UNIVERSITE DE LAUSANNE – FACULTE DE BIOLOGIE ET DE MEDECINE

Département de Médecine
Service de néphrologie et consultation d'hypertension

Reactive rise in blood pressure upon cuff inflation: cuff inflation at the arm causes a greater rise in pressure than at the wrist in hypertensive patients

THESE

préparée sous la direction du Professeur Michel Burnier, chef du service de néphrologie et consultation d'hypertension au CHUV, et présentée à la Faculté de biologie et de médecine de l'Université de Lausanne pour l'obtention du grade de

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par

Alexia CHARMOY

Médecin diplômée de la Confédération Suisse
Originaire d'Ecoteaux (VD)

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Résumé en français :

Il est admis que l'inflation d'une manchette à pression au niveau du bras engendre une augmentation réactionnelle de la tension artérielle qui peut être le résultat d'une gêne lors de l'inflation et peut diminuer la précision de la mesure. Dans cette étude, nous comparons séquentiellement l'augmentation de la tension artérielle lorsque la manchette à pression est positionnée au niveau du bras et au niveau du poignet.

Nous avons étudié un collectif de 34 participants normotendus et 34 patients hypertendus. Chacun d'eux était équipé de deux manchettes à pression, l'une au niveau du bras et l'autre au niveau du poignet. Nous avons randomisé l'ordre d'inflation des manchettes ainsi que la pression d'inflation maximale (180mmHg versus 240mmHg). Trois mesures étaient effectuées pour chaque pression d'inflation maximale, ceci au bras comme poignet, et leur séquence était également randomisée. En parallèle, un enregistrement continu de la tension artérielle avait lieu au niveau du majeur de la main opposée à l'aide d'un photoplethysmographe. Cette valeur était considérée comme la valeur de tension artérielle au repos.

Pour les participants normotendus, aucune différence statistiquement significative n'a pu être mise en évidence en lien avec la position de la manchette à pression, ceci indépendamment de la pression d'inflation maximale. Variation de la pression systolique à 180 mmHg: 4.3±3.0 mmHg au bras et 3.7±2.9 mmHg au poignet (p=ns), à 240 mmHg: 5.5±3.9 au bras et 4.2±2.7 mmHg au poignet (p=0.052). En revanche, concernant les patients hypertendus, une augmentation significative de la tension artérielle a été mise en évidence entre le bras et le poignet. Ceci pour les valeurs de tension artérielle systolique et diastolique et quelle que soit la pression d'inflation maximale utilisée. Augmentation de la pression artérielle systolique : 6.5±3.5 mmHg au bras et 3.8±2.1mmHg au poignet pour une pression d'inflation maximale de 180 mmHg ($p<0.01$) et respectivement 6.4±3.5 mmHg et 4.7±3.0 mmHg pour 240 mmHg ($p=0.01$). L'augmentation des valeurs de tension artérielle était indépendante de la valeur tensionnelle de base.

Ces résultats montrent que les patients hypertendus réagissent significativement moins à l'inflation d'une manchette à pression lorsque celle-ci est positionnée au niveau du poignet par rapport au bras, ceci indépendamment des valeurs de tension artérielle de base des patients. Nous pouvons donc suggérer que l'inflation d'une manchette à pression cause moins de désagrément lorsqu'elle est placée au niveau du poignet, notamment chez les patients hypertendus et qu'elle peut être une alternative à la mesure standard au niveau du bras.

Reactive rise in blood pressure upon cuff inflation: cuff inflation at the arm causes a greater rise in pressure than at the wrist in hypertensive patients

Alexia Charmoy[a], Grégoire Würzner[a], Christiane Ruffieux[b], Christopher Hasler[a], François Cachat[c], Bernard Waeber[d] and Michel Burnier[a]

Objective Cuff inflation at the arm is known to cause an instantaneous rise in blood pressure, which might be due to the discomfort of the procedure and might interfere with the precision of the blood pressure measurement. In this study, we compared the reactive rise in blood pressure induced by cuff inflation when the cuff was placed at the upper arm level and at the wrist.

