

**Title: Changes in Central 24-hour Ambulatory Blood Pressure and Hemodynamics 12 months After Bariatric Surgery. The BARIHTA Study.**

**Short Title: 24h-BP AND BARIATRIC SURGERY**

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## Abstract

**Background:** Weight loss is associated to blood pressure (BP) reduction in obese patients. There is no information on central 24-hour BP changes after bariatric surgery (BS).

**Methods and Results:** In this study, we analyzed changes in 24h-BP 12 months following BS, with intermediate evaluations at 1,3 and 6 months, in severely obese adults. The primary endpoint was aortic (central) 24h-systolic BP changes. Circadian BP patterns and hypertension resolution were also assessed. As secondary endpoints we analyze changes in central 24h-diastolic BP as well as in all office and ambulatory peripheral BP parameters. Obese adults scheduled for BS as routine clinical care were recruited. We included 62 patients (39% with hypertension, 77% women, body mass index,  $42.6 \pm 5.5$  Kg/m<sup>2</sup>). Reduction in body weight was mean (IQR): 30.5% (26.2-34.4) one year after BS. Mean (95%CI) change in central 24h-systolic BP was -3.1 mmHg (-5.5 to -0.7),  $p=0.01$  after adjustment for age, sex and baseline hypertensive status. BP parameters changes were different between normotensives and hypertensives. Mean (95%CI) change in central 24h-systolic BP was -5.2 mmHg (-7.7 to -2.7),  $p<0.001$ , in normotensives and -0.5 mmHg (-5.1 to 4.0),  $p=0.818$ , in hypertensives. There was a remission of hypertension in 48% of patients. Most patients had a reduced-dipping pattern, similarly at baseline and 12 months after BS.

**Conclusions:** among patients with severe obesity, there was a substantial central 24h-systolic BP decrease 12 months following BS. Importantly, this change was observed in those patients with normal BP at baseline.

**Clinical Trial Registration**—URL:<http://www.clinicaltrials.gov>. Unique identifier:NCT03115502

**Key words:** central blood pressure, bariatric surgery, obesity, cardiac output, arterial stiffness.

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## Introduction

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4 Obesity has become a global epidemic, with an estimated of nearly 500 million obese  
5 people worldwide[1,2], and is an important contributor to cardiovascular morbidity and  
6  
7 to cardiac and all-cause mortality[3]. This mortality increased risk is independent of  
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9 other associated cardiovascular risk factors[4,5] and decreases after losing weight,  
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11 especially in those patients undergoing bariatric surgery (BS), although the fully  
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13 underlying mechanisms remain misunderstood.  
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19 High blood pressure (BP) is one of the most important cardiovascular risk factors in  
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21 general population. In obese subjects, hypertension is approximately 6 times more  
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23 frequent than in lean subjects[6]. Increased body mass, hyperinsulinemia and the  
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25 increase in sympathetic activity, among others, have been pointed as the main  
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27 responsible factors.  
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31 Most studies have reported a return to normal BP values in around 40-50% of obese  
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33 hypertensive subjects after BS, without differences among the two most widely used  
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35 techniques worldwide, gastric by-pass and sleeve gastrectomy[7-10]. However, in the  
36  
37 vast majority of published studies the results rely on office BP measurements, and do  
38  
39 not report data on ambulatory BP monitoring (ABPM), even though 24h-ABPM has  
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41 **been considered a better estimate of BP, with a recognized higher value in**  
42  
43 **predicting cardiovascular events in hypertensives [11]. (reviewer #1, comment #3)**  
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48 Some of the few groups that evaluate ambulatory-BP changes after BS are small-  
49  
50 sized[12] or do not track beyond six months[13]. Moreover, there are very few  
51  
52 prospective reports addressing changes in BP after BS in normotensive patients and not  
53  
54 just hypertensives[14-16]. **On the other hand, central BP has been increasingly**  
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56 **recognized as a better estimator of the “true” BP than the traditional peripheral**  
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**BP measurement [17]. Central BP measured by non-invasive methods has shown a more closely relationship with target organ damage [18] and in most, but not all, longitudinal studies, has been found to have a better predictive value for cardiovascular events and mortality [19-21]. In all of these studies, central BP was obtained from a few number of measurements taken at the office. More recently, central BP assessed throughout 24h has become possible due to the development of several devices [22] that using pulse wave analysis at the brachial level permit simultaneously evaluate central and peripheral BP, obtaining estimates based on 24h mean values, as well as during day and night periods. Regarding changes in central BP after BS in the patients with severe obesity, the information is almost non-existent [23]. (reviewer #1, comments #1 & #3)**

