REVIEW ARTICLE

Ambulatory Blood Pressure Monitoring

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ABSTRACT

Hypertension is a major health care concern. With office and home blood pressure monitoring giving insufficient information, ambulatory blood pressure monitoring has emerged as the investigation of choice for hypertension.

Keywords: Ambulatory blood pressure monitoring, white coat hypertension, morning surge, masked hypertension

The specter of hypertension looms large over the world as a leading cause of morbidity and mortality. Almost 1 billion adults representing about a quarter of the world's population had hypertension in 2000. We are likely to see this number go up to 1.56 billion by the year 2025—an increment of almost 60%. In India, the prevalence of hypertension has increased from 2% to 25% among urban residents and from 2% to 15% among the rural residents in the last 60 years.[1] Small reductions in mean systolic blood pressure (SBP) are known to significantly reduce mortality from stroke and ischemic heart disease.[2]

Office blood pressure (BP) recordings are like a single snap-shot in time and give only limited information. They have a low reproducibility as office readings taken by different personnel and/or on different instruments may differ. With office recordings, we cannot assess variations in BP at different times and in different situations, effect of stress, exercise or sleep. They may, therefore at times, not be truly representative of the BP of the patient.

Out-of-office BP measurements, therefore become necessary and these may be obtained by:

➲ Home blood pressure monitoring (HBPM) where the patient or his attendants measure the BP at home at specified intervals.

➲ Ambulatory blood pressure monitoring (ABPM) using an automatic computer-based programable BP monitoring system.

The superiority of ABPM over HBPM is well-established. ABPM provides a good estimate of the 'true' or 'mean BP' level, a record of the 'diurnal variation' of BP and of BP variability.[3]

INDICATIONS OF ABPM

Several recent guidelines now recommend ABPM as the investigation of choice in the following conditions:[4-7]

➲ Identification of white coat hypertension

➲ Identification of masked hypertension

➲ Identification of abnormal 24-hour BP trends:
  ● Increased BP variability
  ● Daytime hypertension
  ● Night-time hypertension
    ▪ Dipping patterns
    ▪ Morning surges
    ▪ Obstructive sleep apnea and BP

➲ Assessment of treatment

➲ Assessment of BP trends in the elderly, in young patients, in high-risk patients and in pregnancy.

➲ Identification of resistant hypertension, endocrine hypertension and hypertension in Parkinsonism.

AMBULATORY BLOOD PRESSURE RECORDING

ABPM was developed about four decades ago but never gained popularity due to cumbersome equipment and lack of proper standardization. Modern equipment is light-weight and portable. It consists of a cuff, a small monitor typically weighing <1 pound that is attached to a belt, and a tube connecting the cuff and monitor

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(Fig. 1 a and b). The monitor automatically records BPs every 30 minutes during waking hours and every 60 minutes during rest. These timings are programable. The monitors use the 'oscillometric technique', (i.e., they assess oscillations caused by arterial pulse pressure). Data recorded by the monitor is analyzed by device-specific software.

Limits for normal SBP and diastolic blood pressure (DBP) during daytime waking hours and night-time sleeping periods can be programed separately. Most of the currently available ABPM devices are independently validated according to the European Society of Hypertension International Protocol.[8]
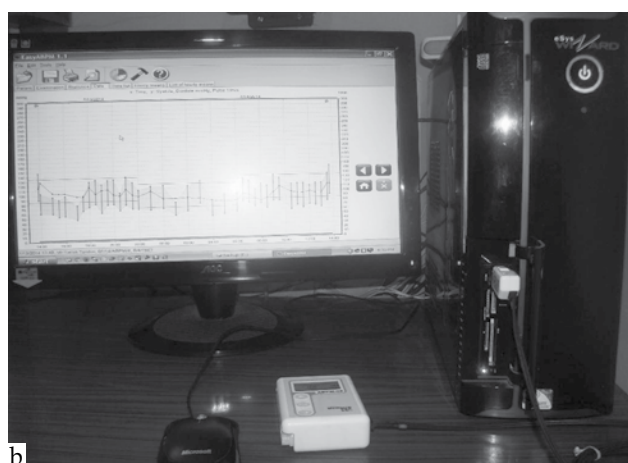
Figure 1 a and b. Modern light-weight and portable ABPM device.