Participants and methods The reactive rise in systolic and diastolic blood pressure to cuff inflation was measured in 34 normotensive participants and 34 hypertensive patients. Each participant was equipped with two cuffs, one around the right upper arm (OMRON HEM-CR19, 22–32 cm) and one around the right wrist (OMRON HEM-CS 19, 17–22 cm; Omron Health Care Europe BV, Hoofddorp, The Netherlands). The cuffs were inflated in a double random order (maximal cuff pressure and position of the cuff) with two maximal cuff pressures: 180 and 240 mmHg. The cuffs were linked to an oscillometric device (OMRON HEM 907; Omron Health Care). Simultaneously, blood pressure was measured continuously at the middle finger of the left hand using photoplethysmography. Three measurements were made at each level of blood pressure at the arm and at the wrist, and the sequence of measurements was randomized.

Results In normotensive participants, no significant difference was observed in the reactive rise in blood pressure when the cuff was inflated either at the arm or at the wrist irrespective of the level of cuff inflation. Inflating a cuff at the arm, however, induced a significantly greater rise in blood pressure than inflating it at the wrist in hypertensive participants for both systolic and diastolic pressures ($P<0.01$), and at both levels of cuff inflation. The blood pressure response to cuff inflation was independent of baseline blood pressure.

Conclusions The results show that in hypertensive patients, cuff inflation at the wrist produces a smaller reactive rise in blood pressure. The difference between the arm and the wrist is independent of the patient's level of blood pressure. *Blood Press Monit* 12:275–280 © 2007 Wolters Kluwer Health | Lippincott Williams & Wilkins.

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[a]Division of Nephrology and Hypertension Consultation, [b]Institute for Social and Preventive Medicine, [c]Division of Pediatric Nephrology, [d]Division of Physiopathology and University Hospital, Lausanne, Switzerland

Correspondence to Professor Michel Burnier, Service de Néphrologie et Consultation d'Hypertension, Centre Hospitalier Universitaire Vaudois, Rue du Bugnon 17, 1011 Lausanne, Switzerland
Tel: +41 21 314 11 54; fax: +41 21 314 11 39;
e-mail: michel.burnier@chuv.ch

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Introduction

Hypertension is a well-established cardiovascular risk factor, which is widely prevalent around the world [1]. The standard of care for the detection and management of hypertensive patients is blood pressure (BP) measurement at the physician's office because this determination correlates with the cardiovascular risk [2]. BP measurement at the office is, nevertheless, known to have numerous limitations linked to the physician as well as to the clinical situation during which BP is taken (such as the observer bias or the digit preference or the white coat effect) [3]. Methods of BP monitoring, independently of physicians, such as ambulatory BP recording or home BP measurements have, therefore, been developed [2,4]. Both ambulatory BP monitoring and home BP measurements are increasingly used in clinical practice, and recent data have shown that BP values obtained with these methods actually correlate better with target organ damage than office BP values [5,6]. To monitor home BP, patients can use devices that measure BP either at the arm or at the wrist. In recent years, several arm devices have been validated for home BP monitoring. Wrist devices have, however, become increasingly popular [7], probably because they are relatively inexpensive and user-friendly [8,9], and validated wrist devices have also become available [10,11].

The measurement of BP represents a transient stress for the patient. Previous studies have demonstrated that cuff inflation *per se* induces a reactive rise in BP, in particular when BP is high [12–14]. In this situation, cuff inflation above systolic BP (SBP) might provoke a pain reaction and a rise in BP via the activation of the sympathetic nervous system. This reactive rise in BP might lead to a falsely elevated BP measurement.

The importance of the reactive rise in BP produced by the inflation of a cuff at the level of the wrist has never been investigated so far. We have hypothesized that cuff inflation at the wrist is less painful than that at the arm level, and hence produces a smaller reactive increase in BP. To test this hypothesis, we have compared the BP responses induced by cuff inflation when the cuff is placed around the upper arm and around the wrist in normotensive participants and hypertensive patients.

Participants and methods

Selection of patients and participants

Two groups of participants were investigated, that is, healthy normotensive participants with a BP below 130/80 mmHg and hypertensive patients, either treated (irrespective of their BP) or untreated (with a BP $\geq$ 140/90 mmHg). All participants were duly informed of the protocol and signed an informed consent before entering the study. The protocol was approved by the local hospital's ethics committee.