**Several studies have analyzed changes in BP after BS, mainly between two points in time, before and after BS. However, there is no information on the modifications of BP evaluated in each subject over time at different points of follow-up, which could be of interest to better understand the chronology of hemodynamic changes after BS. (reviewer #1, comment #2)**

Thus, here we sought to assess changes in central 24h-ambulatory-BP values in severely obese patients undergoing BS, at different time points follow-up. Additionally, we evaluated peripheral BP changes and baseline circadian BP patterns and their changes at 12-months after BS in them.

## **Materials and Methods**

### ***Study Design and Patients***

1 The BARIHTA (Hemodynamic Changes And Vascular Tone Control After **BARI**atric  
2 Surgery. Prognostic Value Regarding **HyperT**ension And Target Organ Damage) study  
3  
4 is a prospective, observational, unicenter trial in a cohort of consecutively recruited  
5  
6 patients with severe obesity scheduled to undergo BS (clinicaltrials.gov  
7  
8 identifier:NCT03115502). The BARIHTA study enrolled outpatients attending consults  
9  
10 in the Endocrinology Department of the Hospital del Mar in Catalonia, Spain, because  
11  
12 of severe obesity and looking for surgical treatment. All participants of both sexes aged  
13  
14 between 18 and 65 years who had indication for treatment with BS because of severe  
15  
16 obesity and agreed to undergo the surgical intervention, were invited to participate.  
17  
18 According to the local protocol, patients were candidates if they had grade III obesity  
19  
20 (body mass index [BMI] >40 Kg/m<sup>2</sup>) or grade II obesity (BMI >35 Kg/m<sup>2</sup>) plus  
21  
22 associated comorbidities (type 2 diabetes mellitus, sleep-apnea syndrome, obesity-  
23  
24 associated hypoventilation disease, hypertension or dyslipidemia). Both normotensive  
25  
26 and hypertensive patients were included. Patients with any endocrine disease causing  
27  
28 obesity and/or psychiatric alteration that contraindicated surgery were discarded. Other  
29  
30 exclusion criteria comprised the exclusion of the BS program for any reason, or the  
31  
32 refusal to give informed consent. Patients received detailed information on the trial in  
33  
34 the Hypertension and Vascular Risk Unit (Nephrology Department, Hospital del Mar).  
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36 The trial was approved by the local institutional Ethic Committee in accordance with  
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38 the Declaration of Helsinki and written informed consent was obtained from all  
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1 periods, at the same time points. Finally, we assessed modifications in circadian BP  
2 profiles after BS.  
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5 Demographic and clinical data were recorded from all participants **during (reviewer**  
6 **#1, comment #5)** the inclusion visit. Height was measured at the initial visit, and other  
7 anthropometric characteristics, i.e. body weight and waist circumference, as well as  
8 laboratory tests and office- and 24h-BP recordings were obtained at baseline and **on**  
9 **(reviewer #1, comment #6)** follow-up visits 1, 3, 6 and 12 months after surgery.

10  
11 **Median and [IQR] (reviewer #1, comment #7)** number of days between  
12 measurements and surgery was 12 [7; 47]. Information on pharmacological treatment  
13 was also recorded at each visit. Hypertension was considered if patient received  
14 antihypertensive drugs and/or if the baseline peripheral 24h systolic and diastolic BP  
15 was  $\geq 130$  and/or  $\geq 80$  mmHg, respectively. Hypertension remission was considered  
16 when peripheral 24-hour-BP  $< 130/80$  mmHg was achieved in the absence of  
17 antihypertensive drugs.  
18  
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## 20 ***Procedures***

