Received: 15 August 2019 | Revised: 12 November 2019 | Accepted: 17 November 2019

DOI: 10.1111/jch.13758

REVIEW PAPER

WILEY

Central blood pressure for the management of hypertension: Is it a practical clinical tool in current practice?

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Abstract

Since noninvasive central blood pressure (BP) measuring devices are readily available, central BP has gained growing attention regarding its clinical application in the management of hypertension. The disagreement between central and peripheral BP has long been recognized. Some previous studies showed that noninvasive central BP may be better than the conventional brachial BP in association with target organ damages and long-term cardiovascular outcomes. Recent studies further suggest that the central BP strategy for confirming a diagnosis of hypertension may be more cost-effective than the conventional strategy, and guidance of hypertension management with central BP may result in less use of medications to achieve BP control. Despite the use of central BP being promising, more randomized controlled studies comparing central BP-guided therapeutic strategies with conventional care for cardiovascular events reduction are required because noninvasive central and brachial BP measures are conveniently available. In this brief review, the rationale supporting the utility of central BP in clinical practice and relating challenges are summarized.

1 | INTRODUCTION

To maintain the circulation of blood flow, the ejection of the stroke volume into the central aorta requires the pressure generated by left ventricle to overcome the pulsatile and resistive loads of the entire arterial tree.[1] As the pressure wave (PW) propagating along the arterial bed, it increases in the whole amplitude of the pulse pressure (PP) as it travels distally, that is, a "gradual widening" of the PP between two sites of the arterial bed. The increased amplitude of arterial pulse along the elastic and conduit arteries is quantified as the blood pressure (BP) amplification; that is, systolic BP (SBP) and PP are higher at peripheral arteries than that in the central aorta. Mean BP and diastolic BP remain almost unchanged (or slightly decreases because of viscous dissipation) between the two sites.[2] Although brachial BP has been routinely measured in daily practice, many studies have been conducted to address the prognostic and therapeutic impact of this noticeable discrepancy between brachial BP and central BP. Central BP, the pressure measured from the central aorta or common carotid arteries,[1] is determined by the interaction between function of left ventricle, large arteries and arterioles, and structure of aortic root, arterial bifurcations and arterial narrowing, and may therefore directly and better reflect the impact of pulsatile load.[3]

Central BP has gained growing attention concerning its clinical application in the management of hypertension since noninvasive central BP measuring devices are readily available. After the introduction of cuff-based techniques developed to obtain noninvasive central BP,[4-6] its convenient measurement can realize the use of central BP concept in daily clinical practice. Moreover, the Artery Society task force, in response to the burgeoning noninvasive central BP monitoring devices, has proposed a validation standard.[7] One of the major suggestions in the consensus statement is the further classification of central BP monitoring devices based on its purpose. It suggests to classify the devices into two types: Type I devices estimate central BP relative to the measured brachial BP, and type II devices estimate the intra-arterial central BP.[7] The features are a relatively accurate pressure difference between central and peripheral sites for type I devices and a relatively accurate absolute central BP value for type II devices.

BP measurements are conventionally obtained at the brachial arteries. Although brachial BP readings highly correlate with central BP and are the gold standard for the diagnosis and management of hypertension, substantial individual discrepancies between central and peripheral BP exist. Such discrepancies have long been a popular research topic, and whether central BP is a better clinical indicator than brachial BP has also been debated vehemently.[8,9] In this review, we will address briefly the rationale supporting the clinical use of central BP monitoring.[10]

2 | METHODS AND DEVICES USED FOR NONINVASIVE ESTIMATION OF CENTRAL BP

Pressure waveform of carotid artery is a good surrogate for central aortic pressure waveform.[11-13] However, the commonly used methodology utilizes waveforms obtained from peripheral arteries for noninvasive central BP estimation with either tonometry-based[11,14-16] or cuff-based techniques.[4-6] The common working principles of these central BP estimations include transfer function, pulse waveform analysis, and N-point moving average (NPMA). Transfer function is a mathematical relationship between two physical properties. The details of the measurement concept and procedures can be found in research performed with a commercial apparatus.[17,18] It has been the most popular central BP measurement device to date. Pulse waveform analysis can be used to identify waveform characteristics.

It has been shown that peak of SBP2, the late systolic should of a pressure waveform resulting from distal PW reflections agrees well with the peak of central aortic pressure waveforms (central SBP).[19,20] Besides, using comprehensive waveform analysis including SBP2 and corresponding regression equations, central SBP and PP can be accurately estimated.[4,21] Recently, it has been demonstrated that one can use NPMA method to estimate central aortic SBP (SBP-C).[16] NPMA is a mathematical low-pass filter that is frequently used in the engineering field for removing random noise from a time series by using a common denominator related to the sampling frequency. The high-frequency components, which cause substantial transformations from central to peripheral aortic pressure waveforms resulting primarily from arterial wave reflections,[22] can be eliminated by the application of the NPMA.[16,23] Table 1 summarizes current available devices for measuring central BP.

