Medical Policy

Blue Cross Blue Shield Blue Care Network of Michigan

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***Current Policy Effective Date: 11/1/24**
(See policy history boxes for previous effective dates)

Title: Noninvasive Measurement of Central Blood Pressure (e.g., SphygmoCor® System, Vicorder® Pulse Wave Velocity, PhysioWave)

Description/Background

Cardiovascular diseases (CVD), including coronary artery disease, stroke and hypertension, are the leading causes of morbidity and mortality in the United States. Vascular disease is the major contributor to cardiovascular morbid events and ideally is identified early, before symptoms are detected or irreversible damage has occurred. Arterial compliance (elasticity), carotid intima-media thickness (CIMT) and advanced lipoprotein analysis are tests used to measure and monitor atherosclerosis.

Conventionally, assessments of endothelial function and arterial stiffness require different sets of equipment, making the inclusion of both tests impractical for clinical and epidemiological studies. Pulse wave analysis (PWA) is a simple and noninvasive technique that may provide useful information regarding the mechanical properties of the arterial tree and the ventricular-vascular interaction.

The SphygmoCor® family of products provides tools for non-invasive assessment of the cardiovascular system, focused on central blood pressures, measures of arterial stiffness and autonomic function. The technology that powers these products is centered on an algorithm that derives the pressure wave at the ascending aorta from an external measurement taken at the radial artery. The SphygmoCor® System may allow non-invasive measurement of the pressure the heart, brain and kidneys experience. The technology behind these products is said to be centered on an algorithm that derives the pressure wave at the ascending aorta from an external measurement taken at the radial artery.

Regulatory Status

K080670: Modification to the SphygmoCor Cardiovascular Management System (AtCor Medical) cleared on April 23, 2008
K070795: SphygmoCor Cardiovascular Management System (AtCor Medical Pty Ltd.) cleared on August 31, 2007
K012487: SphygmoCor Px, Model SCOR-PX (AtCor Medical Pty Ltd.) cleared on February 21, 2002.
K002742: SphygmoCor MX, Model SCOR-MX (PWV Medical Pty. Ltd.) cleared on May 1, 2001

The most recent 510(k) clearance (**K122129**) for the SphygmoCor XCEL device was issued to AtCor Medical on November 16, 2012. According to the clearance documents: "…**Device Description**: The SphygmoCor3 XCEL System is indicated to perform non-invasive cardiovascular measurements as an adjunct to manage various cardiovascular conditions. The device can be used in any of 2 modes:

- **Pulse Wave Analysis Measurement (PWA or CP)** - A brachial cuff is used to measure the peripheral blood pressure and arterial pulses to derive the central blood pressure waveform and corresponding parameters. The brachial blood pressure measurement is calculated using the oscillometric technique, This feature is implemented essentially by a 3rd party NIBP Module (SunTech Medical Advantage Mini OEM BP module).
- **Pulse Wave Velocity Measurement (PWV)** - Using a non-invasive Tonometer pressure sensor and Cuff, this mode measures the time difference between the Carotid and Femoral arterial pulses measured simultaneously. To determine the carotid to femoral pulse wave velocity, the distance measured between the two arterial sites is divided by measured time difference. This Pulse Wave Velocity (PWV) is an indicator of arterial stiffness. Higher PWV's are associated with increased arterial stiffness.

PhysioWave Cardiovascular Analyzer
K172431: PhysioWave Cardiovascular Analyzer cleared in March 2018. The PhysioWave is intended to obtain pulse wave velocity (PWV) and pulse rate through a combination of impedance plethysmography and weight measurements in adults 18 years of age and older. The PhysioWave also measures body weight and calculates BMI.

Medical Policy Statement

This is not an established or medically necessary procedure. While noninvasive measurement of central blood pressure may be safe, its effectiveness in this clinical indication has not been scientifically determined. Therefore, this service is experimental/investigational.

