24-Hour Central Hemodynamic Load in Adults With and Without a History of COVID-19

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Abstract

BACKGROUND: Although hypertension is an established risk factor for severe COVID-19 illness, little is known about the effects of COVID-19 on blood pressure (BP). Central BP measures taken over a 24-hour period using ambulatory blood pressure monitoring (ABPM) adds prognostic value in assessing cardiovascular disease (CVD) risk compared to brachial BP measures taken at a single time point. We assessed CVD risk between adults with and without a history of COVID-19 via appraisal of 24-hour brachial and central hemodynamic load from ABPM. METHODS: Cross-sectional analysis was performed on 32 adults who tested positive for COVID-19 (29±13 years, 22 females) and 43 adults without a history of COVID-19 (28±12 years, 26 females). Measures of 24-hour hemodynamic load included brachial and central systolic and diastolic BP, pulse pressure, augmentation index (AIx), pulse wave velocity (PWV), nocturnal BP dipping, the ambulatory arterial stiffness index (AASI), and the blood pressure variability ratio (BPVR). RESULTS: Participants who tested positive for COVID-19 experienced 6±4 COVID-19 symptoms, were studied 122±123 days after testing positive, and had mild-to-moderate COVID-19 illness. The results from independent samples t-tests showed no significant differences in any of our 24-hour, daytime, or nighttime measures of central or peripheral hemodynamic load across those with and without a history of COVID-19 (p > 0.05 for all). CONCLUSIONS: No differences in 24-hour brachial or central ABPM measures were detected between adults recovering from mild-to-moderate COVID-19 and controls without a history of COVID-19. Adults recovering from mild-to-moderate COVID-19 do not have increased 24-hour central hemodynamic load.

Keywords: COVID-19; cardiovascular; blood pressure; central blood pressure; ABPM
Graphical abstract

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**METHODS**
- 32 adults who tested positive for COVID-19
- 43 adults without a history of COVID-19
- 24-hour brachial and central hemodynamic load from ambulatory blood pressure monitoring (ABPM)

**RESULTS**
- 6±4 COVID-19 symptoms
- 122±123 days since positive test date
- Mild-to-moderate disease severity

No significant differences in any of the 24-hour, daytime, or nighttime measures of central or peripheral hemodynamic load across those with and without a history of COVID-19 \( (p > 0.05) \).

**CONCLUSION**: No differences in 24-hour brachial or central ABPM measures were detected between adults recovering from mild-to-moderate COVID-19 and controls without a history of COVID-19. Adults recovering from mild-to-moderate COVID-19 do not have increased 24-hour central hemodynamic load.
Introduction

Hypertension is a modifiable risk factor for cardiovascular disease (CVD) that affects more than half of the U.S. adult population \(^1\). Although hypertension is an established risk factor for severe COVID-19 illness \(^2\), little is known about the effect of COVID-19 on blood pressure (BP). SARS-CoV-2, the virus responsible for COVID-19, may damage the cardiovascular system through angiotensin converting enzyme 2 (ACE2) receptor-mediated viral cell entry into vascular endothelial cells and an exacerbated hyperinflammatory immune response \(^3\). COVID-19 is associated with autonomic dysfunction, reductions in baroreflex sensitivity, endothelial dysfunction, and increases in arterial stiffness observed during \(^4\,^6\) and following \(^3\,^7\,^9\) active infection. Each of these cardiovascular mal-adaptations is important for BP regulation and may precede the development of hypertension \(^10\,^13\). There is emerging data to suggest that persistent elevations in BP may be part of the post-acute sequelae of COVID-19 illness \(^8\), with the potential for increases in brachial systolic and diastolic pressures leading to new onset hypertension following COVID-19 \(^2\). However, these studies have focused on the recovery of hospitalized COVID-19 patients \(^2\), and their findings stress the need for further studies exploring the BP response in those with less severe COVID-19 illness in non-clinical settings.