ABPM TERMINOLOGY

Percentage Successful

The machine should record at least 2 readings per hour during waking hours from 6.00 am to 10.00 pm and at least 1 reading per hour from 10.00 pm to 6.00 am. At least 80% of the recordings should be successful for a good record.

Mean Blood Pressure (Mean)

Is the average SBP and DBP over a 24-hour period. It includes both the active (awake) and the passive (sleep) periods. Normal value is 130/80 mmHg.

Percent Time Elevation

The percent time elevation (PTE) also called 'pressure load' is calculated individually for the entire 24-hour period, the active period and the passive period. It is the percentage of time during which the SBP, DBP or both are above limits of normal. A PTE above 25% indicates hypertension.

Hyperbaric Impact/Index

The hyperbaric impact/index (HBI) is a quantitative measure of high BP. It indicates the time and magnitude of BP excess above the upper limit of the tolerance level in a given period of time. Expressed in mmHg × hour, an HBI of >15 mmHg × hour would indicate suspected hypertension and an HBI of >50 mmHg × hour would indicate hypertension. It would also help in evaluating the response to medication.[9]

Diurnal Index

The diurnal index (DI) is the difference in mean BP between awake and sleeping BP calculated as a percentage.

- ➲ DI = [1 – (night mean SBP/awake mean SBP)] × 100
- ➲ Dipping patterns based on the DI are described later.

Morning Surge[10]

This is the percentage difference between mean SBP during the first 2 hours of waking-up and the lowest level recorded at night.

Double Product

It is also known as rate pressure product (RPP) is a marker of cardiac load. It gives a direct indication of

the energy requirements of the heart and is a good measure of energy consumption.

Double product (RPP) = Heart rate (HR) × SBP

Based on RPP the hemodynamic response can be classified as:

➲ High: >30,000

➲ High intermediate: 25,000-29,999

➲ Intermediate: 20,000-24,999

➲ Low intermediate: 15,000-19,999

➲ Low: <14,999

Mean Arterial Pressure

Mean arterial pressure = DBP + [(SBP-DBP)/3]

Normal range is 70-110 mmHg.

Pulse Pressure

Pulse pressure = SBP-DBP

Normal range is 40-50 mmHg.

ILLUSTRATIVE ABPM RECORDINGS

White Coat Hypertension

White coat hypertension is a condition, where the subject demonstrates raised SBP and DBP in the doctor's office or in other stressfull situations but has normal BP recordings measured at home or elsewhere. The readings measured by a doctor are usually higher than those measured by a nurse.

The patient often has tachycardia and usually does not exhibit evidence of any target organ damage. This condition is more common in women, in the elderly and those with high levels of anxiety.