Inclusionary and Exclusionary Guidelines

N/A

CPT/HCPCS Level II Codes *(Note: The inclusion of a code in this list is not a guarantee of coverage. Please refer to the medical policy statement to determine the status of a given procedure.)*

Established codes:

N/A

Other codes (investigational, not medically necessary, etc.):

93050

Rationale

In 2006, the Conduit Artery Function Evaluation (CAFE) study, a substudy of the Anglo-Scandinavian Cardiac Outcomes Trial (ASCOT), examined the impact of 2 different BP lowering-regimens (atenolol+/-thiazide-based versus amlodipine+/-perindopril-based therapy) on derived central aortic pressures and hemodynamics.[2] The CAFE study recruited 2199 patients in 5 ASCOT centers. Radial artery applanation tonometry and pulse wave analysis (Sphygmocor) were used to derive central aortic pressures and hemodynamic indexes on repeated visits for up to 4 years. Most patients received combination therapy throughout the study. Despite similar brachial systolic BPs between treatment groups (Delta0.7 mm Hg; 95% CI, -0.4 to 1.7; P=0.2), there were substantial reductions in central aortic pressures with the amlodipine regimen (central aortic systolic BP, Delta 4.3 mm Hg; 95% CI, 3.3 to 5.4; P<.0001; central aortic pulse pressure, Delta 3.0 mm Hg; 95% CI, 2.1 to 3.9; P<.0001). Cox proportional-hazards modeling showed that central pulse pressure was significantly associated with a post hoc-defined composite outcome of total cardiovascular events/procedures and development of renal impairment in the CAFE cohort (unadjusted, P<.0001; adjusted for baseline variables, P<.05). The investigators concluded that BP-lowering drugs can have substantially different effects on central aortic pressures and hemodynamics despite a similar impact on brachial BP. Moreover, central aortic pulse pressure may be a determinant of clinical outcomes, and differences in central aortic pressures may be a potential mechanism to explain the different clinical outcomes between the 2 BP treatment arms in ASCOT. An accompanying editorial[3] stated that, "in the context of clinical trials, radial tonometry adds to our knowledge of the pharmacodynamic effects of vasoactive drugs… whether radial tonometry should be performed routinely in individual patients as a diagnostic or therapeutic indicator, however, remains a matter of considerable debate. At present, the technique is probably not quite ready for 'prime time' in routine clinical practice."

Weber et al (2011) reported that increased arterial wave reflections are independent predictors of renal as well as cardiorenal events in patients with chronic kidney disease.[4] The investigators prospectively quantified wave reflections as pressure augmentation (AP) and augmentation index (AIx) using radial applanation tonometry and a transfer function, in 111 patients (mean age 53.6 years; 71 men, 31 diabetics) with chronic kidney disease not requiring dialysis. Primary endpoint was a composite of doubling of serum creatinine, need for dialysis,

and transplantation. Secondary endpoint was a combination of renal and cardiovascular events. After a mean follow-up of 41.3 months, 37 and 46 patients reached the primary and the secondary endpoint. AIx and AP proved statistically significant predictors of the renal endpoint ($p<.05$ for all), with a 2.5- and 3-fold increased risk for patients in the highest vs. the lowest tertile, respectively. After adjustment for mean blood pressure (MBP), age, gender, diabetes, serum albumin, hemoglobin, urine albumin/creatinine ratio, and renal function at baseline, AIx (hazard ratio 1.474/10% increase in AIx, P=.04) as well as AP (hazard ratio 1.559/10 mm Hg increase in AP, p=.04) remained significant predictors of the renal endpoint. In addition, AIx and AP were significant ($p<.05$) predictors of the combined cardiorenal endpoint in univariate analysis and multivariable models. A commentary noted that pulse wave velocity was not measured in this study. The commentator concluded: "Where do we go from here? The authors note that AIx may be a suitable target for intervention, and indeed propose that a trial be undertaken to determine whether therapeutically targeting wave reflections independent of blood pressure reduction would lessen CKD progression. This may also partly address the unanswered question of causality. Unfortunately, we still lack any suitable pharmacological interventions to achieve this, as current options for lowering AIx all have significant blood pressure lowering effects. Furthermore, given the inextricable relationship between AIx and blood pressure, targeting one of these factors and not the other is perhaps inappropriate, and indeed the current findings may support the use of antihypertensive agents which also reduce AIx. Regardless of the therapeutic intervention employed, measuring AIx may facilitate earlier and more aggressive targeting of at-risk patients, and larger studies are certainly justified to explore the prognostic value of AIx in more detail."