It is suggested that the accuracy of central BP should be examined against the invasive measurements counterparts.[7] Accuracy of current central BP methods and devices has been investigated in several systematic review.[24-26] It seems that the accuracy is device specific,[27] and the major limitation is the accuracy of cuff BP used for waveform calibration.[24,28]

3 | UTILITY OF CENTRAL BP MONITORING IN CLINICAL PRACTICE

3.1 | Peripherally obtained BP does not accurately reflect central pressure because of pressure amplification

As shown in a previous study, a large proportion of subjects with high-normal brachial SBP had comparable central SBP as those with stage 1 hypertension.[29] This discrepancy was also noted for subjects with normal brachial BP, many of whose central BPs were in the same category as those with stage 1 hypertension. If central BP is a better target for therapy, the misclassification by brachial BP may lead to over- or under-treatment of hypertension and may be clinically relevant.[30] The diagnosis of hypertension, according to either office, home, or ambulatory BP measurements, is currently based on recordings from the brachial arteries. Because of the phenomenon of PP amplification, brachial SBP and PP are usually higher than the corresponding readings in the central aorta.[2,15,31-33] However, either by the auscultatory method or automatic oscillometric sphygmomanometers, the noninvasively measured brachial SBP and PP, are usually lower than the invasively measured intra-arterial readings.[34] As a consequence, noninvasive brachial SBP readings may approach to values of central SBP[35]; therefore, it might be reasonable to use noninvasive brachial SBP as an estimate of central SBP. Nonetheless, robust evidence suggests that there are substantial disagreements of central BP among individuals with similar brachial BP.[36,37] Moreover, although the averaged invasive central SBP is similar to averaged brachial cuff SBP, there is substantial variability, that is, under and over estimation of central SBP by the cuff SBP, which refutes brachial cuff SBP being an accurate representation of central SBP.[26,37,38] The PP amplification, the disagreement between central and peripheral BP varies within- and between-individuals.[39] More importantly, such variability depends on a number of factors, including age, sex, body height, heart rate, medications, and systemic vascular diseases.[36,40,41] Besides, noninvasive brachial SBP as a surrogate for central SBP has been shown to have a large random error.[37]

3.2 | Central aortic pressure is a better predictor of cardiovascular outcome than peripheral pressure

Central BP may reflect the pulsatile load on the heart and large arteries better than brachial BP, particularly in individuals with a prominent PP amplification.[3] It has been demonstrated that central SBP was more closely associated with left ventricular mass index, carotid intima-media thickness, and pulse wave velocity, compared with brachial SBP,[42,43] whereas brachial SBP might be superior to central SBP in identifying albuminuria in patients with type 2 diabetes.[44] In addition, longitudinal studies further support that the changes of central BP rather than brachial BP related to the regression of left ventricular mass index and carotid intima-media thickness,[45,46] and microalbuminuria and cognitive aging.[43,47]

In a systematic review of 85 studies, central compared with brachial BP seems to be more strongly associated with most of the investigated indices of preclinical organ damage.[42] With regard to the relationship between central BP and cardiovascular outcomes, we previously showed that central SBP and PP were more predictive of cardiovascular mortality than brachial SBP and PP in a Taiwanese cohort.[48] In addition, central SBP and PP were significantly associated with cardiovascular events, as well as brachial measurements, in a meta-analysis of 11 studies with 5648 subjects,[49] while the superiority of central over brachial measurements was marginal (nonsignificant) for central PP and nonapparent for central SBP. The cohort studies investigating the prognostic role of central BP have been summarized in Table 2. Recently, the clinical benefits of different antihypertensive agents observed in the ASCOT study were more associated with the reduction of central rather than brachial BP,[17] which ignited the application of central BP for clinical practice.[50] If precision of central BP measurement could be improved, as for some type II central BP devices, we may see more substantial prognostic difference.

3.3 | Antihypertensive medications have differential effects on central pressures despite similar reductions in brachial BP

It has long been recognized that individual discrepancies between central and peripheral BP may be magnified during hemodynamic changes or after pharmacological interventions.[24] The differential responses of central BP vs brachial BP to various antihypertensive