### 21 *Blood pressure measurements*

22 All BP measurements were performed by means of a noninvasive automated  
23 oscillometric device (Mobil-O-Graph PWV; IEM, Stolberg, Germany), validated for  
24 central and brachial BP measurement, according to the European Society of  
25 Hypertension International protocol[24] and using suitable sized cuffs, mostly large or  
26 very large. After BS some subjects required smaller cuffs than at baseline; therefore, in  
27 each study visit the arm circumference was measured and an appropriate sized cuff was  
28 accordingly used. The monitor was placed on a working day, starting between 08:00  
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1 and 10:00h A.M. After 5-min rest, BP was consecutively determined four times at 1-  
2 min intervals and the mean was settled as office BP. Thereafter, BP was recorded  
3  
4 automatically at 20-min intervals throughout both the awake and asleep periods, as  
5  
6 defined in the patients' diary. All included patients had recordings of good technical  
7  
8 quality (valid readings  $\geq 70\%$ ). **If the recording did not reach 70% of valid readings,**  
9  
10 **(reviewer #1, comment #8) a new** ambulatory BP monitoring (ABPM) was repeated  
11  
12 within 1 week **and used as the valid one.** According to the European Society of  
13  
14 Hypertension guidelines[11], circadian patterns were defined as follows: *dipping*:  
15  
16 nocturnal BP fall  $>10\%$  of daytime values; *reduced dipping*: nocturnal BP fall from 1 to  
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18  $10\%$  of daytime values; *nondipping* and *rising*: no reduction or increase in nocturnal  
19  
20 BP, and *extreme dipping*: nocturnal BP fall  $>20\%$ .

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26 The Mobil-O-Graph PWV<sup>®</sup> device also allowed an indirect non-invasive calculation of  
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28 several cardiovascular variables including total peripheral resistance and cardiac output  
29  
30 from an arterial pressure. Regarding pulse wave velocity (PWV), as validated by  
31  
32 referring to tonometric devices and/or invasively obtained values[25], the ARC Solver  
33  
34 method (algorithm) allows to calculate PWV using data derived from pulse wave  
35  
36 analysis and wave separation analysis. Cardiac output was defined as the product of  
37  
38 stroke volume and heart rate (L/min). Total peripheral resistance was calculated as the  
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40 ratio of mean arterial pressure to cardiac output, assuming zero venous pressure (at the  
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42 right atrium) (mmHg/mL).  
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### 53 *Surgical Techniques*

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56 Either laparoscopic Roux-in-Y gastric bypass (LRYGB) or laparoscopic sleeve  
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58 gastrectomy (LSG) were chosen for each patient based on clinical criteria and the  
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1 consensus of the Bariatric Surgery Unit. Thus, LSG was preferred in younger patients,  
2 in those with BMI in the 35-40kg/m<sup>2</sup> range, as a first-step treatment in cases with a BMI  
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4 >50kg/m<sup>2</sup> and when drug malabsorption was to be avoided[26].  
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## 10 STATISTICAL ANALYSES

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12 Ordinary statistical methods were applied with statistical package SPSS for Windows  
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14 version 25.0 (Cary,North Carolina,USA). Briefly, variables following normal  
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16 distribution are summarized as mean±S.D. and categorical data are presented as  
17  
18 frequencies and percentages. Comparisons of analyzed variables between two observed  
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20 periods were carried out by paired t-tests in continuous normally distributed data, by  
21  
22 nonparametric Wilcoxon test in asymmetrically distributed data, or by  $\chi^2$ -test in  
23  
24 categorical data. Spearman's approach was used to calculate correlations between time-  
25  
26 variation of two variables. Repeated measures on continuous variables were assessed by  
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28 the one-way analysis of variance with repeated measures or through Generalized  
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30 Estimating Equations (using STATA package version 15 [STATA Corp.,Texas,USA]),  
31  
32 and changes in the different BP parameters were adjusted by age, sex, and baseline  
33  
34 hypertensive status. . A change was considered significant if the two-side alpha level  
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36 was  $\leq 0.05$ . A sample size calculation showed that accepting an alpha risk of 0.05 and a  
37  
38 beta risk <0.2 in a bilateral contrast, 61 subjects are required to detect a difference equal  
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40 to or greater than 3 mmHg of central 24h-SBP. A standard deviation of 8 is assumed. It  
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42 was estimated a rate of tracking losses of 10%.  
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## 58 **Results**