Study protocol

On the day of investigation, participants were comfortably installed on a bed in a supine position for the duration of the experiments with the wrist positioned at the level of the heart. Participants were not allowed to smoke within an hour of the investigation. All measurements were begun after at least 5 min of rest. All measurements were performed by the same investigator (AC, MD student) and under the same experimental conditions. To blunt the alarm reaction, the procedure was carefully explained and demonstrated to each participant before the study was initiated. Each participant was equipped with two cuffs, one around the right upper arm (OMRON HEM-CR19, 22–32 cm) and one around the right wrist (OMRON HEM-CS 19, 17–22 cm, pediatric cuff). The cuffs were inflated (by group of three measurements) in a double random order (maximal cuff pressure and position of the cuff) every 2 min with two maximal cuff pressures: 180 and 240 mmHg. The cuffs were linked to a validated oscillometric device (OMRON HEM 907) [15]. During cuff inflations, BP was measured continuously and noninvasively on the left middle finger of the contralateral arm by photoplethysmography (Finapres; Ohmeda, Louisville, Colorado, USA). This device was connected to a printer (Philips PM8272 recorder) to record the reactive rise in BP induced by cuff inflations. For each recording, the baseline pressure (mean of a 1-min recording), the maximal pressure (mean of the peak over 10–15 s) and the recovery pressure (mean of a 1-min recording) were recorded while BP was being measured at the upper arm or at the wrist. The reactive rise in BP due to cuff inflation was defined as the difference between the baseline and the maximal BP measured at the finger of the left arm when cuff was inflated at the right arm either at the wrist or above the elbow.

Statistical analysis

Data are presented as means ± SD. Statistical differences between groups (normotensive vs. hypertensive) or levels of cuff inflation (180 vs. 240 mmHg) were analyzed using Student's *t*-test. Moreover, a two-way analysis of variance for repeated measurements (arm vs. wrist; level of cuff inflation: 180 vs. 240 mmHg) was performed separately for data in normotensive participants and hypertensive patients. Correlations were made between the participants' BPs and the reactive rise in BP to ensure that the reactive rise in BP were independent of the patients' BP levels. The limit of significance was determined at $P < 0.05$.

Results

The baseline characteristics of the participants are shown in Table 1. Hypertensive patients were older than the normotensive participants and had more comorbidities than control participants. The percentage of smokers or exsmokers was, however, comparable in both groups.

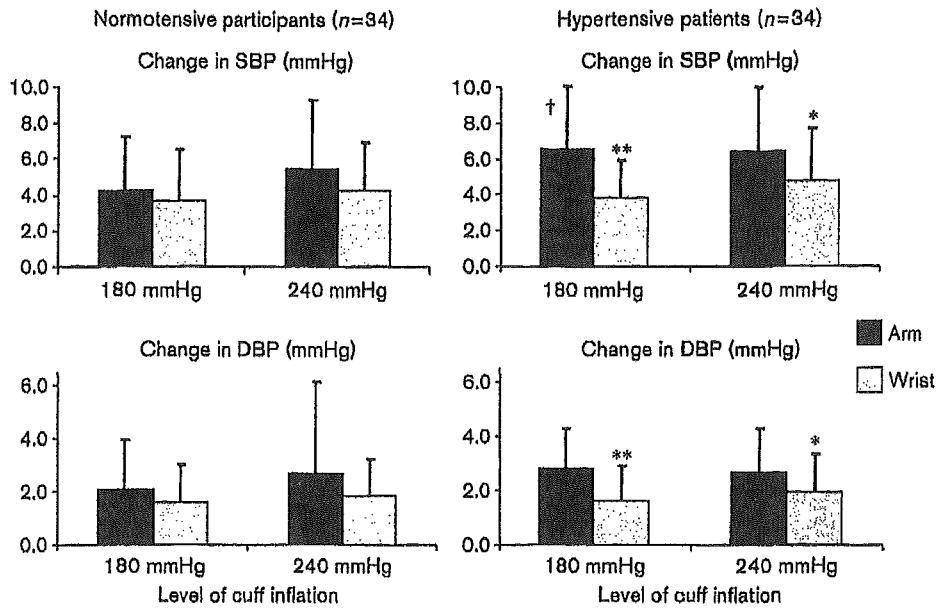
Table 1 Baseline characteristics of the participants