TABLE 1 Summary of devices capable of measuring central blood pressure

Device company	Site of record	Method of waveform recording (sensor)	Method of estimation calibration	Calibration	Invasive validation/ FDA approval
Office central BP monitoring					
PulsePen DiaTecne srl., Italy	Carotid artery	Applanation tonometry Single, manual	Simple substitution	Brachial cuff MAP/DBP	Yes/No
Complior Alam Medical, France	Carotid artery	Applanation tonometry, Single, fixed	Simple substitution	Brachial cuff MAP/DBP	Yes/No
NIHem Cardiovascular Engineering Inc, USA	Carotid artery	Applanation tonometry, Single, manual	Simple substitution	Brachial cuff MAP/DBP	Yes/No
HEM-9000AI Omron Healthcare, Japan	Radial artery	Applanation tonometry Arrayed, fixed	SBP2 + regression	Brachial cuff SBP/DBP	Yes/No
GaonHanbyul Meditech, Korea	Radial artery	Applanation tonometry Single, fixed	GTF	Brachial cuff SBP/DBP	Yes/Yes
SphygmoCorCVMS, AtCor Medical, Australia	Radial artery	Applanation tonometry Single, manual	GTF	Brachial cuff SBP/DBP	Yes/Yes
SphygmoCor XCELAtCor Medical, Australia	Brachial artery	Subdiastolic brachial cuff plethysmography	GTF	Brachial cuff SBP/DBP	Yes/Yes
Oscar 2 with SphygmoCor SunTech Medical, USABrachial	Brachial artery	Subdiastolic brachial cuff plethysmography	GTF	Brachial cuff SBP/DBP	Yes/Yes
cBP301Centron Diagnostics, UK (acquired bySunTech Medical)	Brachial artery	Brachial cuff plethysmography	GTF	Brachial cuff SBP/DBP	Yes/Yes
Mobil-O-GraphI.EM GmbH, GermanyBrachialartery	Brachial artery	Brachial cuff pulse volume plethysmography	GTF	Brachial cuff SBP/DBP	Yes/Yes
ArteriographTensioMed Ltd., Hungary	Brachial artery	Supra-systolic brachial cuff plethysmography	SBP2 + regression	Brachial cuff MAP/DBP	Yes/No
Vicorder Skidmore Medical Ltd., UK	Brachial artery	Brachial cuff pulse volume plethysmography	GTF	Brachial cuff MAP/DBP	Yes/Yes
BPLab Petr Telegin, Russia	Brachial artery	Brachial cuff pulse volume plethysmography	GTF	Brachial cuff SBP/DBP	Yes/Yes
BP + Uscom Ltd., Australia (acquire Pulsecor Ltd., Cardioscope II)	Brachial artery	Supra-systolic brachial cuff plethysmography	Physical model Brachial supra-systolic waveform	Brachial cuff SBP/DBP	Yes/No
DynaPulse Pulse Metric Inc, USA	Brachial artery	Supra-systolic brachial cuff plethysmography	Physical model	Brachial cuff SBP/DBP	Yes/Yes
WatchBP Microlife Corp, Taiwan	Brachial artery	Brachial cuff pulse volume plethysmography	(SBP2, DBP, As, Ad) + regression	Brachial cuff SBP/DBP	Yes/Yes
ARCsolver + VaSeraVS-1500Austrian Institute of Technology, Austria	Brachial artery	Brachial cuff pulse volume plethysmography	GTF	Brachial cuff SBP/DBP	Yes/Yes
Ambulatory Central BP Monitors					
BPro + A-Pulse, HealthSTATS, Singapore (acquired by Hillrom)	Radial artery	Applanation tonometry Single, fixed (watch type)	N-point moving average	Brachial cuff SBP/DBP	Yes/Yes
Mobil-O-Graph NGI.EM GmbH, Germany	Brachial artery	Brachial cuff pulse volume plethysmography	GTF	Brachial cuff SBP/DBP	Yes/Yes
Arteriograph 24 h, TensioMED Ltd., Hungary	Brachial artery	Supra-systolic brachial cuff plethysmography	SBP2 + regression	Brachial cuff MAP/DBP	Yes/No
ABPM 7100Welch Allyn, Inc (acquired by Hillrom)	Brachial artery	Brachial cuff pulse volume plethysmography	GTF	Brachial cuff SBP/DBP	No/No
WatchBP O3 (2G), Microlife AG, Widnau, Switzerland	Brachial artery	Brachial cuff pulse volume plethysmography	(SBP2, DBP, As, Ad) + regression	Brachial cuff SBP/DBP	Yes/Yes
Oscar 2 with SphygmoCor, SunTech Medical	Brachial artery	Subdiastolic brachial cuff plethysmography	GTF	Brachial cuff SBP/DBP	Yes/Yes

Abbreviations: Ad, area under pressure wave curve during diastole; As, area under PW curve during systole; DBP, diastolic blood pressure; GTF, generalized transfer function; MAP, mean arterial blood pressure; SBP, systolic blood pressure; SBP2, second peak of radial or brachial PW.

agents are highly variable among individuals in clinical studies.[51,52] It has been suggested that angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, and dihydropyridine calcium channel blockers, as well as nitrates, may have a more beneficial effect on central BP than beta-blockers, despite their similar effects on brachial BP.[53,54] Randomized controlled trials investigating the differential response between central and peripheral BP to different classes of pharmacological interventions have been summarized in Table 3.

Similarly, various classes of antihypertensive drugs may exert different effects on the PP amplification. Compared with diuretics and beta-blockers, angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, dihydropyridine calcium blockers, and nitrates may exert a favorable effect on the PP amplification.[3,30,55,56] The observed less beneficial effect of beta-blockers (mainly atenolol) on cardiovascular outcomes[57] could be explained by the unfavorable effect on the PP amplification.[17,53] These speculations were supported by the CAFE substudy of the ASCOT trial,[17] and the J-Core study.[58]

Although we have previously proposed the central BP threshold of 130/90 mm Hg for the diagnosis of hypertension,[33] the treatment targets in patients with elevated central BP have yet to be defined. Previous studies have shown that guidance of hypertension management with central BP may result in less use of medications to achieve BP control without adverse effects.[30] A recent randomized controlled trial demonstrated that maximization of goal-directed medical therapy in heart failure patients could be more achieved by using central BP, as compared with conventional office brachial BP, during additional medicine titration, which subsequently enhanced afterload reduction and lead to reverse remodeling without increased risk of hypotension or worsening renal function.[59]

In uncomplicated hypertensive subjects with low to medium risk, it is reasonable to lower central BP to <130/90 mm Hg. However, outcome-driven central BP-guided treatment target studies should be conducted for other specific compelling disease status.