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1 In total, two hundred subjects were evaluated. Bariatric surgery was ruled out in 22 of  
2 them for medical reasons. Another 49 patients rejected BS or to participate in this study.  
3  
4 Bariatric surgery was performed in 127 subjects. Eighteen were discarded because of  
5 lack of valid baseline analyses, and 49 others were lost to follow up. A complete  
6  
7 evaluation is available for the remaining 62 patients and these constitute the final cohort  
8  
9 of the BARIHTA study. Baseline clinical characteristics are shown in Table 1. Thirty-  
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11 nine percent of the included patients had hypertension. None of the patients died at  
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13 follow-up.  
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#### 23 *Variation of body weight, body mass index and waist circumference*

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26 Overall, there was a mean (IQR) 30.5% (26.2-34.4) reduction in body weight one year  
27 after BS. Body mass index was  $42.7 \pm 5.6$  Kg/m<sup>2</sup> and  $29.7 \pm 4.8$  Kg/m<sup>2</sup>, at baseline and 12  
28 months after BS, respectively ( $p < 0.001$ ). Figure S1 (Supplemental material) shows  
29 mean(95% CI) body weight in each follow up point. Waist circumference was  
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**12-months variation of peripheral 24h-SBP, there was a statistically significant correlation with variation of both body weight ( $\rho=0.453$ ,  $p=0.001$ ) and waist circumference ( $\rho=0.316$ ,  $p=0.030$ ).**

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3 *Changes in central and peripheral blood pressure*  
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6 Baseline values of central and peripheral BP are compared in Table S1 (Supplemental  
7 material). Changes in office and ambulatory BP, both central and peripheral, are shown  
8 in Table 2. Central 24h-SBP decreased at 12 months, mean (95% confidence interval) -  
9 3.1 mmHg (-5.5 to -0.7), p=0.01 after adjustment for age and sex. As shown, both  
10 central and peripheral SBP significantly decreased 12 months after BS when evaluated  
11 in the 24-hour and daytime periods, whereas there was no decrease in nighttime SBP.  
12  
13 Diastolic BP (DBP) significantly decreased only in the central, but not peripheral,  
14 daytime period. Otherwise, office systolic and diastolic BP, both central and peripheral,  
15 significantly decreased at 12 months.  
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19 Results on BP changes 12 months following BS are shown separately for normotensive  
20 and hypertensive subjects (Table 2). In normotensive subjects there was a statistically  
21 significant decrease in all central and peripheral BP parameters, except in nighttime-  
22 DBP, both when analyzing raw data and after sex- and age- adjustments. These changes  
23 were not statistically significant in the hypertensive patients. Accordingly, evolution of  
24 BP is shown in Figure 1A (for the whole cohort) and Figures 1B and 1C (for  
25 hypertensives and normotensives, separately). This decrease in BP was observed as  
26 early as 1 month after BS and was sustained at 12 months follow-up in the whole cohort  
27 (Figure 1A). The decrease in SBP lost the statistical significance at 12 months in  
28 previous hypertensive subjects (Figure 1B) but not in those with baseline normal BP  
29 values (Figure 1C). **Table S2 shows similar changes for all BP estimates at 1 month**  
30 **in both normotensives and hypertensives. One month after BS, the number of**  
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3 **antihypertensive drugs did not change with respect to baseline (1.1±1.0 and**  
4 **1.6±1.1, respectively, p=NS).**

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6 There was a remission of hypertension in 48% of patients who at baseline had a  
7  
8 peripheral 24h-systolic and/or diastolic BP  $\geq$ 130/80mmHg, or who were taking  
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10 antihypertensive drugs. Figure S2 shows the number of antihypertensive drugs  
11  
12 prescribed in initially hypertensive patients, mean number being 1.6±1.1 and 0.4±0.6 at  
13  
14 baseline and 12 months follow-up, respectively (p<0.001). The proportion of  
15  
16 hypertensive patients receiving antihypertensive drugs was 82.6% and 34.8% at  
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18 baseline and 12 months after BS, respectively.  
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23 BP changes did not significantly differ according to the type of surgical technique  
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25 (Table S3).  
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29 Table 3A shows the changes of both central and peripheral 24h-SBP at the different  
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31 time points (1, 3, 6 and 12 months) according to the Generalized Estimating Equations,  
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33 with adjustment for age, sex and baseline hypertensive status.  
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38 Table 3B shows the 24h-SBP changes at the different observed time points after  
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40 adjustment by age, sex, baseline hypertensive status and body weight variation,  
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42 demonstrating that these two latter determine the changes of SBP.  
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#### 48 49 *Circadian patterns of blood pressure* 50