White coat hypertension may be seen in 20-30% of subjects. It is no longer considered to be innocuous. Recent evidence suggests that subjects with white coat hypertension have more than double the risk of developing hypertension, have increased risk of developing diabetes and increased left ventricular mass over time (Fig. 2).[5,11,12]
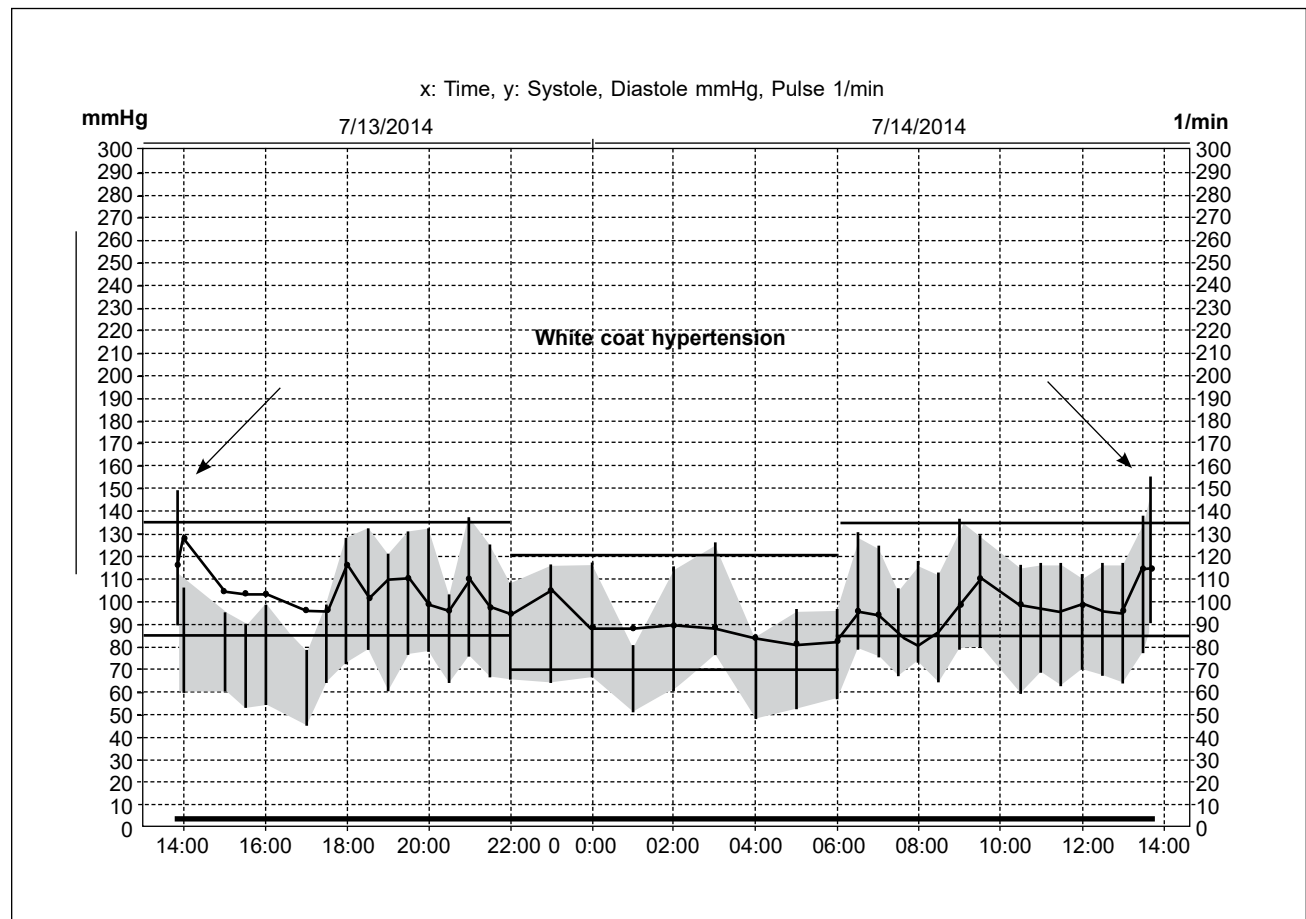

Figure 2. White coat hypertension.

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Nocturnal Dipping Patterns

Based on DI nocturnal dipping patterns are described as:[5,6]

- **Normal dipping:** The DI is between 10% and 20% (Fig. 3)
- **Nondipper:** The DI is between 0% and 10% (Fig. 4)
- **Extreme dipper:** The DI is >20% (Fig. 5)
- **Reverse dipper:** The DI is <0% (Fig. 6).

Nondipping may be seen in as much as 39% of the population with a much greater prevalence reaching 78% in diabetics. It correlates with cardiovascular (CV) autonomic neuropathy in diabetics. Nondippers, patients with exaggerated nocturnal BP fall (extreme-dippers) and those with increased nocturnal BP (reverse dippers), all have greater hypertensive target organ damage, CV events and stroke.