3.4 | Isolated central hypertension and isolated brachial hypertension are associated with increased cardiovascular risks

The discrepancy between central and brachial BP could be used to define phenotypes of hypertension.[60,61] Based on the ESH/ESC hypertension guidelines for brachial hypertension (brachial SBP ≥ 140 mm Hg or brachial DBP ≥ 90 mm Hg or using antihypertensive medicine) and central hypertension criteria[33] (≥130 mm Hg for central SBP or ≥ 90 mm Hg for central DBP or using antihypertensive medicine), in a national representative cohort, phenotypes of isolated central and isolated brachial hypertension among adults have been identified (Figure 1).[62] Subjects with isolated central hypertension had a significantly higher estimated 10-year coronary heart disease risk than those without central or brachial hypertension.[62] In the Northern Shanghai study, elderly Chinese subjects with isolated central hypertension had higher levels of left ventricular mass index, carotid-formal pulse wave velocity, and urinary albumin-creatinine ratio than those without central or brachial hypertension.[63] Moreover, based on the 2017 ACC/AHA hypertension threshold, a higher proportion of subjects with isolated brachial hypertension thresholds (130/80 mm Hg) has been identified (Figure 1).[64] Subjects with isolated brachial hypertension had an increased risk of coronary heart disease similar to those with isolated central hypertension and were characterized by young age, male sex, and a high prevalence of isolated diastolic hypertension, implying minimal evidence of the presence of arterial stiffness or vascular aging.[64]

4 | FUTURE PERSPECTIVES ON THE USE OF CENTRAL BP TO MANAGE HYPERTENSION

We have evidently shown in a previous systematic review that current central BP estimating methods are theoretically suitable.[24] However, the major errors of these central BP measurement techniques result from the inaccurate noninvasive BP used to calibrate the peripheral waveforms. In a recently published systematic review, cuff BP has variable accuracy for measuring either brachial or aortic intra-arterial BP, which adversely influences correct BP classification[26] and inevitably makes waveform calibration inadequate. Therefore, the measurement accuracy of both noninvasive brachial and central BP still has room for improvement.[26,65] Recently, World Hypertension League, International Society of Hypertension, and other supporting hypertension organizations have together issued a position statement to call for regulating manufacture and marketing of BP devices and cuffs.[66] With the joint efforts, validated automatic BP devices are more readily available and more accurate noninvasive brachial and central BP measurements could be rendered in the care of cardiovascular patients.

Despite that central BP may be better than brachial BP in predicting cardiovascular outcomes,[5,20,22] it is arguable that the inconsistent superiority of central BP over brachial BP may reflect a true pathophysiological issue or is potentially biased by methodological weakness.[67,68] It should be acknowledged that most outcome studies were conducted in the elderly in whom brachial and central pressures are similar, and no outcome studies have been conducted in younger patients, in whom a much greater difference between brachial and central pressures is expected. Convincing evidence has shown that different antihypertensive treatments can differentially reduce central vs brachial BP; however, studies investigating whether such therapeutic difference could be translated into clinical outcomes are required. Future prospective studies are needed to demonstrate the superiority of the central BP-guided strategy over conventional brachial BP strategy in hypertension screening in the community or management at clinical practice.[69] Even central BP may have superior advantages over

TABLE 2 Overview of studies on the association between central pressures and augmentation index and clinical endpoints sorted by date of publication

Study	Population-sample size	Age (y)	Men (%)	Follow-up duration	Events	Index	Modality	Attrition bias (loss/events ratio)	Index modeled	Adjusted for
Lu et al[70]	Stable CAD/angioplasty (n = 87)	72.5 ± 5.1	92	6.1 ± 4.1 m	39 cases of restenosis	Aortic PP; Aortic PP/MAP; Aortic PP/DBP	Invasive (fluid-filled system, 7F pig-tail catheter)	Not reported	Continuous; optimal cut-off by ROC curve	Risk factors for restenosis (not specified)
London et al[71]	ESRD (n = 180)	54 ± 16	60	52 ± 36 m	70 deaths; 40 CV deaths	Carotid AIx	Tonometry of CCA	0%	Continuous; quartiles; optimal cut-off by ROC curve	Age, sex, DBP, HR, smoking, duration of dialysis, blood chemistry analyses, ACEI prescription, PWV
Safar et al[72]	ESRD (n = 180)	54 ± 16	60	52 ± 36 m	70 deaths; 40 CV deaths	Carotid SBP, PP, brachial-carotid PP amplification	Tonometry of CCA	0%	Continuous; quartiles; optimal cut-off by ROC curve	Age, sex, DBP, HR, smoking, duration of dialysis, blood chemistry analyses, ACEI prescription, PWV
Ueda et al[73]	CAD/angioplasty (n = 103)	62 ± 9 78	78	6 m	36 cases of restenosis	Aortic AIx; aortic inflection time	Invasive (fluid-filled system, 5F pig-tail catheter)	0%	Continuous; tertiles	Age, sex, smoking, hypertension, diabetes, hypercholesterolemia, stent size, HR, inflection time
Chirinos et al[74]	CAD or nonobstructive coronary atherosclerosis (n = 297)	63.8 ± 10.3	100	40 ± 14 m	58 deaths; 128 CV events	Aortic PP, AP, AIx	Invasive (low-compliance fluid-filled system)	11%	Continuous	Age, diastolic or MAP, diabetes, smoking, HR, height, use of drugs, lipid levels, ejection fraction, C-reactive protein, extent of CAD
Weber et al[75]	CAD/angioplasty (n = 262)	65 ± 10	71	24 m	12 deaths; 61 CV events	HR-corrected aortic AIx	Tonometry of radial artery, GTF	1.60%	Continuous; tertiles	Age, sex, smoking, prior MI or stroke, diabetes, peripheral artery disease, extent of CAD, medications, triglycerides, creatinine clearance, BMI, SBP, or PP
Dart et al[76]	Elderly female hypertensives (n = 484)	72 ± 5	0	49 m (median)	53 CV events	Carotid SBP, PP; AIx	Tonometry of CCA	Not reported	Dichotomous	Age, cholesterol, smoking
Covic et al[77]	ESRD (n = 92)	42.6 ± 11.2	54	61 ± 25 m	15 deaths	HR-corrected aortic AIx	Tonometry of radial artery, GTF	Not reported	Tertiles	Age, sex, time on dialysis, SBP, PP, LVMI, use of ACE inhibitors
Roman et al[18]	American Indians free of CVD (n = 2403)	63.5 ± 7.5	35	58 ± 16 m	386 deaths; 67 CV deaths; 319 CV events	Aortic SBP, PP	Tonometry of RA—transfer function	0.80%	Continuous	Age, sex, current smoking, BMI, total/HDL ratio, creatinine, fibrinogen, diabetes, HR