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52 Regarding circadian patterns of BP, patients were classified at baseline and at 12  
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54 months after BS, according to the consensus document of the European Society of  
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56 Hypertension on 24h-ambulatory BP monitoring[18]. At these two time-observed  
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58 periods, the corresponding circadian pattern was attributed to each patient according to  
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1 both systolic and diastolic BP and to either peripheral or central BP. At baseline, 42%  
2 and 56% of subjects had a dipper pattern and a reduced dipper pattern, respectively,  
3  
4 when considering peripheral SBP. There was no significant change in the distribution of  
5  
6 patients according to circadian patterns between baseline and 1 year after BS for any of  
7  
8 the parameters evaluated (data not shown). Fourteen patients had sleep-apnea syndrome  
9  
10 at baseline and 11 out of these 14 patients received continuous positive airway pressure  
11  
12 (CPAP) treatment. At 12 months follow-up after BS, 10 patients did not receive CPAP  
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14 and this information is missing for the 4 remaining patients. There were no differences  
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16 in circadian patterns between both periods for this subgroup of patients (p=NS).  
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#### 26 *Arterial stiffness and hemodynamic parameters*

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29 Patients with hypertension at baseline had higher 24h-PWV and higher systemic  
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31 vascular resistance values than normotensives (Supplemental material, Table S4).  
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35 No statistically significant change in 24h-PWV was observed at 12 months after BS  
36  
37 (Table 4). Another parameter of arterial stiffness, the augmentation index corrected at  
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39 75 beats per minute (AIx@75), decreased at 12 months (Z= -1.92; p= 0.055). When the  
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41 one-way analysis of variance with repeated measures was applied to assess changes in  
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43 these variables at different time points, a significant decrease in both of them was  
44  
45 observed (p=0.001 for PWV and p=0.005 for AIx@75). The decrease in IAx@75 was  
46  
47 only statistically significant in the hypertensive group. Evolution of 24-hr PWV and of  
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49 IAx@75 is shown in Figure 2A.  
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55 Regarding hemodynamic parameters, there was a statistically significant decrease in  
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57 cardiac output (mean change -0.17L/min, 95%CI -0.30 to -0.03, p=0.015), maintained  
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59 after adjusting by age, sex, baseline hypertensive status and baseline cardiac output: -  
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0.17L/min (-0.27 to -0.06), adjusted  $r^2=0.343$ ,  $p=0.034$  (Figure 2C). There was a statistically significant correlation between change in central 24h-SBP and variation of cardiac output at 12 months ( $Rho=0.454$ ,  $p=0.001$ ). No changes in systemic vascular resistance were observed.

## Discussion

The main finding of the BARIHTA study is that in severely obese subjects, the BS produces a significant decrease in central (aortic) BP, a decrease that occurs not only in those with hypertension prior to surgery, but also in those with confirmed normotension. Indeed, the decrease in SBP was observed as early as 1 month after BS but was not sustained at 12 months follow-up in previous hypertensive subjects, probably due to the withdrawal or reduction in the number of antihypertensive drugs after BS in most of them. **As observed, the changes in most of the BP estimates were statistically significant in both normotensive and hypertensive subjects one month after the BS, when the modification of antihypertensive therapy was still not relevant.**

With respect to BP changes after BS, several studies have shown a BP decrease with an improvement and even normalization of BP levels[27,28]. However, this reduction of BP values was not confirmed by other groups, such as the Swedish Obesity Study[29]. It must be highlighted that these studies are based on office BP measurement, and studies with evaluation of ABPM changes, a BP measurement more reliable and with better cardiovascular risk prognostic value as shown in general population[30,31], are very scarce and small-sized in severely obese patients following BS. In these few studies, the results on 24-hour BP changes in severely obese patients after BS are controversial. In a report on 20 patients undergoing laparoscopic gastric bypass, a