	Normotensive participants	Hypertensive patients
Number	34	34
Age (range)	36 (23–84)	56 (19–68)*
Ratio men/women	12:22	12:22
BMI ± SD	23.3 ± 4.5	26.6 ± 5.3
SBP ± SD (mmHg)	114 ± 12	135 ± 20*
DBP ± SD (mmHg)	68 ± 10	78 ± 12*
Mean arm circumference ± SD (cm)	28 ± 3	30 ± 4
Mean wrist circumference ± SD (cm)	17 ± 2	18 ± 3
Cardiovascular risk factors		
Smokers (*n*)	9	10
Exsmokers (*n*)	4	7
Diabetes (*n*)	–	6
Dyslipidemia (*n*)	–	9
Obesity BMI ≥ 30 (*n*)	3	9
Number of patients untreated	34	4
Number of patients treated	0	30
IEC/ARB	–	17
BB	–	14
CA	–	11
D	–	15
AB	–	1

*P<0.05 hypertensive vs normotensive participants.

AB, α-blockers; ARB, angiotensin II receptor blockers; BB, β-blockers; CA, calcium antagonists, D, diuretics; DBP, diastolic blood pressure; IEC, converting enzyme inhibitor; SBP, systolic blood pressure.

Fig. 1



Reactive rises in systolic (upper panels) and diastolic (lower panels) blood pressure (SBP, DBP) in normotensive participants and hypertensive patients. *$P<0.05$, **$P<0.01$ arm vs. wrist. †$P<0.05$ hypertensive patients vs. normotensive participants. All values are means ± SD.

Reactive rise in blood pressure in normotensive participants

The reactive rises in SBP and diastolic BP (DBP) in normotensive participants are presented in Fig. 1. No significant difference was found between the arm and the wrist in these participants, irrespective of the level of cuff inflation. In the analysis of variance, neither the position of the cuff (arm vs. wrist) nor the level of cuff inflation (180 vs. 240 mmHg) had any statistical significant effect on the reactive rise.

Reactive rise in blood pressure in hypertensive patients

The reactive rise in SBP at 180 mmHg was 6.5 ± 3.5 mmHg (mean ± SD) at the arm and 3.8 ± 2.1 mmHg at the wrist ($P < 0.01$, arm vs. wrist). At 240 mmHg, the reactive rise in SBP was 6.4 ± 3.5 mmHg at the arm and 4.7 ± 3.0 mmHg at the wrist ($P = 0.01$, arm vs. wrist). The rise in DBP at 180 mmHg was 2.8 ± 1.5 mmHg at the arm and 1.6 ± 1.3 mmHg at the wrist ($P < 0.01$, arm vs. wrist). At 240 mmHg, it was 2.7 ± 1.6 mmHg at the arm and 2.0 ± 1.4 mmHg at the wrist ($P < 0.05$, arm vs. wrist) (Fig. 1). At 180 mmHg, the reactive rise in BP measured at the arm was significantly higher in the hypertensive group than in the control group ($P < 0.05$, hypertensive vs. normotensive). In the two-way analysis of variance, a significant effect of the site of cuff inflation was found (arm vs. wrist, $P < 0.01$), but no significant effect of the level of cuff inflation was observed.

As shown in Fig. 2, the reactive rise in BP upon cuff inflation was of comparable magnitude, irrespective of the baseline SBP of the patients or the control participants. Thus, the response to cuff inflation was independent of the level of BP itself, and no correlation was found between the baseline BP and the reactive rise in BP. In both groups of participants, the rise in BP was also independent of body mass index or the upper arm or wrist circumferences.