BP measures taken over a 24-hour period using ambulatory BP monitoring (ABPM) may add prognostic value in assessing CVD risk compared to single point BP measures \(^14\). 24-hour ABPM more strongly predicts CVD events and mortality compared to BP measures at a single time point \(^14\). Numerous measures of BP load can be obtained from ABPM, including nocturnal BP dipping, BP variability, and the ambulatory arterial stiffness index (AASI), each a significant predictor of target organ damage and future CVD events \(^14\,^15\). Moreover, BP technology has progressed such that 24-hour ambulatory central BP can now be assessed using a brachial
oscillometric cuff. Central pressures may have more pathophysiological relevance to CVDs than brachial pressures as the organs most susceptible to hypertension-induced target organ damage (i.e., the heart, kidneys, and brain) are exposed to central pressures. Indeed, 24-hour central ABPM shows stronger associations with preclinical measures of CVD risk and future CVD events compared to 24-hour brachial ABPM. Thus, with the potential for CV mal-adaptations to persist after COVID-19 and influence BP regulation, and considering the improved utility of central BP over brachial BP in assessing CVD risk, measuring central BP over a 24-hour period may improve CVD risk assessment in individuals recovering from COVID-19.

Therefore, the aim of this study was to use 24-hour brachial and central ABPM to compare hemodynamic load between adults with and without a history of COVID-19 infection. Measures of hemodynamic load included absolute brachial and central measures of systolic BP, pulse pressure (PP), as well as the augmentation index (AIx) and pulse wave velocity (PWV). We additionally assessed brachial and central nocturnal BP dipping, the AASI and the BP variability ratio (BPVR). We hypothesized that individuals with a history of COVID-19 would exhibit higher hemodynamic load, manifesting as higher brachial and central systolic BP, PP, BPVR, AASI, AIx and PWV, as well as lesser nighttime BP dipping, compared to those without a history of COVID-19.

Methods

Participants: 75 participants, 32 adults who tested positive for COVID-19 and 43 adults without a history of COVID-19, participated in this study. Participants who tested positive for COVID-19 provided documentation of either a positive viral or antibody test to confirm previous infection. Participants without a history of COVID-19 were asked to provide documentation of a
negative antibody test to rule out previous infection. All participants provided oral informed consent over a Zoom meeting prior to study initiation and all study procedures were approved by the Syracuse University Institutional Review Board.

**Study Design:** This study consisted of two study meetings: a Zoom consent meeting and a data collection study meeting. During the data collection study meeting, participants completed anthropometric and pulse oximetry assessments and initiated ABPM measures. Participants were asked to avoid food, sugary drinks, alcohol and caffeine for 12 hours, as well as exercise for 24 hours, prior to this meeting. This study was initially designed to be completed virtually when in-person human research was not allowed at Syracuse University. As such, 57 participants completed the data collection meeting over a Zoom video call. A box containing the study equipment and an information sheet with pictures and written explanations for how to use the equipment was dropped off at the participant’s place of residence prior to the Zoom meeting. During the Zoom meeting, participants were guided through the information sheet and given verbal instructions and visual demonstrations for how to complete study procedures. Additionally, prior to ABPM initiation, participants were guided through appropriately sizing and putting on the BP cuff as well as connecting the cuff to the ABPM device. Participants then performed one manual blood pressure measurement to ensure that the blood pressure cuff was appropriately sized and connected to the ABPM device prior to initiation of the 24-hour measurements. When in-person research was allowed to return at Syracuse University, 18 participants completed the aforementioned data collection procedures during a single 15-minute visit to the Human Performance Lab on the Syracuse University campus. After completion of the data collection study meeting, all participants were sent a Research Electronic Data Capture
(REDCap) link with a series of online questionnaires to complete in the 24 hours while undergoing ABPM.

Study Measurements

Anthropometrics and Pulse Oximetry: Participants self-reported height, a method that has been validated in young and middle-aged adults \(^1^8\). In older adults (≥ 60 years), a regression equation was applied to calculate actual height from self-reported height \(^1^8\). In the subsample of participants who completed in-person data collection \((n = 18)\), height was also measured using an automatic stadiometer for comparison of self-reported height to stadiometer measured height. Metrics for the criterion-related validity of self-reported height based on this subsample are provided in an online supplement (Figure S1). Weight and body composition were assessed using a digital scale with bioelectrical impedance analysis (Taylor Glass Body Composition Scale 5789FW, USA). Body mass index (BMI) was calculated as kg/m\(^2\). Arterial oxygen saturation was assessed at rest using a fingertip pulse oximeter placed on the index finger.