1 statistically significant decrease on BP at 10 days was observed both in hypertensive  
2 and normotensive patients[14]. However, more recently Głuszevska et al. reported a  
3 significant decrease in 24h- systolic and diastolic BP in patients with baseline  
4 hypertension, but not in those with normal BP values[13]. This is in accordance with a  
5 previous report, where the authors also found that the reduction in BP after BS was  
6 evident only in hypertensive patients[32]. Importantly, we have demonstrated that there  
7 is an early (one month) and sustained (for 12 months) significant decrease in BP in  
8 patients with baseline normotension, as confirmed by 24h-ABPM.  
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20 Over recent years, several articles documented a preponderant role played by BS on  
21 SBP reduction, but a diminished effect on decreasing of DBP, mostly after several  
22 months[12,13]. Of note, we have found a significant decrease in SBP but not in overall  
23 DBP when analyzing the whole cohort; however, there is a statistically significant  
24 reduction in both central and peripheral 24h- and daytime DBP in normotensive obese  
25 subjects. This finding remarks the importance of focusing the analyses of BP changes  
26 on patients with normotension and perhaps with non-treated hypertension, in order to  
27 better understand the underlying mechanisms between obesity, BP, vascular changes,  
28 and the effects of weight loss.  
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42 On the other hand, several recent studies have suggested that measuring central BP  
43 could be a better predictor of poor cardiovascular outcomes than peripheral BP[19]. To  
44 our knowledge, our study is the first one showing changes in central BP 12 months after  
45 BS. Although 24h-SBP at baseline was, as usual, lower in central than in peripheral BP,  
46 the decrease in 24h-SBP was statistically significant in both of them. In fact, the  
47 reductions in ambulatory BP values seem to be of higher magnitude for central than for  
48 peripheral BP measurements, albeit lower baseline values for central BP as mentioned.  
49 To explain this effect on central BP, it has been suggested that the imbalance between  
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1 vasodilating and vasoconstricting factors could increase peripheral vascular resistance  
2 and induce hypertension in obesity and be responsible for increased SBP[33]. This  
3  
4 could be in line with our findings, with a higher effect of this imbalance on central than  
5  
6 peripheral BP, although this hypothesis deserves confirmation by further studies.  
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10 Regarding BP circadian patterns, **subjects with reduced dipping status (as assessed**  
11 **by a nighttime average BP <10% of the daytime average BP or a night-to-day**  
12 **ratio >0.9) or those in whom there is no night-time dip in BP or have a higher**  
13 **night-time than daytime average BP, have a substantially increased**  
14 **cardiovascular risk[11].** It has been highlighted the higher incidence of nondipping  
15 status in obesity[34]. In our cohort we found 42% of patients with a normal dipper  
16 pattern, which is in agreement with this previous report. Recent accurately reported data  
17 showed a lack of changes in dipping status in a cohort of 90 patients six months after  
18 BS[13], which is similar to our findings. In the same line, Careaga and col.[15] did not  
19 observe changes in dipping status in a smaller cohort of patients 1 year after BS. **This**  
20 **consistent maintenance of a non-dipping status after BS suggest that the dipping**  
21 **status in these subjects is not determined by the increased body weight itself but**  
22 **other mechanisms, or that not only the body weight loss but to reach normal**  
23 **weight should be necessary to achieve a normal dipping pattern. However these**  
24 **are mere speculations that would deserve future in depth research.**  
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48 **We have found a statistically significant correlation between 12 months change in**  
49 **both central 24h-SBP and body weight, but not with change in waist**  
50 **circumference. Previously it was reported that in obese population BMI is**  
51 **associated with peripheral and central SBP, whilst waist circumference is**  
52 **significantly correlated with peripheral but not central SBP[35]. In addition, in a**  
53 **cohort of youth, central BP was found to be elevated among individuals with**  
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1 **severe obesity and associated with BMI but not with body fatness (total-body**  
2 **percent fat mass or visceral adipose tissue)[36]. Although these were mere**  
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4 **observational associations and central BP was estimated in an office setting by**  
5 **applanation tonometry (and not by oscillometric 24h-ABPM), these data are in**  
6  
7 **accordance with the correlations we have observed 12 months after BS, reinforcing**  
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9 **these associations. Mechanisms of high BP in severe obesity are not well-**  
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11 **established, but two with a possible important role must be pointed: increased**  
12  
13 **vascular resistance and volume-dependent hypertension.** Obesity is characterized by  
14  
15 arterial stiffness, an important factor underlying the development of hypertension. Many  
16  
17 epidemiological studies have shown a positive association between BMI and PWV,  
18  
19 independent of age and BP. Interestingly, an experimental study of diet-induced obese  
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21 mice showed that arterial stiffness preceded hypertension, suggesting that arterial  
22  
23 stiffness may be a cause rather than a consequence of hypertension[37] **and this was**  
24  
25 **supported by similar findings in participants from both the Framingham**  
26  
27 **Offspring Study[38] and the Baltimore Longitudinal Study of Aging[39].** It is  
28  
29 suggested that obesity causes arterial stiffening as a result of an increase in  
30  
31 inflammation and vascular remodeling molecules and a change in endothelial function.  
32  
33 A meta-analysis of 20 studies showed a significant reduction in arterial stiffness  
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35 following weight loss[40]. **Accordingly, it has also been found that the reduction in**  
36  
37 **arterial stiffness correlates with adipokine secretion and inflammation[41]. Thus,**  
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39 **the reduction of BP after BS in patients with severe obesity could be related to its**  
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41 **effect on arterial stiffness by modifying adipokines and inflammatory pathways.** In  
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43 accordance with those previous reports, our study shows a trend to a decrease in PWV  
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45 and AIx@75 over time, recognized markers of arterial stiffness, even though the follow  
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47 up time to observe changes in PWV is perhaps too short. **On the other hand,** patients  
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1 with obesity have an increased cardiac output[42]. We have found a significant decrease  
2 in cardiac output 12 months after BS. Some decades ago it was suggested that the  
3 decrease on BP after weight loss in obese patients could be attributable to reductions in  
4 total circulating and cardiopulmonary blood volume[43]. Recently, a small study in  
5 fifteen women showed a decrease in BP six weeks after BS associated with a decrease  
6 in cardiac output, as noninvasively measured with a Nexfin® device [23]. Here we  
7 confirm this relationship between BS and a decrease in 24h-ambulatory cardiac output  
8 in both men and women 12 months after BS. **Although both mechanisms for**  
9 **hypertension probably coexist in obesity, the relationship of change in BP with**  
10 **change in BMI but not with waist circumference perhaps would suggest a great**  
11 **predominance of volume-dependent hypertension rather than vascular resistance**  
12 **as the main mechanism of BP changes in relation to obesity. We hypothesize that**  
13 **central and not peripheral BP may contribute to the knowledge of the**  
14 **physiopathology of hypertension in obesity, since the central SBP can more**  
15 **accurately reflect the variation in cardiac output.**