Abnormal dipping patterns are also a risk for left ventricular hypertrophy, silent cerebrovascular disease, microalbuminuria and progression of renal damage.[13]

Morning Surge

More than 20% difference between the average SBP of the first 2 hours after awakening and the lowest SBP recorded during the night is taken as an abnormal morning surge (Fig. 7).[10]

Shearing mechanical stress, inflammatory cascades and endothelial dysfunction associated with morning surge are responsible for the increase in CV and cerebrovascular events caused by it. Morning surge also causes increase in left ventricular mass index, increased carotid intima-media thickness and development of microalbuminuria.[11,13]

Masked Hypertension

Masked hypertension or pseudo-normotension is the reverse of white coat hypertension. The patient has normal BP recordings in the doctor's office but on ABPM is found to be hypertensive. It has a prevalence of 10-20%. These patients have similar CV risk and complications as hypertensives.[8,11]
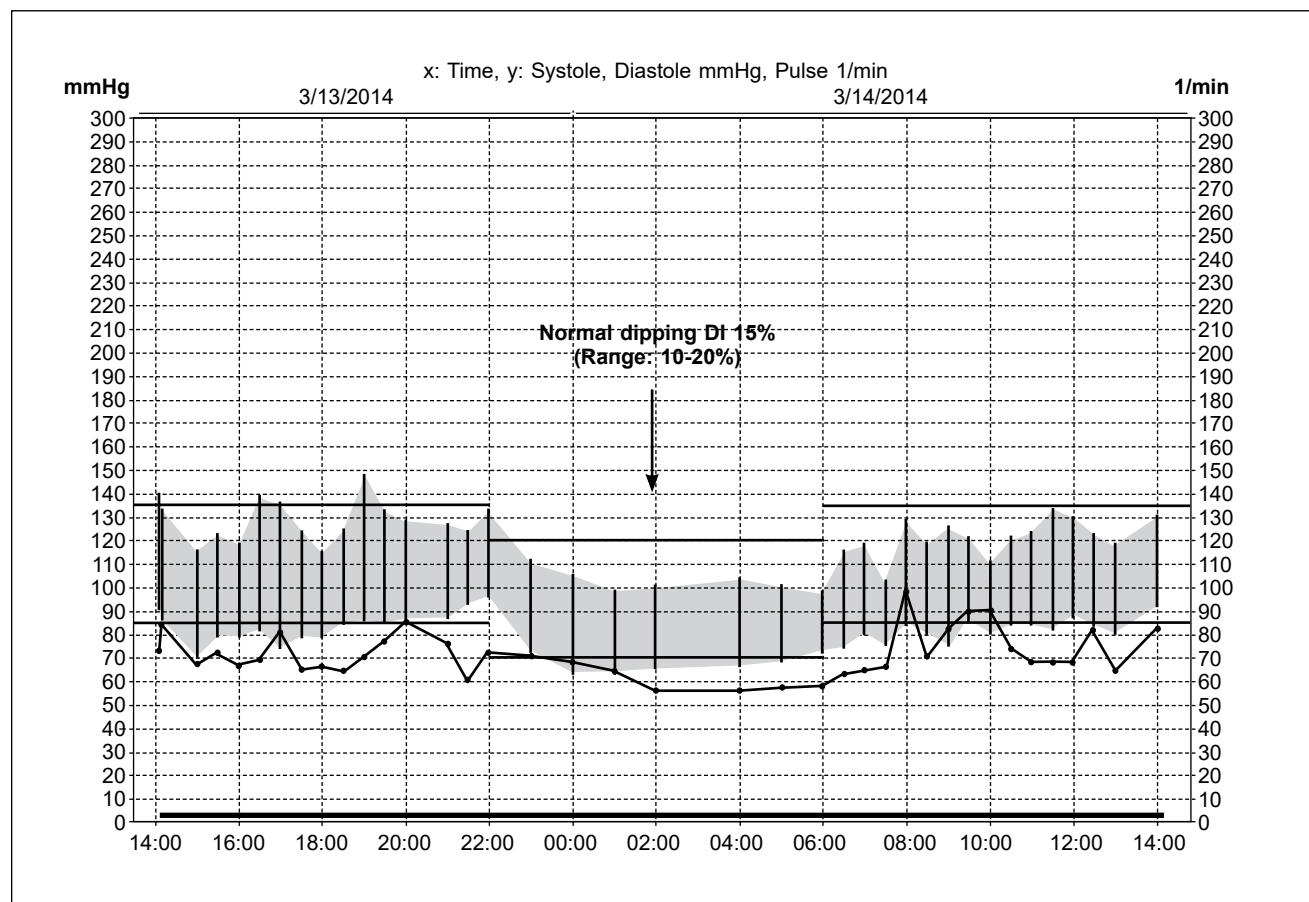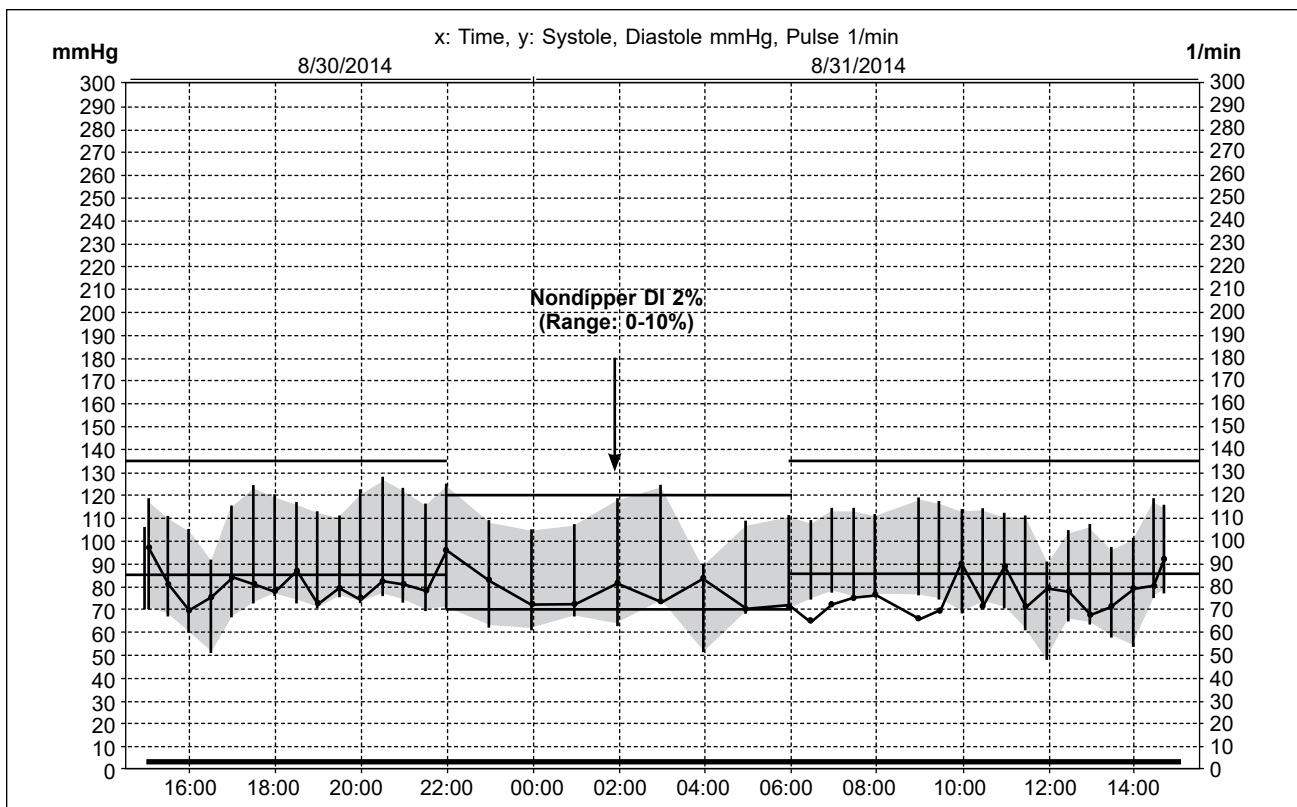