(Continues)

TABLE 2 (Continued)

Study	Population-sample size	Age (y)	Men (%)	Follow-up duration	Events	Index	Modality	Attrition bias (loss/events ratio)	Index modeled	Adjusted for
Jankowski et al[78]	Subjects undergoing nonemergency coronary angiography (n = 1109)	52.7 ± 19.2	74	52.7 ± 19.2 m	90 deaths; 71 CV deaths; 246 CV events	Aortic PP; PPf	Invasive (low-compliance fluid-filled system)	15%	Continuous; quartiles	Age, sex, ejection fraction, mean coronary artery stenosis, heart failure, HR, risk factors, CVD, eGFR, drugs
Pini et al 2E + 07	Community-dwelling individuals 65 y (n = 398)	73 ± 6	45	94 ± 24 m	106 deaths; 45 CV deaths; 122 CV events	Aortic SBP, PP, AIx	Tonometry of CCA	Not reported	Continuous	Age, sex
Wang et al[48]	normotensive and untreated hypertensive (n = 1272)	M: 52.4 ± 12.9 F: 52.0 ± 12.7	53	10 y	130 died, 37 CV deaths	central and brachial SBP and PP	sequential nondirectional Doppler (Parks model 802; Parks Medical Electronics, Aloha, Oregon, USA)	Not reported	Continuous	Age, sex, heart rate, BMI, current smoking, fasting plasma glucose levels, cholesterol/HDL ratio, PWV, LVM, IMT, and eGFR
Chirinos. et al[79]	White, African American, Hispanic, or Chinese and who were free of clinically apparent CV disease (n = 5960)	62 (53-70)	48	7.61 y	407 first CVE 281 first hard CVE 117 first episode of CHF	Reflection magnitude AIx PP amplification	tonometry device–GTF noninvasively	5%	Continuous	*Adjusted model 1* age, gender, total cholesterol, HDL-cholesterol, smoking, SBP, DBP, diabetes mellitus *Adjusted model 2* further adjusts for ethnicity, body height, body weight, antihypertensive medication use, HR, and eGFR
Wassertheurer et al[80]	patients with CKD stages 2-4 (n = 159)	59.9 ± 15.2	55	42 mo (range 30-50 mo	13 patients died nine CV deaths	brachial SBP aortic SBP	oscillometric method (Mobil-OGraph PWA monitor; IEM, Stolberg, Delaware, USA)	Not reported	Continuous	Age, sex, and anthropometric measures

Abbreviations: ACEI, angiotensin-converting enzyme inhibitor; AIx, augmentation index; BMI, body mass index; CAD, coronary artery disease; CCA, common carotid artery; CKD, chronic kidney disease; CHF, congestive heart failure; CV, cardiovascular; CVE, cardiovascular event; DBP, diastolic blood pressure; eGFR, estimated glomerular filtration rate; ESRD, end-stage renal disease; GTF, generalized transfer function; HDL, high-density lipoprotein; HR, heart rate; IMT, intima-media thickness; LVM, left ventricular mass; MAP, mean arterial pressure; MI, myocardial infarction; PI, pulsatility index (pulse pressure/DBP); PP, pulse pressure; PPf, fractional pulse pressure (pulse pressure/mean pressure); PWV, pulse wave velocity; ROC, receiver operating characteristic curve; SBP, systolic blood pressure

TABLE 3 Randomized controlled trials investigating the differential response of central vs brachial blood pressure to antihypertensive agents