Ambulatory Blood Pressure Monitoring: ABPM with simultaneous pulse wave analysis (PWA) was performed using the oscillometric Mobil-O-Graph device (IEM, Stolberg, Germany). This device has been validated against gold standard invasive (intra-aortic catheter) and non-invasive (applanation tonometry) methods in a variety of populations \(^1^9,2^0\). Participants wore an appropriately sized upper arm BP cuff and BP readings were taken every 20 minutes during the day (07:00 hr – 22:00 hr) and every 30 minutes during the night (22:00 hr – 07:00 hr). The device initially inflates and deflates to perform a brachial oscillometric BP measurement, then reinflates again for approximately 10 seconds at the level of diastole to record brachial pulse waveforms. Participants were instructed to avoid exercise and vigorous activity during ABPM
and to keep their arm still and relaxed during measurements. \( \geq 70\% \) of the expected number of measurements in the 24-hour period were required to be valid for the data to be included for analysis. These methods follow 2017 American College of Cardiology/American Heart Association guidelines for 24-hour BP monitoring in adults \(^{21}\).

Mean arterial pressure (MAP) was determined as the lowest cuff pressure where the oscillations are maximal \(^{22}\). Central aortic pulse waves were derived from brachial pulse waveforms using an applied transfer function derived from the ARCSolver algorithm \(^{23}\). Central aortic waveforms were calibrated using the C2 methods (MAP/diastolic BP) \(^{14}\). The central aortic waveforms was decomposed into forward and reflected waves \(^{23}\) and used to estimate PWV \(^{24}\). AIx was calculated as the ratio of the augmented pressure to pulse pressure, and was also normalized to a heart rate of 75 bpm (AIx@75). Additional ABPM calculations of hemodynamic load were performed using both peripheral and central BP measures and included:

- Pulse pressure (PP): the difference of the systolic and diastolic BPs,
- Nighttime SBP and DBP Dipping: calculated as a percentage, and defined as \[ \frac{(\text{daytime BP} - \text{nighttime BP})}{\text{daytime BP}} \times 100 \] \(^{25}\),
- SBP, DBP and PP Variability: standard deviation (SD) of 24-hour SBP, DBP and PP \(^{26}\),
- BP Variability Ratio (BPVR): ratio of systolic to diastolic BP variability \(^{26}\),
- AASI: calculated from 24-hour BP measures as 1 – regression slope of diastolic on systolic BP values \(^{27}\).
Questionnaires: Participants self-reported their health history via a health questionnaire. COVID-19 disease severity was determined using the World Health Organization’s guidelines for COVID-19 disease severity based on self-reported symptomology

Increases in BP and new onset hypertension following COVID-19 infection mirrors trends in rising BP and increased rates of hypertension observed during the COVID-19 pandemic. It is possible that COVID-19 illness may cause changes in lifestyle and mental health symptomology (i.e., reduced physical activity, disturbed sleep, and increased anxiety, depressive symptomology, and stress symptomology) which could impact BP separate from the direct effects of COVID-19 illness on the vasculature. Thus, we included potential lifestyle and psychosocial factors that may have been affected by the COVID-19 pandemic as potential covariates in our analytic approach (described below). Sleep quality was determined using the Pittsburgh Sleep Quality Index (PSQI). Anxiety and depressive symptomology were appraised using the Generalized Anxiety Disorder 7-item scale (GAD-7) and Center for Epidemiological Studies Depression scale (CES-D), respectively. Perception of stress was assessed using the 10-item Perceived Stress scale (PSS-10). Post-traumatic stress disorder (PTSD) symptoms since the beginning of the COVID-19 pandemic were assessed using the PTSD Checklist for the DSM-5 (PCL-5). Physical activity levels were appraised using the International Physical Activity Questionnaire (IPAQ).