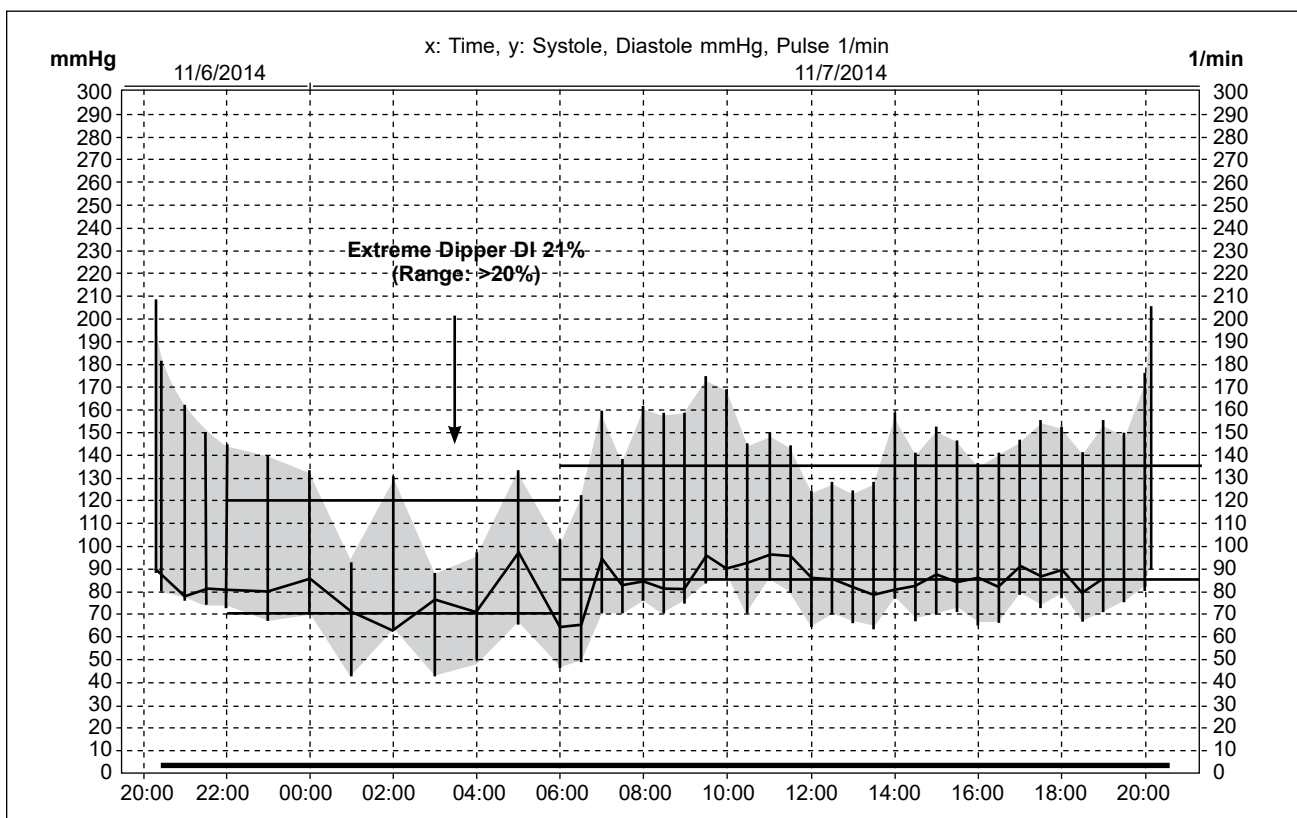**Figure 3.** Normal dipping.

Figure 4. Nondipper.