Study	Drug class or placebo studied	Sample size	Type of study	Site of artery	Design	Comments
Guerin 1992[81]	BB vs CCB	20	Drug comparison	Carotid	Parallel	
London 1994[82]	CCB vs ACEI	24	Drug	Carotid	Parallel	End-stage renal disease
Chen 1995[83]	ACEI vs BB	79	Drug comparison	Carotid	Parallel	cSBP not reported
Mahmud 2000[84]	ARB	18	Drug comparison	Radial	Parallel	
Asmar 2001b[85]	Placebo vs ARB	27	Placebo controlled, drug comparison	Radial	Crossover	Hypertension + Diabetes
Asmar 2001a[86]	ACEI + D vs BB	471	Drug comparison, combination	Carotid	Parallel	
Mitchell 2002[87]	ACEI vs omapatrilat	167	Drug comparison	Carotid	Parallel	
Deary 2002[88]	Placebo vs ACEI vs CCB vs ACEI vs BB vs AB vs D	30	Placebo controlled, drug comparison	Radial	Crossover	Separate data for sexes
de Luca 2004[89]	ACEI + D vs BB	146	Drug comparisons	Radial	Parallel	Essential hypertension
Neal 2004[90]	CCB vs ACEI vs BB	24	Drug comparison	Radial	Crossover	Liver transplantation
London 2004[91]	ACEI + D vs BB	181	Drug comparison, combination	Carotid	Parallel	
Morgan 2004[92]	Placebo vs CCB vs ACEI vs BB vs D;	321	Placebo controlled, drug comparison	Radial	Crossover	
Mahmud 2005[93]	D vs spironolactone	24	Drug comparison	Radial	Crossover	
Dart 2007[94]	ACEI vs D	479	Drug comparison	Carotid	Parallel	
Jiang 2007[95]	ACEI vs D	101	Drug comparison	Radial	Parallel	
Williams 2006[17]	BB + D vs ACEI + CCB	2199	Combination	Radial	Parallel	
Dhakam 2006[96]	BB vs ARB	21	Drug comparison	Radial	Crossover	
Schneider 2008[97]	ARB vs BB	156	Drug comparison	Radial	Parallel	
Dhakam 2008[98]	Placebo vs BB vs nebivolol	16	Placebo controlled, drug comparison	Radial	Crossover	
Mahmud 2008[99]	BB vs nebivolol	40	Drug comparison	Radial	parallel	
Matsui 2009[58]	ARB + CCB vs ARB + D	207	Drug comparison	Radial	Parallel	
Mackenzie 2009[100]	CCB vs ACEI vs BB vs D	59	Drug comparison	Radial	Parallel	Isolated systolic hypertension
Boutouyrie 2010[101]	ARB + CCB vs ARB + BB	393	Combination	Radial	Parallel	
Dol 2010[102]	CCB vs D	37	Drug comparison	Radial	Parallel	
Kaufman 2010[103]	losartan 100 mg, isosorbide mononitrate (ISMN) 60 mg, losartan 100 mg + ISMN 15 mg, losartan 100 mg + ISMN 60 mg, and placebo	13	Double-blind, crossover study	Radial	Crossover	Essential hypertension
Cockburn 2010[104]	propranolol 80 mg vs bisoprolol 20 mg vs placebo	20	Double-blind, crossover study	Finger	Crossover	
Manisty 2010[105]	amlodipine with perindopril vs atenolol with bendroflumethiazide-K	259	Prospective, randomised, open-label, blinded endpoint parallel group	Carotid	Parallel	Essential hypertension
Ferdinand 2011[51]	Aliskiren + D vs CCB	53	Drug comparison, combination	Carotid	Parallel	African American
Takenaka 2012[106]	benidipine vs amlodipine	67	Open-label, parallel group, randomized, controlled study	Radial	Parallel	Chronic kidney disease

(Continues)

TABLE 3 (Continued)

Study	Drug class or placebo studied	Sample size	Type of study	Site of artery	Design	Comments
Virdis 2012[107]	aliskiren (150-300 mg/daily) or ramipril (5-10 mg/daily)	50	Drug comparison	Radial	Parallel	Essential hypertension
Izzo 2012[108]	carvedilol vs valsartan	30	Forced-titration, random order-entry crossover study	Radial	Crossover	Essential hypertension
Vitale 2012[109]	BB + D vs ARB + D	65	Drug comparison	Radial	Parallel	Essential hypertension
Takami 2012[110]	Azelnidipine (16 mg daily) + D vs amlodipine (5 mg daily) (25 patients/group) + D	50	Prospective, randomized, open-label parallel group	Radial	Parallel	Essential hypertension
Ruilope 2013[111]	ARB + CCB vs ACEI + CCB	486	Parallel group, noninferiority study,	Radial	Parallel	Hypertensive subjects
Fogari 2013[112]	imidapril vs ramipril (R)	176	Prospective, randomised, open-label, blinded endpoint parallel group	Radial	Parallel	Diabetic hypertensive patients with microalbuminuria
Koumaras 2013[113]	quinapril vs aliskiren vs atenolol vs nebivolol	72	Drug comparison	Radial	Parallel	Treatment-naive, adult patients with uncomplicated, stage I-II, essential hypertension
Eeftinck Schattenkerk 2013[114]	nebivolol/hydrochlorothiazide vs metoprolol/hydrochlorothiazide	22	Randomized, double-blind	Radial	crossover	Aged 40-70 y, with untreated stage 2 hypertension
Radchenko 2013[115]	ARB + D vs BB + D	59	Drug comparison	Radial	Parallel	Moderate-to-severe hypertension
Ihm 2013[116]	CCB vs ARB	200	Drug comparison	Radial	Parallel	Mild to moderate essential hypertensives.
Agnoletti 2013[117]	amlodipine 5mg, or candesartan 8mg, or indapamide sustained-release 1.5mg, in comparison with placebo.	145	Drug comparison	Radial	Parallel	BP ≥ 150 to < 180 mm Hg and DBP of ≥ 95 to < 110≥160 to < 180 mm Hg and DBP of < 90 mm Hg
Takami 2013[118]	Azelnidipine plus olmesartan vs amlodipine plus olmesartan	52	Prospective, randomized, open-label parallel group study	Radial	Parallel	Patients with SBP ≥ 140 mm Hg and/or diastolic BP ≥ 90 mm Hg
Hare 2013[119]	spironolactone 25mg daily (n = 58) or placebo (n = 57)	115	Randomized, double-blind, placebo-controlled	Radial	Parallel	Hypertensive response to exercise
Park 2013[120]	Bisoprolol vs Atenolol	209	Prospective, randomized, open-label, active-controlled trial	Radial	Parallel	
Dorresteijn 2013[121]	aliskiren 300 mg vs sympathoinhibition (using moxonidine 0.4 mg) vs D (using hydrochlorothiazide 25 mg) vs placebo	31	Four-way, double-blind, single-center, crossover study	Radial	Crossover	Obesity-related hypertension
Matthesen 2013[122]	placebo vs amiloride vs spironolactone	23	Drug comparison	Radial	Parallel	Essential hypertension
Parati 2013[123]	acetazolamide 250 mg b.i.d. or placebo	42	Drug comparison	Radial	Parallel	Healthy lowlanders without known cardiovascular disease