Statistical Analysis: Descriptive statistics are reported as mean ± SD for continuous variables and n (%) for categorical variables. All data were checked for normality using box plots, histograms, Q-Q plots, and skewness and kurtosis values. An independent samples t-test for continuous variables and a chi square test for categorical variables were used to compare participant characteristics and potential covariates between the COVID-19 and control groups. Potential
confounders found to be significantly different between groups would be entered as covariates using Analysis of Covariance (ANCOVA) as needed. Subsequently, an independent samples \( t \)-test was conducted to compare our ABPM central hemodynamic measures between the COVID-19 and control groups. Statistical significance was set \textit{a priori} at \( p < 0.05 \). All statistical analyses were performed using IBM SPSS Statistics version 27 (SPSS Inc., Chicago, IL).

**Results**

\textit{Participant Characteristics}: Participant characteristics are presented in Table 1. Age, sex, race/ethnicity, BMI, body composition, and arterial oxygen saturation did not significantly differ between the COVID-19 and control groups (Table 1, \( p > 0.05 \) for all). Ten participants reported a history of asthma (6 COVID-19, 4 controls), one participant reported a history of pulmonary disease (1 COVID-19), and one participant reported a history of breast cancer (1 control). No participants reported a history of diabetes, renal, or hepatic disease. One participant identified as a current smoker (1 control). Six participants identified as taking blood pressure lowering medication (1 COVID-19, 5 controls). Analyses were performed excluding the six participants who reported taking blood pressure lowering medications and we found that the removal of these participants did not change our overall results. Therefore, results are presented with these individuals included. Participants in our COVID-19 group were tested 121.9 ± 122.8 days after their positive COVID-19 test date, experienced 5.9 ± 4.4 COVID-19 symptoms, and had mild-to-moderate COVID-19 disease severity. There were no significant differences between our COVID-19 and control groups in scores for the PSQI, GAD-7, CES-D, PSS-10, PCL-5 or IPAQ (all \( p > 0.05 \)).
**Ambulatory Blood Pressure Monitoring**: Overall, our COVID-19 and control groups did not significantly differ in any of our central hemodynamic load measures (Table 2, \( p > 0.05 \) for all). 24-hour, nighttime, and daytime peripheral and central BPs did not significantly differ between the COVID-19 and control groups (all \( p > 0.05 \)). Furthermore, peripheral and central PPs, A\( \text{l} \)x, A\( \text{l} \)x\@75, and PWV did not significantly differ between the COVID-19 and control groups in 24-hour, daytime, or nighttime measures (all \( p > 0.05 \)). Additional measures comparing BP dipping and variability between our COVID-19 and control groups are presented in Table 3. There were no significant differences in peripheral and central measures for nighttime systolic and diastolic BP dipping, systolic and diastolic BP variability, PP variability, BPVR, or AASI between our COVID-19 and control groups (Table 3, \( p > 0.05 \) for all).

**Discussion**

In order to explore CVD risk in adults following mild-to-moderate COVID-19, this study utilized 24-hour brachial and central ABPM to compare hemodynamic load between adults with and without a history of COVID-19. Contrary to our hypothesis, there were no differences in brachial or central measures of systolic and diastolic BP, PP, A\( \text{l} \)x, PWV, nocturnal BP dipping, the AASI or BPVR between adults with and without a history of COVID-19. These findings suggest that adults recovering from mild-to-moderate COVID-19 may not have augmented hemodynamic load compared to those without a history of COVID-19.

Our findings differ from previous studies observing an increased risk for new-onset hypertension and vascular dysfunction in individuals after COVID-19 illness\(^2\)\(^-\)^\(^5\),\(^7\). However, there are important differences in our study design that may explain our differential findings. Most notably, previous studies finding an increased risk for new onset hypertension and ongoing...
vascular dysfunction have either not included a control group \(^2\), or used a control group on whom data collection occurred prior to the COVID-19 pandemic \(^3,7\). As we have mentioned, the COVID-19 pandemic itself may cause important changes to potential confounders of cardiovascular function – including sleep quality \(^36\) and mental health symptomology \(^34\) – that may not have been controlled for in these studies. Additionally, increases in BP and rates of new onset hypertension following COVID-19 parallels trends in rising BP and hypertension rates occurring during the COVID-19 pandemic \(^30\). Thus, whether the cardiovascular decrements observed in these studies are directly attributable to COVID-19 illness, or whether they are emblematic of the COVID-19 pandemic as a whole, may still need further investigation. That being said, our COVID-19 and control groups did not significantly differ in sleep quality, perceptions of stress or anxiety, depressive symptomology, PTSD symptomology, or physical activity levels, possibly contributing to our lack of group differences in our measures of hemodynamic load between our COVID-19 and control groups.