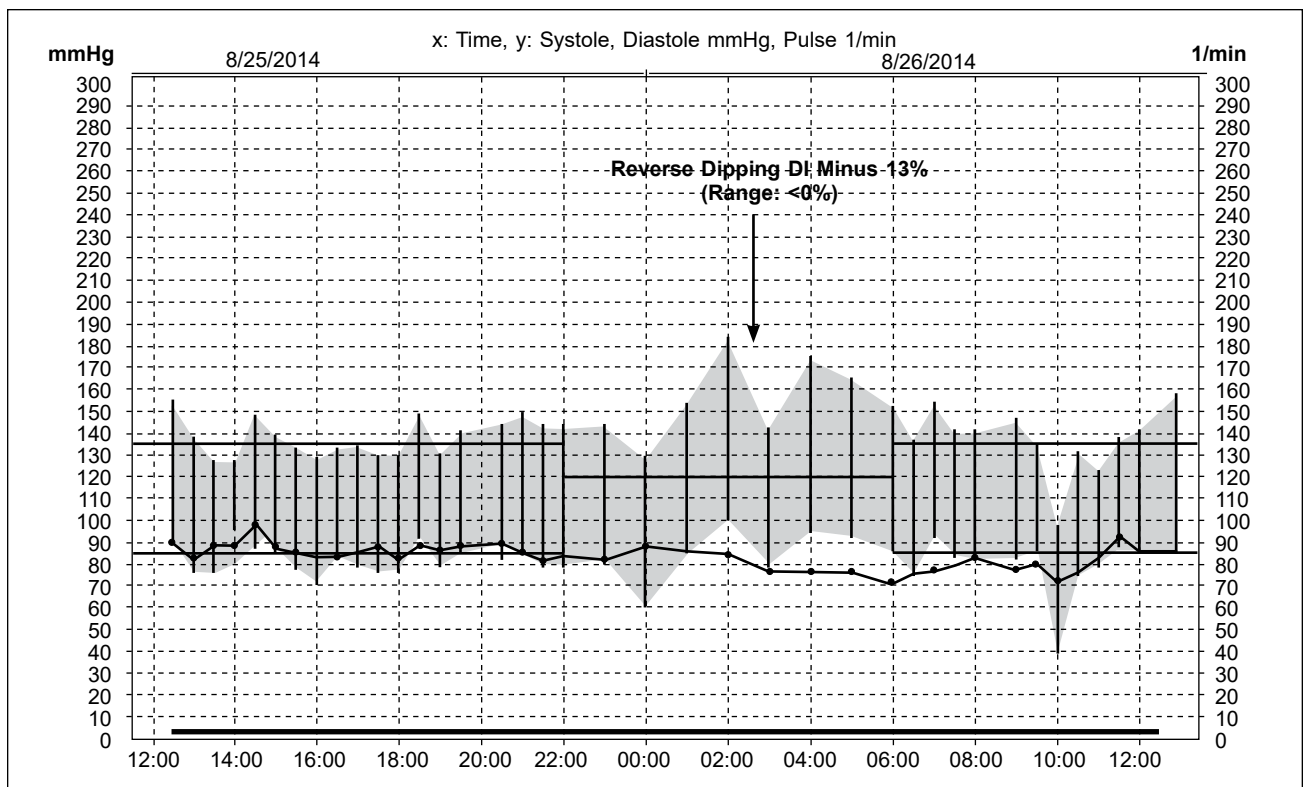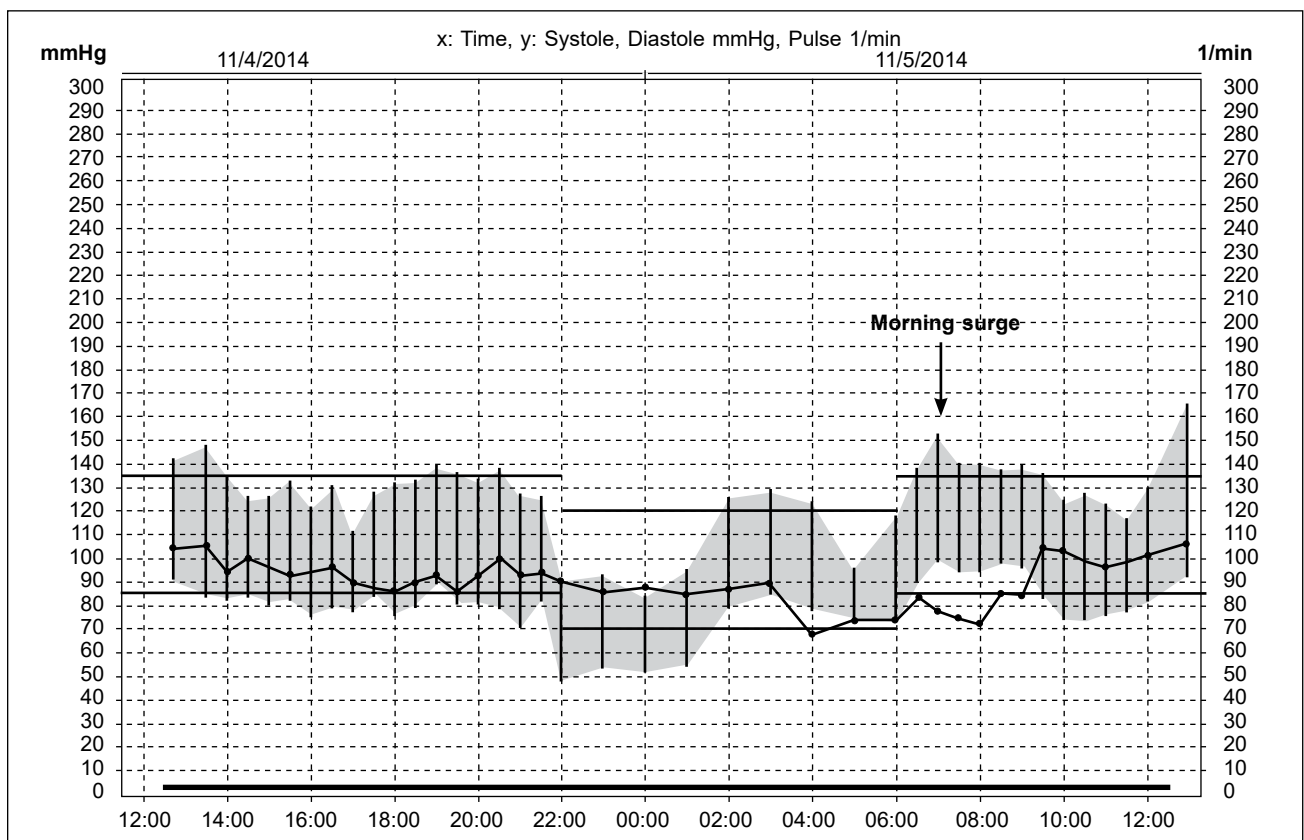**Figure 5.** Extreme dipper.