(Continues)

TABLE 3 (Continued)

Study	Drug class or placebo studied	Sample size	Type of study	Site of artery	Design	Comments
Park 2014[124]	ARB + CCB vs maximal ARB vs Maximal CCB	391	Open-label, randomized, active-controlled	Radial	Parallel	Aged 20-70 y with grade 2 or grade 3 hypertension
Dillinger 2015[125]	Ivabradine vs Placebo	12	Randomized, double-blind	Radial	Crossover	Normotensive subjects with CAD
Bruno 2015[126]	ACEI + CCB vs ACEI + Diuretics	76	Randomized, open labeled	Radial	Parallel	Hypertensive subjects with metabolic syndrome
Metoki 2015[127]	Maximal ARB vs ARB + Diuretics	200	Drug comparison, combination	Radial	Parallel	Essential hypertension aged from 20 to 80 y
Redon 2016[128]	ACEI + CCB vs ARB + CCB	88	Noninferiority, randomized, double-blind, double-dummy parallel group, controlled design trial,	Radial	Parallel	After Missed Dose in Type 2 Diabetes.
Rimoldi 2016[129]	Ivabradine vs Placebo	46	Single-blinded fashion	Invasive central BP	Parallel	Chronic stable coronary artery disease
Rosenbaek 2017[130]	Placebo or diluted NaNO2 in three different doses	12	Placebo controlled, dose-response	Radial	Crossover	Healthy volunteer
Suojanen 2017[131]	Placebo vs BB	16	Double-blind, randomized, placebo-controlled trial	Radial	Crossover	never-treated 16 Caucasian males with grade I-II primary hypertension
Schreglmann 2017[132]	Pyridostigmine bromide vs fludrocortisone	13	Double-blind, randomized, active-control, crossover,	Radial	Crossover	Parkinson's disease with orthostatic hypotension
Fraig 2018[133]	fixed-dose combination of amlodipine 10 mg/valsartan 160 mg vs nebivolol 5 mg/valsartan 160 mg	137	Drug comparison, combination	Brachial cuff pulse volume plethysmography	Parallel	Grade 2 or more hypertensive patients
Rosenbaek 2018[134]	Placebo, allopurinol 150 mg twice daily (TD), enalapril 5 mg TD, or acetazolamide 250 mg TD	16	Placebo controlled, drug comparison,	Radial	Crossover	Healthy volunteer
Georgianou 2019[135]	nebivolol (5 mg/d), olmesartan (20 mg/d), or no-treatment	60	Single-blinded fashion	Brachial cuff pulse volume plethysmography	Parallel	Acute phase of ischemic stroke

Abbreviations: AB, alpha-blocker; ACEI, angiotensin-converting enzyme inhibitor, ARB, angiotensin receptor blocker, BB, beta-blockers, CCB, calcium channel blocker; D, diuretics.

conventional office BP, its current utility is still mainly restricted in research field. Using central BP in the assessment of cardiovascular health, such as predicting one's cardiovascular risk or diagnosing whether he or she has hypertension can be implemented soon if more convincing evidence can be accumulated. However, using central BP to guide clinical practice is a more difficult case given that even home BP or ambulatory BP are not often used as a therapeutic guidance tool. The management of hypertension is largely based on BP obtained from conventional office BP over decades. More randomized controlled trials demonstrating that controlling both brachial and central hypertension can bring benefits to patients may be the first step of the application of central BP in routine clinical practice. Other barriers from knowledge, attitudes, and external factors such as guideline and reimbursement