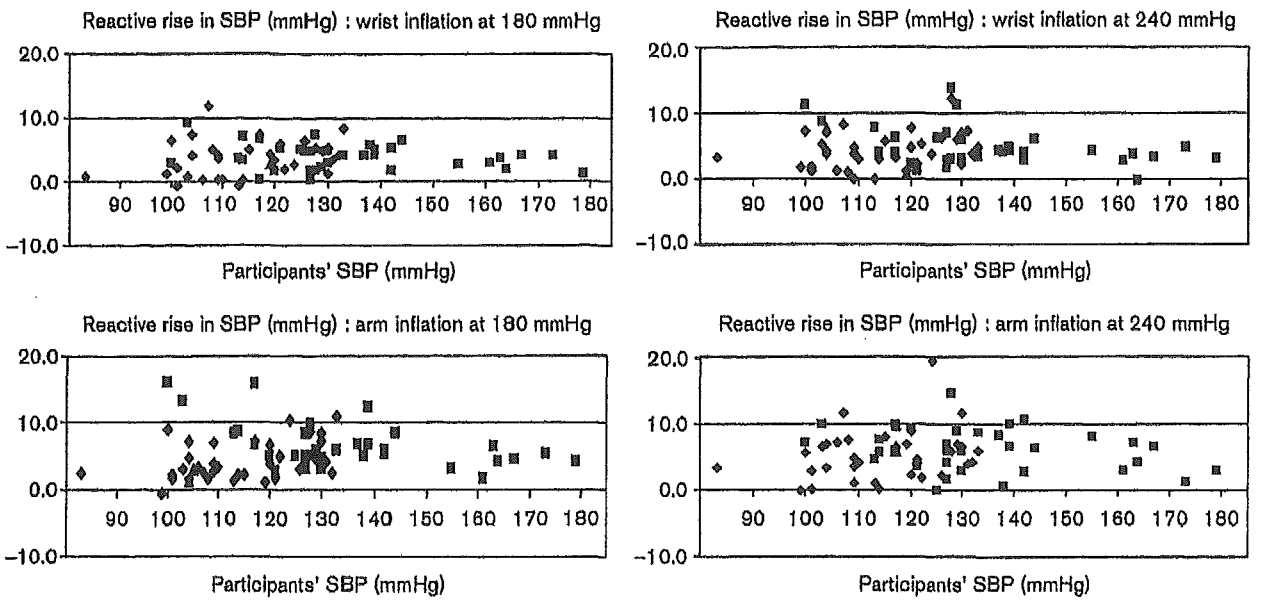
Effect of drug treatments

The majority of patients (30/34) were receiving an antihypertensive treatment at the time of investigation. As β-blockers could interfere with the response of the sympathetic nervous system, we analyzed separately the data of patients treated with β-blockers ($n = 14$) and those without β-blockers ($n = 18$). As shown in Table 2, the reactive response of SBP to cuff inflation at the arm tended to be lower in patients receiving β-blockers, although the difference did not reach statistical significance. When measured at the wrist, there was no effect of the β-blocker. The difference between the wrist and the arm was observed in both groups. Similar results were obtained with the DBP (data not shown).

Discussion

Sphygmomanometry is the gold standard method for measuring BP. With this method, a cuff is inflated to a pressure of 30 mmHg above the expected SBP at the

Fig. 2



Reactive rises in blood pressure (BP) according to baseline BP in normotensive and hypertensive participants during wrist inflation (upper panels) and arm inflation (lower panels).

Table 2 Impact of β-blockers on the reactive rise in blood pressure upon cuff inflation in hypertensive patients

Change in systolic BP (mmHg)	With β-blocker (n=14)	Without β-blocker (n=18)
Arm		
180	5.3±2.3	6.9±3.6
240	5.7±2.3	6.9±4.1
Wrist		
180	3.5±1.9**	3.7±2.0**
240	4.8±3.2	4.6±2.2*

Values are means±SD; BP, blood pressure; *P<0.05, **P<0.01 wrist vs. arm.

arm level, and the SBP and DBP are determined by the auscultation of Korotkoff sounds [16]. It is well established that this method has multiple potential limitations and sources of errors, which might sometimes lead to a misclassification of individuals as hypertensive. In recent years, new devices have been developed that enable clinicians to measure BP at the level of the wrist [10,11]. The main possible source of error in these devices is related to the position of the wrist relative to the heart [12,13,17,18]. Wrist devices are often appreciated by patients because they are small, simple to use and comfortable, even if a very high pressure has to be exerted on the wrist because the patient has a high arterial BP.