Figure 6. Reverse dipper.

Figure 7. Morning surge.

REFERENCES

1. Association of Physicians of India. J Assoc Physicians India. 2013;61(2 Suppl):6-36.

2. Lewington S, Clarke R, Qizilbash N, Peto R, Collins R. Lancet. 2002;360(9349):1903-13.

3. McGrath BP. Med J Aust. 2002;176(12):588-92.

4. NICE-BHS Guidelines on the diagnosis and treatment of high blood pressure (hypertension). (2011). [online] Available from http://guidance.nice.org.uk/CG127 [Accessed on June, 2015].

5. O'Brien E, Parati G, Stergiou G, Asmar R, Beilin L, Bilo G, et al. J Hypertens. 2013;31(9):1731-68.

6. 2013 ESH/ESC Guidelines for the management of arterial hypertension. [online] Available from http://dx.doi.org/10.1093/eurheartj/eht151 [Accessed on June, 2015].

7. Flynn JT, Daniels SR, Hayman LL, Maahs DM, McCrindle BW, Mitsnefes M, et al. Hypertension. 2014;63:1116-35.

8. Stergiou GS, Karpettas N, Atkins N, O'Brien E. Blood Press Monit. 2010;15(1):39-48.

9. Hermida RC, Fernández JR, Mojón A, Ayala DE. Hypertension. 2000;35(1 Pt 1):118-25.

10. Kario K, Shimada K, Pickering TG. J Cardiovasc Pharmacol. 2003;42(Suppl 1):587-91.

11. Leitão CB, Canani LH, Silveiro SP, Gross JL. Arq Bras Cardiol. 2007;89(5):347-54.

12. Mancia G, Sega R, Bombelli M, Quarti-Trevano F, Facchetti R, Grassi G. Pro Diabetes Care. 2009;32(Suppl 2):S305-9.

13. Kario K. Hypertension. 2010;56(5):765-73.

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