate radial waveforms for application of a transfer function, it has been advised to use a form factor of 0.40 instead of 0.33 to estimate MAP [17,37]. Several other formulas for MAP-calculation have been proposed, with Papaioannou *et al.* [20] comparing six formulas with direct oscillometric MAP. However, this study did not aim to examine the *accuracy* of the formulas for true intra-arterial MAP estimation, but to identify the MAP most closely associated with target organ deterioration (TOD). It was found that MAP calculated with a form factor of 0.412 had superior predictive value for TOD. Future studies need to explore the accuracy of formulas for MAP estimation compared with direct intra-arterial BP measurement [20].

In our study, the large difference in estimated central SBP between the two calibration methods as well as high brachial-to-radial SBP amplification, could imply that the form factor is different in T1DM patients compared with nondiabetic individuals. To be as accurate as possible, form factors should differ between individuals – because of different waveform morphologies – as well as between measurement sites, that is form factors are neither uniform nor constant [21]. Accordingly, the use of fixed form factors was criticized after comparing invasive MAP calculated by waveform integration (i.e. reference MAP) with estimated MAPs calculated with form factors of 0.33 and 0.40. Estimating MAP via FFs led to 'nonphysiological and inaccurate values', mainly due to variable aortic-to-brachial SBP amplification [38]. Although an estimated MAP using a fixed form factor is often the best we can obtain noninvasively [17], more precise estimation methods would be appreciated [21].

### Clinical impact of different blood pressure calibration approaches

Comparing BP calibration approaches, oscillometric MAP/DBP-derived central SBP was a better predictor of cardiac structural abnormalities and mortality compared

with SBP/DBP-derived central SBP [18,39]. In our study, using a hypothetical aortic SBP threshold of 130 mmHg, 17% of the cohort was reclassified with central hypertension when using brachial MAP/DBP-calibration. A study in T2DM showed that when radial waveforms were calibrated using radial instead of brachial SBP, estimated central SBP was significantly higher with also an increase in the number of participants classified with central hypertension [34].

### Clinical use of central blood pressure: promising, (still) not proven

The clinical importance of central BP is less compelling than initially assumed, and it is yet to be proven that central SBP is superior to brachial SBP for predicting hard clinical outcomes [21]. The meta-analyses by Vlachopoulos *et al.* [3] and Kollias *et al.* [2] were important when first published (although central SBP was not prognostically superior for all outcomes), but have since been superseded by stronger analyses, indicating that there is no added prognostic value of central BP estimated by radial tonometry [40,41]. The main issue with brachial vs. GTF-based central SBP is their high correlation ( $r > 0.95$ ), so there is little extra value of central SBP as an independent, superior prognostic marker [21,40]. A recent prospective study found that central SBP was statistically but likely not *clinically* superior to brachial SBP. In that study, radial waveforms were calibrated using brachial SBP/DBP and not MAP/DBP [42]. It would be interesting to know whether secondary analyses, using MAP/DBP calibration, confer similar findings. Summarizing, taken all current data into account, clinically relevant improvement in cardiovascular risk stratification by using central BP beyond peripheral BP may be promising however still not proven [43]. Therefore, routine assessment of central SBP in clinical practice is not supported [43–45]. Future studies are needed to determine if guidance of cardiovascular risk management with central vs. conventional peripheral cuff BP can result in improved outcomes [43,46]. In patients with T1DM in particular, it remains to be investigated whether central BP could predict cardiovascular outcomes better than brachial BP [9]. Patients with T1DM often show early arterial stiffening and increased *peripheral* PP [47,48], progressing with longer disease duration [7,49] and associated with the development of CVD [47,50]. The prognostic significance of *central* SBP and *central* PP herein needs to be further examined in long-term follow-up studies.

In conclusion, the present study demonstrated for the first time in patients with T1DM that there are significant differences in central BP parameters estimated with radial artery tonometry, depending on the method used for calibration of the radial waveforms. Brachial MAP/DBP-calibration resulted in consistently higher central BP as compared to using brachial SBP/DBP, leading to patient re-stratification. Hence, the accuracy of noninvasive estimation of central BP by radial tonometry is dependent on the calibration approach, and this problem must be resolved in validation studies using an invasive reference standard to determine which method best estimates true central BP.

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S.H. was the primary person responsible for the conduct of the study and data analysis, and wrote, reviewed and edited the manuscript. T.D.B., J.S., B.S., P.S. and L.V.B. contributed to writing and editing the manuscript. B.S. contributed to data-analysis and making the figures. P.C. and B.L. reviewed the manuscript and contributed to the discussion. S.H. and T.D.B. are the guarantors of this work and, as such, had full access to all study data and take responsibility for the integrity of the data and accuracy of the data analysis. The authors thank Jos Op 't Roodt (research technician at Maastricht University Medical Centre, MUMC) for his help in pulse wave analysis and training thereof.

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## Conflicts of interest

There are no potential conflicts of interest relevant to this article to be disclosed.

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