Laugesen et al (2014) noted that the SphygmoCor is used for non-invasive assessment of ascending aortic BP.[5] However, the validity of the SphygmoCor transfer function had not been tested in an exclusively type 2 diabetic patient sample. Calibration with systolic (SBP) and diastolic (DBP) brachial BP has previously been associated with substantial imprecision of central BP estimates. These investigators hypothesized that different non-invasive calibration strategies might improve the accuracy of the estimated ascending aortic BPs. In 34 patients with type 2 diabetes, these researchers estimated ascending aortic SBP and DBP using the SphygmoCor device and compared these data with invasively recorded data. The validity of the transfer function was assessed by calibrating with invasively recorded DBP and mean BP (MBP). The influence of non-invasive calibration strategies was assessed by calibrating with brachial oscillometric SBP+DBP versus DBP+MBP using a form factor (ff) of 0.33 and 0.40, respectively. When calibrating with invasive BP, the difference between estimated and invasively measured ascending aortic SBP and DBP was -2.3 ± 5.6/1.0 ± 0.9 mm Hg. When calibrating with oscillometric brachial BPs, the differences were -9.6 ± 8.1/14.1 ± 6.2 mm Hg (calibration with SBP and DBP), -8.3 ± 11.7/13.9 ± 6.1 mm Hg (DBP and MBP; ff = 0.33), and 1.9 ± 12.2/14.1 ± 6.2 mm Hg (DBP and MBP; ff = 0.40), respectively. Calibration with the average of 3 brachial BPs did not improve accuracy. The authors concluded that the SphygmoCor transfer function seems valid in patients with type 2 diabetes. They stated that non-invasive calibration with DBP and MBP (ff = 0.40) enables accurate estimation of mean ascending aortic SBP at the group level; however, the wide limits of agreement indicate limited accuracy in the individual patient.

Yule et al (2016) utilized the SphygmoCor non-invasive assessment tools to monitor the short-term impact of whole-body vibration (WBV) on arterial stiffness in chronic stroke. Stoke has been associated with increased arterial stiffness, which can be diminished through physical activity.[11] However, stroke patients are not always able to engage in programmed physical activity. The investigators conducted a 6-week trial in which 6 chronic stroke patients engaged in escalating WBV treatment. Treatment was administered 3 times per week for 4 weeks with a 2-week washout period. Arterial stiffness decreased over time for WBV. In contrast, arterial stiffness increased over time in the control group, but the interaction effect was not significant (p=.166). For the distensibility coefficient (DC) there was no significant interaction effect (p = 0.124) or main effect between the WBV and control groups (p=.431). Similarly, there was no significant interaction effect (p=.237) or main effect for compliance coefficient (CC) between the WBV and control groups (p=.496). The researchers reported no significant findings in this study although a non-significant decrease in carotid arterial stiffness was detected post-WBV compared to pre- WBV.