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**In light of our results, the evaluation of the central BP is not superior to the**  
**peripheral BP to assess changes in BP in severely obese patients undergoing BS**  
**and, therefore, its use as routine clinical practice should not be recommended.**  
**However, we can not rule out that measuring central BP may be of interest to**  
**deepen the pathophysiology of hemodynamic changes in these patients, perhaps**  
**seeking relationships with adipokines, inflammatory markers or other pathways.**

Our study has strengths and limitations to be addressed. Firstly, most patients are women, and because of the sample size, sex-separated analysis are unfeasible. However, this sex imbalance is in accordance with the majority of reported BS series. Secondly, this is a single-center study; moreover, it lacks a control group. However, the huge

1 amount of measures recorded at various times prevents obese people who will not  
2 undergo BS from participation. Thirdly, cardiac output and peripheral resistance as well  
3 as central BP were not really measured, but derived from BP, therefore this information  
4 should be evaluated with caution. **In this sense, it is worth mentioning that the**  
5 **majority of parameters are derived directly from the same raw data** (reviewer #1,  
6 **comment #9**). However, since the same method was applied in the different time  
7 points, changes should be considered as valid. This last reason applies also to the  
8 limitation regarding the method used to calculate PWV and AIX@75. Thus, Mobile-O-  
9 Graph® device could underestimate the PWV measurement, although there is an  
10 acceptable agreement with this marker obtained by other validated methods[44].

11 However, we honestly consider that changes evaluated at 5 different time points with  
12 the same device at each visit add value to our findings. Finally, the follow up is 12  
13 months, and we cannot extrapolate the results in the longer term. The strengths include  
14 the accuracy of the assessment of BP and hemodynamic parameters by specialized  
15 nurses, the several time-point observations, and, specially, the novelty of providing data  
16 for the first time on the changes of 24 hours-central BP after BS.