Another important distinction in our study is that our COVID-19 participants experienced predominantly mild-to-moderate COVID-19 illness, were studied, on average, four months after infection, and had no hospitalizations. Most of the previous literature on this subject has examined hospitalized COVID-19 patients in the acute phase of recovery from more severe cases of COVID-19 illness \(^4,5\). These patients may experience more severe cardiovascular consequences extending into recovery compared to our COVID-19 cohort. Few studies have examined outcomes from mild COVID-19 illness. Of these studies, those observing vascular dysfunction were mainly during the acute recovery period (<3 months) from COVID-19 \(^3,7\), while an emerging literature from the post-acute recovery phase (~3 months) is showing that vascular function may return and BP may normalize four to six months into recovery \(^8\). Our
findings support this emerging literature and suggest that, in mild-to-moderate cases, hemodynamic load may not significantly differ between adults with and without a history of COVID-19. Thus, while it should still be noted that hypertension remains an important risk factor for an increased COVID-19 disease severity, our findings suggest that mild-to-moderate COVID-19 may not, in turn, increase the risk for new onset hypertension.

**Limitations:** Although this study provides novel insight into the effect of COVID-19 on hemodynamic load, it is not without limitations. Due to the cross-sectional study design, hemodynamic data on COVID-19 participants prior to their infection was not available for the longitudinal assessment of hemodynamic measures. COVID-19 participants were also asked to self-report their COVID-19 symptomology, on average, four months after infection; potentially subjecting this study to the limitations of recall bias. Participants were given a list of COVID-19 symptoms as prompts to help improve recall accuracy. In addition, if participants under- or over-reported COVID-19 symptomology, we do not believe this would alter interpretation of our results given our null findings. Furthermore, this study utilized an estimated measure of PWV. Although the criterion-related validity of the Mobil-O-Graph device in estimating PWV has been established against gold standard invasive and non-invasive measures, the derivation of PWV is dependent on age and SBP, limiting its ability to accurately estimate PWV. That being said, the Mobil-O-Graph device provides accurate assessments of our other measures of CVD risk, notably BP, and the conclusions of our study are not affected by the one measure of PWV. The mean age of our study population was 28 years so results may not be generalizable to middle-aged and older adults. Finally, our study population is predominantly White, limiting the external validity of our findings to other racial and ethnic groups. COVID-19 has been shown to disproportionately affect racial and ethnic minority groups in both overall case numbers and the
severity of disease outcomes \(^{39}\). Minority populations already carry an excessive cardiovascular burden compared to White populations \(^{40}\); thus, it will be important for future studies to examine the impact of COVID-19 on hemodynamic load in minority populations to better understand how COVID-19 may affect future cardiovascular risk in these groups.

**Conclusion:** This study found no differences in 24-hour measures of hemodynamic load, including brachial and central measures of systolic and diastolic BP, PP, AIx, PWV, nocturnal BP dipping, the AASI, and BPVR, between adults with and without a history of COVID-19. These findings of similar central hemodynamic load may be taken to suggest comparable CVD risk in adults recovering from mild-to-moderate COVID-19 compared to adults without a history of COVID-19. Given the limitations of cross-sectional study design, future studies should longitudinally examine measures of hemodynamic load in individuals recovering from COVID-19 to better determine the risk for developing future hypertension.