Sharman et al (2013) sought to determine the usefulness of central BP to guide hypertension management.[6] The investigators conducted a prospective, open-label, blinded–end point study in 286 patients with hypertension randomized to treatment decisions guided by best-practice usual care (n=142; using office, home, and 24-hour ambulatory BP) or, in addition, by central BP intervention (n=144; using SphygmoCor). Therapy was reviewed every 3 months for 12 months, and recommendations were provided to each patient and his/her doctor on antihypertensive medication titration. Outcome measures were as follows: medication quantity (daily-defined dose), quality of life, and left ventricular mass (3-dimensional echocardiography). There was 92% compliance with recommendations on medication titration, and quality of life improved in both groups (post hoc p<.05). For usual care, there was no change in daily defined dose (all p>.10), but with intervention there was a significant stepwise decrease in daily defined dose from baseline to 3 months (p=.008) and each subsequent visit (all p<.001). Intervention was associated with cessation of medication in 23 (16%) patients versus 3 (2%) in usual care (p<.001). Despite this, there were no differences between groups in left ventricular mass index, 24-hour ambulatory BP, home systolic BP, or aortic stiffness (all p>0.05). The investigators concluded that guidance of hypertension management with central BP results in a significantly different therapeutic pathway than conventional cuff BP, with less use of medication to achieve BP control and no adverse effects on left ventricular mass, aortic stiffness, or quality of life. An accompanying editorial by Avolio commented that "there is still insufficient evidence for central aortic BP to be integrated in guidelines for treatment and management of hypertension."[7] The editorialist stated that trials are required to assess hard end points, where subjects are followed up for a longer period (of the order of 5 years, similar to many other intervention studies). In addition, the design should be expanded where central aortic BP is measured in all subjects, but where one group is assessed by measurements of brachial cuff pressure but blind to the results of central BP and the other groups guided by the results of central BP but blind to the results of brachial BP.

Methods employed for pulse wave analysis (PWA) and peripheral blood pressure (PBP) calibration vary. The purpose of this study was to evaluate the agreement of SphygmoCor PWA parameters derived from radial artery tonometry when considering (1) timing (before vs. after tonometry) and side selection (ipsilateral vs. contralateral limb) for PBP calibration and (2) side selection for tonometry (left vs. right arm). In 34 subjects (aged 21.9 ± 2.3 years), bilateral radial artery tonometry was performed simultaneously on three instances. PBP assessment via oscillometric sphygmomanometry in the left arm only and both arms simultaneously occurred following the first and second instances of tonometry, respectively. Significant within arm differences in PWA parameters derived before and after PBP measurement were observed in the right arm only (for example, aortic systolic blood pressure, Δ=0.38 ± 0.64 mm Hg). Simultaneously captured bilateral PWA variables demonstrated significant between arm differences in 88% (14/16) and 56% (9/16) of outcome variables when calibrated to within arm and equivalent PBP, respectively. Moreover, the right arm consistently demonstrated lower values for clinical PWA variables (for example, augmentation index, bias=-2.79%). However, 26% (n=9) of participants presented with clinically significant differences (>10 mm Hg) in bilateral PBP and their exclusion from analysis abolished most between arm differences observed. SphygmoCor PWA in the right radial artery results in greater variability independent of the timing of PBP measurement and magnitude of calibration pressures in young subjects. Moreover, bilateral PBP measurement is imperative to identify subjects in whom a significant difference in bilateral PWA outcomes may exist.

Omboni et al (2015) compared central BP and vascular indices estimated non-invasively over the 24 hours between normotensive volunteers and hypertensive patients by a pulse wave analysis of ambulatory BP recordings.[8] Digitalized waveforms obtained during each brachial oscillometric BP measurement were stored in the device memory and analyzed by the validated Vasotens technology. Averages for the 24 hours and for the awake and asleep sub-periods were computed. A total of 142 normotensives and 661 hypertensives were evaluated. Overall, 24-hour central BP, pulse wave velocity (PWV), and aortic augmentation index (AI) were significantly higher in the hypertensive group than in the normotensive group (119.3 versus 105.6 mm Hg for SBP, 75.6 versus 72.3 mm Hg for DBP, 10.3 versus 10.0 m/sec for aortic PWV, -9.7 versus -40.7 % for peripheral AI, and 24.7 versus 11.0 % for aortic AI), whereas reflected wave transit time (RWTT) was significantly lower in hypertensive patients (126.6 versus 139.0 ms). After adjusting for confounding factors a statistically significant between-group difference was still observed for central BP, RWTT, and peripheral AI. All estimates displayed a typical circadian rhythm. The authors concluded that non-invasive assessment of 24-hour arterial stiffness and central hemodynamics in daily life dynamic conditions may help in assessing the arterial function impairment in hypertensive patients.