Previous studies have shown that inflating one's cuff at the arm for a BP measurement results in a transient rise in BP [19,20], which might be due to the muscle activity in the case of self measurement [21], or to muscle compression [22] as well as pain or discomfort [23] and to the overall stress of knowing that BP was being recorded [24]. As a result, Veerman *et al.* [19] have reported that cuff inflation at the arm causes an instantaneous rise in SBP of about 12 to 13 mmHg in hypertensive as well as normotensive participants, which might essentially affect the determination of SBP, as it occurs within the first 20 s of BP measurement. As inflating a cuff at the wrist level might cause less discomfort and muscle compression than at the arm level, we have investigated the impact of cuff inflation at the wrist in normotensive participants and hypertensive patients using the same methodology as the one used by Veerman *et al.* [19]. In contrast to these investigators, however, we have assessed two levels of cuff inflation (180 and 240 mmHg), the sequence of each level being randomized. As observed by Veerman *et al.* [19], we found that cuff inflation at the arm induces a rapid and transient rise in SBP and DBP both in normotensive participants and hypertensive patients, but that the magnitude of this rise was less important, that is, 4–6 mmHg for SBP and 2–4 mmHg for DBP. Whereas the reactive rise in DBP was comparable in normotensive participants and hypertensive patients irrespective of the level of cuff inflation at the arm level, the systolic response to the cuff pressure tended to be greater in hypertensive patients than in controls. The difference was significant when the cuff was inflated at 180 mmHg (P < 0.05) but not at 240 mmHg. In normotensive participants, the BP reactivity was

comparable if the cuff was inflated at the arm or at the wrist. In hypertensive patients, however, both the DBP and SBP responses to cuff inflation were significantly lower at the wrist than at the arm. When the cuff was inflated at the wrist, the BP changes were comparable in normotensive participants and hypertensive patients.

The significant difference between the BP response to cuff inflation at the arm and at the wrist in hypertensive patients cannot be explained on the basis of our data. Several studies have, nevertheless, suggested that hypertensive patients and, in particular, patients with borderline hypertension, have an increased responsiveness to experimental and daily life stresses, which might be due to an increased sensitivity of the sympathetic nervous system [25,26]. Consequently, the alarm reaction and white coat effect might be greater in hypertensive patients than in normotensive participants. Moreover, our hypertensive patients were older and heavier than controls. Although the impact of age is not clear, excess weight and obesity have also been associated with an increased sympathetic nerve activity, which could contribute to the enhanced responsiveness of BP to cuff inflation [27]. Interestingly, Fig. 2 demonstrates that the reactive rise in BP upon cuff inflation does not depend on the level of baseline BP either in normotensive participants or hypertensive patients. Thus, the observed difference is not due to the fact that hypertensive patients had a higher baseline BP. In fact, many of these patients had a well-controlled BP at the time of investigation and, if anything, the BP responsiveness tended to be higher at low levels of baseline BP than at the higher levels. Our data would, therefore, rather suggest that hypertensive patients have a greater reactivity to the inflation of a cuff at the arm even though they are more used to this procedure than control participants. Whether the speed of inflation of the cuff, which might be different at the wrist and at the arm, plays a role in the differences observed in hypertensive patients cannot be ascertained, as the inflation speed was not controlled. This, however, did not apparently affect the responsiveness in normotensive participants.

Most of our hypertensive patients were treated with antihypertensive agents, which, for ethical reasons, were not interrupted during the investigation. As drugs are potential confounding factors that might contribute to the observed difference between hypertensive patients and normotensive participants, we have analyzed the impacts of these agents on the BP responsiveness to cuff inflation. As shown in Table 2, β-blockers appear to attenuate the BP response to cuff inflation at the arm but have no impact when BP is measured at the wrist. When the same analysis was performed with the other antihypertensive drugs, no significant impact was observed.

In summary, the results of this study suggest that cuff inflation at the wrist induces less discomfort than at the arm level and hence induces a smaller reactive rise in BP even at high pressures. The difference between the arm and the wrist was observed essentially in hypertensive patients; however, it seems to be independent of the patient's level of BP. Our data did not investigate the impact of the difference in the reactive rise in BP on the determination of BP itself. As suggested by Veerman *et al.* [19], however, depending on the deflating rate, the response to cuff inflation at the arm might falsely increase SBP because the first Korotkoff sounds will be heard before systolic pressure has dropped back to baseline. In this respect, cuff inflation at the wrist using a validated device might cause less interference with SBP and might be a useful alternative in patients in whom cuff inflation at the arm produces pain or discomfort, as, for example, in obese patients or patients with severe hypertension.

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