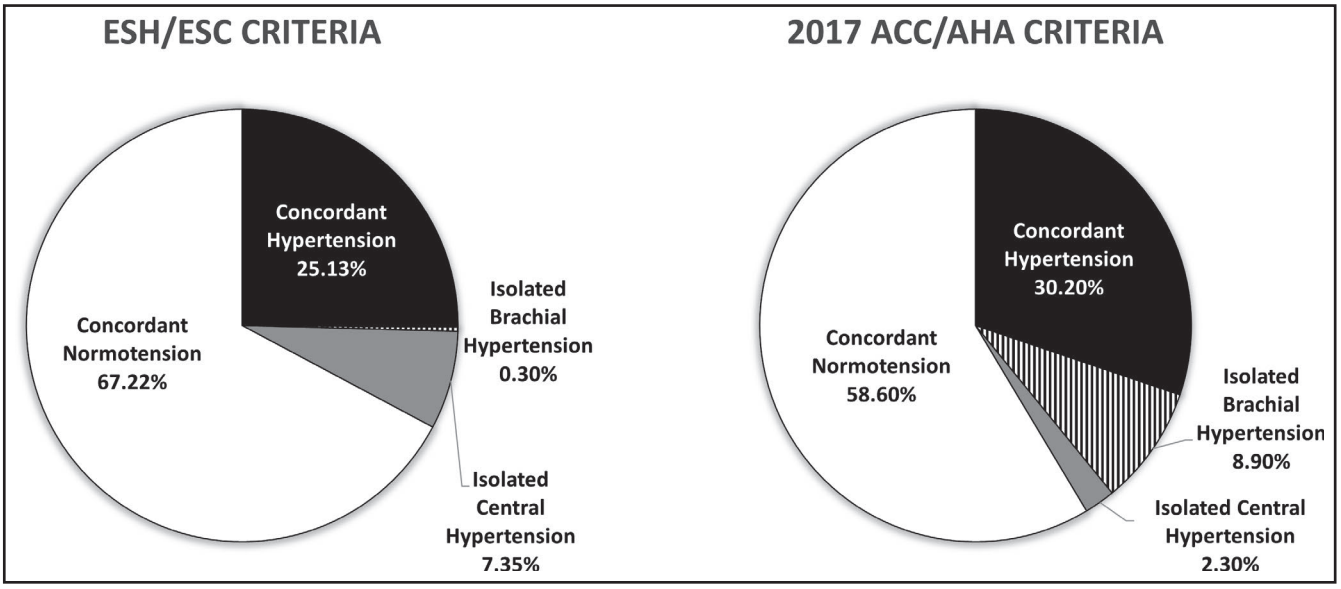

FIGURE 1 The national weighted proportion of concordant hypertension, concordant normotension, isolated brachial hypertension, and isolated central hypertension, according to the criteria of brachial hypertension with ESH/ESC (140/90 mm Hg) and 2017 ACC/AHA (130/80 mm Hg) thresholds. Central hypertension was defined by central blood pressure ≥130/90 mm Hg or using antihypertensive medicine

issue should be dealt with to facilitate the translational process afterward. Longitudinal follow-up studies are also required to prove that isolated brachial hypertension and isolated central hypertension, the two new hypertension phenotypes identified by the simultaneously obtained noninvasive central and brachial BP measures, indeed carry increased cardiovascular risks and deserve respective management.

5 | CONCLUSIONS

In this brief review, we summarized the rationale supporting the clinical utility of central BP since it can be conveniently measured noninvasively. Noninvasive central BP is likely better than the conventional brachial BP in association with target organ damages and long-term cardiovascular outcomes, but more evidence is required to support the use of central BP in diagnosing hypertension and monitoring the management of hypertension with central BP in routine clinical practice.

CONFLICT OF INTEREST

K Kario received research grants from Omron Healthcare, A&D, and Fukuda Denshi Co. Ltd. S Park has received research grants and honoraria from Pfizer. S Siddique has received honoraria from Bayer, Novartis, Pfizer, ICI, and Servier; and travel, accommodation, and conference registration support from Atco Pharmaceutical, Highnoon Laboratories, Horizon Pharma, ICI, Pfizer, and CCL. YC Chia has received honoraria and sponsorship to attend conferences and CME seminars from Abbott, Bayer, Boehringer Ingelheim, GlaxoSmithKline, Menarini, Merck Sharp & Dohme, Novartis, Orient Europharma, Pfizer, and Sanofi; and a research grant from Pfizer. J Shin has received honoraria and sponsorship to attend seminars from Daiichi Sankyo, Takeda, Menarini, MSD, Bristol-Myers Squibb, and Sanofi. CH Chen has served as an advisor or consultant for Novartis Pharmaceuticals Corporation; has served as a speaker or a member of a speakers bureau for AstraZeneca; Pfizer Inc; Bayer AG; Bristol-Myers Squibb Company; Boehringer Ingelheim Pharmaceuticals, Inc; Daiichi Sankyo, Inc; Novartis Pharmaceuticals Corporation; SERVIER; Merck & Co., Inc; Sanofi; TAKEDA Pharmaceuticals International; and has received grants for clinical research from Microlife Co., Ltd. R Divinagracia has received honoraria as a member of speaker's bureaus for Bayer, Novartis, and Pfizer. J Sison has received honoraria from Pfizer, AstraZeneca, Boehringer Ingelheim, and Novartis. GP Sogunuru has received a research grant related to hypertension monitoring and treatment from Pfizer. JC Tay has received advisory board and consultant honoraria from Pfizer. BW TEO has received honoraria for lectures and consulting fees from Astellas, AstraZeneca, Boehringer Ingelheim, Servier, MSD, and Novartis. JG Wang has received research grants from Bayer, Merck Sharp & Dohme, Pfizer, and Phillips; and lecture and consulting fees from Bayer, Daiichi-Sankyo, Merck Sharp & Dohme, Pfizer, Servier, and Takeda. Y Zhang has received research grants from Bayer, Novartis, and Shuanghe; and lecture fees from Bayer, Daiichi Sankyo, Novartis, Pfizer, Sanofi, Servier, and Takeda. All other authors report no potential conflicts of interest in relation to this article.

AUTHOR CONTRIBUTIONS

Scott Solomon, John McMurray, Milton Packer, Jean Rouleau, Michael Zile, Marc Hao-Hao-Min Cheng, Shao-Yuan Chuang contributed to the first draft of the article. Tzung-Dau Wang, Kazuomi Kario, PeeraBuranakitjaroen, Yook-Chin Chia, Romeo Divinagracia, Satoshi Hoshide, Huynh Van Minh, Jennifer Nailes, Sungha Park,

Jinho Shin, Saulat Siddique, Jorge Sison, Arieska Ann Soenarta, Guru Prasad Sogunuru, ApichardSukonthasarn, Jam Chin Tay, Boon Wee Teo, YudaTurana, Narsingh Verma, Yuqing Zhang, Ji-Guang Wang, and Chen-Huan Chen critically reviewed each draft and provided substantial input on the content.

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How to cite this article: Cheng H-M, Chuang S-Y, Wang T-D, et al. Central blood pressure for the management of hypertension: Is it a practical clinical tool in current practice?. *J Clin Hypertens*. 2020;22:391–406. https://doi.org/10.1111/jch.13758