Xiao et al (2015) examined the differences in central hemodynamic indices between hypertensive and normotensive subjects and identified the BP index that the most strongly correlated with arterial stiffness and vascular damage markers.[9] A cohort of 820 hypertensive patients and 820 normotensive individuals matched for age and gender were enrolled in this study. These researchers measured carotid-femoral and carotid-radial PWV, aortic AI and central BP using pulse wave analysis and applanation tonometry. Plasma homocysteine (HCY), high-sensitivity C-reactive protein (hsCRP) and N-terminal pro-B-type natriuretic peptide (NT-proBNP) were also tested in these subjects. In both hypertensive and normotensive subjects, the central SBP and pulse pressure (PP) were significantly lower than brachial SBP and PP; this PP amplification was significantly lower in the normotensives (9.85 +/- 6.55 mm Hg) than in the hypertensives (12.64 +/- 6.69 mm Hg), but the amplification ratios

were comparable between the 2 groups. Blood pressure and age were closely related with aortic arterial stiffness. Compared with normotensive subjects, hypertensive subjects had higher carotid-femoral PWV and AI, and showed significantly lowered PP amplification ratio with age. Central PP was more strongly related to arterial stiffness and vascular damage markers than the other pressure indices. Multi-variate analyses revealed that carotid-femoral PWV and aortic AI were strongly influenced by central PP but not by the mean BP or brachial PP. The authors concluded that central PP is a more direct indicator of central arterial stiffness and a better marker of vascular aging than other BP variables. They stated that these findings support the use of central BP as a treatment target in future trials.

In 2018, Burns et al compared central BP and pulse wave reflection reassures sampled during a single resting laboratory visit against those obtained under ambulatory conditions.[13] Forty healthy participants (21 males; 24 ± 3 years) completed three measurements of brachial artery pulse wave analysis (Oscar 2 with SphygmoCor Inside) in the laboratory followed by 24 hours of ambulatory monitoring. Seventeen participants repeated the 24-hour ambulatory monitoring visit after at least 1 week. Ambulatory measures were divided into daytime (9 AM-9 PM), nighttime (1 AM-6 AM), and 24-hour periods. Compared with laboratory measurements, central systolic BP, augmentation pressure, and augmentation index (with and without heart rate normalization) were higher (all $P < .01$) during daytime and 24-hour periods but lower during the nighttime period (all $P<.001$). The drop in nighttime brachial systolic BP was larger than central systolic pressure (Δ -20 ± 6 vs. -15 ± 6 mm Hg; $p<.0001$). Repeat ambulatory measurements of central BP and pulse wave reflection displayed good-to-excellent intraclass correlation coefficients ($r = 0.58$-0.86; all $P<.01$), although measures of pulse wave reflection had higher coefficients of variation (14%-41%). The results highlight absolute differences in central BP and pulse wave reflection between discrete laboratory and ambulatory conditions. The authors concluded that the use of ambulatory measures of central BP and pulse wave reflection warrant further investigation for clinical prognostic value.