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Table 1. Descriptive characteristics

| Characteristic                          | COVID-19 (n = 32) | Control (n = 43) | p-value |
|----------------------------------------|------------------|-----------------|---------|
| Age (years)                            | 28.5 ± 13.0      | 27.6 ± 12.3     | 0.750   |
| Female sex (n, %)                      | 22, 68.8         | 26, 60.5        | 0.460   |
| Race/Ethnicity (n, %)                  |                  |                 | 0.966   |
| NH White                               | 26, 81.3         | 34, 79.1        |         |
| NH Black                               | 1, 3.1           | 1, 2.3          |         |
| Hispanic/LatinX                       | 3, 9.4           | 4, 9.3          |         |
| Asian/Pacific Islander                | 2, 6.3           | 4, 9.3          |         |
| BMI (kg/m²)                            | 24.8 ± 3.7       | 25.3 ± 3.8      | 0.549   |
| Body Fat (%)                           | 23.3 ± 5.0       | 23.8 ± 5.7      | 0.660   |
| SaO₂ (%)                               | 98.0 ± 0.8       | 97.9 ± 1.9      | 0.769   |
| PSQI                                   | 6.7 ± 3.9        | 5.6 ± 3.0       | 0.191   |
| GAD-7                                  | 4.9 ± 5.2        | 4.8 ± 4.5       | 0.938   |
| CES-D                                  | 14.0 ± 11.6      | 10.1 ± 8.1      | 0.102   |
| PSS-10                                 | 15.9 ± 7.2       | 15.0 ± 6.3      | 0.580   |
| PCL-5                                  | 10.0 ± 11.1      | 10.7 ± 10.4     | 0.783   |
| IPAQ (MET-min/week)                    | 2573.6 ± 2489.6  | 3416.4 ± 2436.7 | 0.165   |
| Days Since Positive Test Date          | 121.9 ± 122.8    | ---             | ---     |
| Number of COVID-19 Symptoms            | 5.9 ± 4.4        | ---             | ---     |
| COVID-19 Disease Severity (1-5)        | 2.29 ± 0.8       | ---             | ---     |

Abbreviations: NH, non-Hispanic; BMI, body mass index; SaO₂, arterial oxygen saturation; PSQI, Pittsburgh sleep quality index; GAD-7, generalized anxiety disorder-7; CES-D, center for epidemiological studies depression scale; PSS-10, 10-item perceived stress scale; PCL-5, PTSD Checklist for the DSM-5; IPAQ, international physical activity questionnaire

*p < 0.05
Table 2. Independent samples t-test results comparing 24-hour ABPM measures between the COVID-19 and control groups

| Characteristic              | COVID-19 (n = 32) | Control (n = 43) | p-value |
|-----------------------------|-------------------|------------------|---------|
| **24-hour measures**        |                   |                  |         |
| Systolic BP (mmHg)          | 117.4 ± 10.8      | 118.8 ± 10.7     | 0.582   |
| Diastolic BP (mmHg)         | 70.9 ± 8.5        | 71.4 ± 8.3       | 0.787   |
| Heart Rate (bpm)            | 66.2 ± 9.2        | 66.3 ± 9.0       | 0.948   |
| MAP (mmHg)                  | 92.2 ± 8.9        | 93.1 ± 8.8       | 0.692   |
| pPP (mmHg)                  | 46.6 ± 6.5        | 47.3 ± 7.2       | 0.686   |
| cPP (mmHg)                  | 46.9 ± 14.0       | 49.5 ± 12.4      | 0.409   |
| cSystolic BP (mmHg)         | 123.3 ± 11.8      | 125.8 ± 12.8     | 0.405   |
| Alx (%)                     | 22.5 ± 5.9        | 21.9 ± 7.5       | 0.709   |
| Alx@75 (%)                  | 17.2 ± 5.7        | 16.9 ± 7.0       | 0.862   |
| PWV (m/s)                   | 5.4 ± 1.1         | 5.5 ± 1.4        | 0.562   |
| **Nighttime Measures**      |                   |                  |         |
| Systolic BP (mmHg)          | 109.5 ± 10.1      | 111.0 ± 10.6     | 0.546   |
| Diastolic BP (mmHg)         | 63.2 ± 7.7        | 64.4 ± 7.9       | 0.540   |
| Heart Rate (bpm)            | 60.0 ± 9.9        | 58.7 ± 9.3       | 0.586   |
| MAP (mmHg)                  | 84.5 ± 8.3        | 85.7 ± 8.5       | 0.561   |
| pPP (mmHg)                  | 46.2 ± 7.1        | 46.7 ± 7.2       | 0.792   |
| cPP (mmHg)                  | 56.4 ± 9.7        | 58.9 ± 14.3      | 0.407   |
| cSystolic BP (mmHg)         | 121.6 ± 12.6      | 125.9 ± 14.8     | 0.206   |
| Alx (%)                     | 24.0 ± 8.6        | 25.4 ± 8.2       | 0.472   |
| Alx@75 (%)                  | 15.6 ± 6.5        | 16.0 ± 7.5       | 0.784   |
| PWV (m/s)                   | 5.0 ± 1.0         | 5.3 ± 1.4        | 0.391   |
| **Daytime Measures**        |                   |                  |         |
| Systolic BP (mmHg)          | 119.1 ± 10.3      | 121.6 ± 11.5     | 0.354   |
| Diastolic BP (mmHg)         | 73.2 ± 9.0        | 74.0 ± 8.9       | 0.714   |
| Heart Rate (bpm)            | 68.5 ± 9.6        | 69.1 ± 9.2       | 0.775   |
| MAP (mmHg)                  | 94.2 ± 9.2        | 95.9 ± 9.3       | 0.457   |
| pPP (mmHg)                  | 45.7 ± 5.9        | 47.6 ± 7.7       | 0.266   |
| cPP (mmHg)                  | 43.6 ± 16.7       | 49.7 ± 10.9      | 0.082   |
| cSystolic BP (mmHg)         | 123.6 ± 12.2      | 125.6 ± 12.9     | 0.518   |
| Alx (%)                     | 21.7 ± 5.4        | 20.5 ± 7.8       | 0.451   |
| Alx@75 (%)                  | 17.0 ± 6.5        | 17.3 ± 7.3       | 0.863   |
| PWV (m/s)                   | 5.3 ± 1.0         | 5.6 ± 1.5        | 0.363   |