Grillo et al (2018) explored the consistency of aortic pulse wave velocity (PWV) as an indirect index of arterial stiffness and an independent cardiovascular risk factor.[14] Since studies providing a comparative estimate of the reproducibility of PWV across different noninvasive devices are lacking, the authors aimed to fill this gap using 6 different devices (Complior Analyse, PulsePen-ETT, PulsePen-ET, SphygmoCor Px/Vx, BPLab, and Mobil-O-Graph). These devices were evaluated in 102 high cardiovascular risk patients hospitalized for suspected coronary artery disease (72 males, 65 ± 13 years). PWV was measured in a single session twice, at 15-minute interval, and its reproducibility was assessed though coefficient of variation (CV), coefficient of repeatability, and intraclass correlation coefficient. The CV of PWV, measured with any of these devices, was <10%. Repeatability was higher with cuff-based methods (BPLab: CV = 5.5% and Mobil-O-Graph: CV = 3.4%) than with devices measuring carotid-femoral PWV (Complior: CV = 8.2%; PulsePen-TT: CV = 8.0%; PulsePen-ETT: CV = 5.8%; and SphygmoCor: CV = 9.5%). In the latter group, PWV repeatability was lower in subjects with higher carotid-femoral PWV. The differences in PWV between repeated measurements, except for the Mobil-O-Graph, did not depend on short-term variations of mean blood pressure or heart rate. This study shows that the short-term repeatability of PWV measures is good but not homogenous across different devices and at different PWV values.

Motau et al (2018) tried to determine the extent to which relations between modifiable risk factors and aortic function translate into increases in central aortic pulse pressure (PPc).[15] In 1232 black South Africans from the South West Township (SOWETO) of Johannesburg, we determined risk factors and aortic function from carotid-femoral pulse wave velocity (PWV) and aortic PPc, forward (Pf) and reflected (backward-Pb) wave pressures (applanation tonometry and SphygmoCor software). With adjustments for alternative risk factors and distending pressure (mean arterial pressure [MAP]), diabetes mellitus (treatment or HbA1c>6.5%, n=151) was associated with an increased PWV (7.10±2.09 versus 6.17±2.00 m/sec, p<.0001), and Pf (26±8 versus 24±8 mm Hg, p<.005), but neither brachial PP (46±14 versus 45±13, p=.19), PPc (36±12 versus 35±11 mm Hg, p=.48), nor Pb (17±6 versus 17±6 mm Hg, p=.83). Moreover, independent of alternative risk factors and MAP, uncontrolled hypertension (office BP>140/90 mm Hg, n=433), was associated with an increased Pf (26±12 versus 24±10 mm Hg, p<.01), but not with changes in brachial PP (45±19 versus 44±17, p=.75), PPc (35±16 versus 35±15 mm Hg, p=.93) or Pb (18±8 versus 17±8 mm Hg, p=.46). The authors concluded that neither brachial nor aortic PP are adequate indexes of relations between the modifiable conventional risk factors, uncontrolled hypertension or diabetes mellitus, and risk-related aortic functional changes.

Milan et al (2019) reviewed different validation studies of pulse wave velocity (PWV) estimation techniques and assessed their conformity to the Artery Society Guidelines and the American Heart Association recommendations.[16] Several devices had been developed and validated to noninvasively measure arterial stiffness, using applanation tonometry (SphygmoCor, PulsePen), piezoelectric mechanotransducers (Complior), cuff-based oscillometry (Arteriograph, Vicorder and Mobil-O-Graph), photodiode sensors (pOpmètre) and devices assessing brachial-ankle pulse wave velocity and cardiac-ankle PWV. Ultrasound technique and MRI remain confined to clinical research. Good agreement was found with the Artery Society Guidelines. Two studies (Complior, SphygmoCor Xcel) showed best adherence with the guidelines. In Arteriograph, MRI, ultrasound and SphygmoCor Xcel validation studies sample size was smaller than the minimum suggested by the guidelines. High discrepancies between devices were shown in distance estimation: in two studies (Arteriograph, Complior) path length was estimated in conformity to the guidelines. Transit time was calculated using the intersecting tangent method, but in two studies (Vicorder, pOpmètre) best agreement was found using the maximum of the second derivative. Six studies reached the accuracy level 'excellent' defined in the Artery guidelines. The authors concluded that the Method to assess transit time and path length need validation in larger populations. Further studies are required in different risk population to implement clinical applicability of every device.

SUMMARY OF EVIDENCE

There is a moderate-size body of literature reporting outcomes using SphygmoCor technology for noninvasive measurement of central blood pressure. In many of the abstracts, evaluation of the performance of SphygmoCor was not the primary focus of the study. Rather, SphygmoCor was the device used to obtain various cardiovascular parameters, including central blood pressure, relevant to the study endpoints. A large number of the abstracts compared SphygmoCor to various other noninvasive devices designed to measure or calculate central blood pressure, as well as other cardiac parameters. Notably, there was some overlap of investigators in some of the studies, which suggests the possibility of overlap in patient groups as well. A more recent study showed that the short-term repeatability of PWV measures is good but not homogenous across different devices and at different PWV values.

Ongoing and Unpublished Clinical Trials
A search of ClinicalTrials.com did not produce any studies that may influence this policy.

Government Regulations
National:
There is no national coverage determination on this topic. Medicare has established a fee for code 93050.

Local:
There is no local coverage determination on this topic.

(The above Medicare information is current as of the review date for this policy. However, the coverage issues and policies maintained by the Centers for Medicare & Medicare Services [CMS, formerly HCFA] are updated and/or revised periodically. Therefore, the most current CMS information may not be contained in this document. For the most current information, the reader should contact an official Medicare source.)

Related Policies

- Ambulatory Blood Pressure Monitoring for Screening and Diagnosis of Hypertension
- Ambulatory Event Monitors and Mobile Cardiac Outpatient Telemetry

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The articles reviewed in this research include those obtained in an Internet based literature search for relevant medical references through May 31, 2024, the date the research was completed.

Joint BCBSM/BCN Medical Policy History

Policy Effective Date	BCBSM Signature Date	BCN Signature Date	Comments
11/1/16	8/16/16	8/16/16	Joint policy established
11/1/17	8/15/17	8/15/17	Updated rationale section. Added reference #10. No change in policy status.
11/1/18	8/21/18	8/21/18	Added Vicorder® Pulse Wave and PhysioWave devices to title, updated rationale section. Added reference # 13-15.
11/1/19	8/20/19		Rationale updated reference #16 added. No change in policy status.
11/1/20	8/18/20		Routine policy maintenance, no change in policy status.
11/1/21	8/17/21		Routine policy maintenance, no change in policy status.
11/1/22	8/16/22		Routine maintenance; no change in policy status
11/1/23	8/15/23		Routine policy maintenance, no change in policy status. Vendor: N/A (ky)
11/1/24	8/20/24		Routine policy maintenance, no change in policy status. Vendor: N/A (ky)

Next Review Date: 3rd Qtr. 2025

Pre-Consolidation Medical Policy History

Original Policy Date	Comments
BCN:	Revised:
BCBSM:	Revised:

BLUE CARE NETWORK BENEFIT COVERAGE
POLICY: NONINVASIVE MEASUREMENT OF CENTRAL BLOOD PRESSURE (E.G., SPHYGMOCOR® SYSTEM, VICORDER® PULSE WAVE VELOCITY, PHYSIOWAVE)

I. Coverage Determination:

Commercial HMO (includes Self-Funded groups unless otherwise specified)	Not covered
BCNA (Medicare Advantage)	May be covered based on medical necessity. See government section of policy.
BCN65 (Medicare Complementary)	Coinsurance covered if primary Medicare covers the service.

II. Administrative Guidelines:

- The member's contract must be active at the time the service is rendered.
- Coverage is based on each member's certificate and is not guaranteed. Please consult the individual member's certificate for details. Additional information regarding coverage or benefits may also be obtained through customer or provider inquiry services at BCN.
- The service must be authorized by the member's PCP except for Self-Referral Option (SRO) members seeking Tier 2 coverage.
- Services must be performed by a BCN-contracted provider, if available, except for Self-Referral Option (SRO) members seeking Tier 2 coverage.
- Payment is based on BCN payment rules, individual certificate and certificate riders.
- Appropriate copayments will apply. Refer to certificate and applicable riders for detailed information.
- CPT - HCPCS codes are used for descriptive purposes only and are not a guarantee of coverage.