Abbreviations: BP, blood pressure; MAP, mean arterial pressure; pPP, peripheral pulse pressure; cPP, central pulse pressure; cSystolic, central systolic; Alx, augmentation index; Alx@75, heart rate-corrected augmentation index; PWV, pulse wave velocity

*p < 0.05
Table 3. Independent samples t-test results comparing blood pressure dipping and variability between COVID-19 and control groups

| Characteristic          | COVID-19 (n = 32) | Control (n = 43) | p-value |
|-------------------------|-------------------|------------------|---------|
| pSBP Dipping (%)        | 7.99 ± 5.1        | 8.49 ± 6.5       | 0.730   |
| pDBP Dipping (%)        | 13.3 ± 7.0        | 12.8 ± 6.7       | 0.727   |
| cSBP Dipping (%)        | 1.40 ± 6.2        | -0.40 ± 7.6      | 0.293   |
| cDBP Dipping (%)        | 12.9 ± 7.1        | 11.4 ± 7.6       | 0.427   |
| pSBP Variability (au)   | 13.3 ± 3.0        | 13.2 ± 2.6       | 0.868   |
| pDBP Variability (au)   | 10.9 ± 2.2        | 10.3 ± 1.7       | 0.234   |
| cSBP Variability (au)   | 13.6 ± 4.4        | 13.4 ± 3.4       | 0.846   |
| cDBP Variability (au)   | 10.8 ± 2.4        | 10.2 ± 1.7       | 0.252   |
| pPP Variability (au)    | 11.7 ± 4.8        | 10.6 ± 2.7       | 0.204   |
| cPP Variability (au)    | 9.2 ± 2.1         | 9.2 ± 1.9        | 0.982   |
| pBPVR (au)              | 1.24 ± 0.3        | 1.29 ± 0.2       | 0.400   |
| cBPVR (au)              | 1.37 ± 0.8        | 1.37 ± 0.5       | 0.970   |
| pAASI (au)              | 0.53 ± 0.2        | 0.51 ± 0.2       | 0.680   |
| cAASI (au)              | 0.69 ± 0.2        | 0.76 ± 0.2       | 0.196   |

Abbreviations: p, peripheral; c, central; SBP, systolic BP; DBP, diastolic BP; PP, pulse pressure; BPVR, blood pressure variability ratio; AASI, ambulatory arterial stiffness index

*p < 0.05