17 In conclusion, a decrease in central 24h-SBP is observed in normotensive subjects 12  
18 months after BS. All other central and peripheral BP parameters lowered in relation to  
19 BS in severely obese patients, except nighttime DBP. The accuracy of the data provided  
20 by this measure of BP, i.e., ambulatory-BP monitoring, increases the value of our  
21 findings. **These** changes in central ambulatory BP occur even in subjects with strictly  
22 normal BP prior to surgery. Moreover, we demonstrate a decrease in central BP at 1  
23 year for the first time.

## Perspectives

The decrease in central ambulatory BP one year after BS, as well as the reduction in cardiac output and arterial stiffness, are interesting findings that may have to do with the normalization of hemodynamic, metabolic or inflammatory changes underlying obesity beyond BP levels, although additional studies would be required to confirm this hypothesis. The modification in the central BP values may be a point in which to focus future studies on BP, obesity and surgical treatment, not only in hypertensives but also in normotensives. Deepening the mechanisms of the reduction of central BP and its comorbidities after BS could improve the knowledge of pathophysiology and the treatment of severe obesity.

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## Compliance with Ethical Standards

**Conflict of Interest** All authors have completed the ICMJE uniform disclosure form and declare that they have no conflict.

**Ethical Consideration** The trial was approved by the local institutional Ethic Committee in accordance with the Declaration of Helsinki and written informed consent was obtained from all participants.

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## Figure Legends

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6 **Figure 1. 1A.** Change in central 24h- systolic blood pressure in the whole cohort. **1B.**  
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8 Change in central 24h- systolic blood pressure in **hypertensive** patients. **1C.** Change in  
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10 central 24h- systolic blood pressure in **normotensive** patients.  
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18 **Figure 2. 2A.** Change of 24-hour **pulse wave velocity** (PWV) at different time points  
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20 for the whole cohort. **2B.** Change of **augmentation index** (AIx) at different time points  
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22 for the whole cohort. **2C.** Change of **cardiac output** at different time points for the  
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24 whole cohort.  
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31 **Figure S1.** Mean body mass index (with 95% confidence intervals) at baseline and at 1,  
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33 3, 6 and 12 months after bariatric surgery.  
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41 **Figure S2.** Proportions of patients according to the prescribed number of  
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43 antihypertensive drugs, at baseline and 12 months after bariatric surgery.  
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**Table 1.** Baseline characteristics.

<b>Variable</b>	
<b>Age, yr (mean±S.D.)</b>	42.1 ± 9.3
<b>Sex, women</b>	48 (77.4%)
<b>Race:</b>	
<b>- Caucasian</b>	56 (90.3%)
<b>- Black</b>	1 (1.6%)
<b>- Hispanic</b>	5 (8.1%)
<b>Current smokers</b>	17 (27.4%)
<b>Body mass index, Kg/m<sup>2</sup> (mean±S.D.)</b>	42.6 ± 5.5
<b>Waist circumference, cm (mean±S.D.)</b>	132.5 ± 12.0
<b>Surgical procedure:</b>	
<b>- Sleeve gastrectomy</b>	27 (43.5%)
<b>- Roux-en-Y gastric bypass</b>	35 (56.5%)
<b>Hypertension</b>	24 (38.7%)
<b>Type 2-Diabetes Mellitus</b>	7 (11.3%)
<b>Previous cardiovascular disease</b>	3 (4.8%)
<b>Sleep apnea syndrome</b>	14 (22.6%)
<b>- CPAP, n (% of those with sleep apnea syndrome)</b>	11 (78.6%)

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CPAP = continuous positive airway pressure

**Table 2.** Changes in office and ambulatory blood pressure, both central and peripheral, 12-months after bariatric surgery.

	Change at 12 months post-BS Mean (95% CI)	<i>p</i>	<i>p</i> -adjusted*	Change at 12 months post-BS Mean (95% CI)	<i>p</i>	<i>p</i> -adjusted*
	<b>CENTRAL</b>			<b>PERIPHERAL</b>		
<b>ALL PATIENTS (n= 62)</b>						
<b>Office-SBP</b>	-7.4 (-10.4 to -4.4)	<0.001	<0.001	-7.7 (-10.9 to -4.5)	<0.001	<0.001
<b>Office-DBP</b>	-4.8 (-7.1 to -2.4)	<0.001	<0.001	-4.5 (-6.9 to -2.1)	<0.001	<0.001
<b>24h-SBP</b>	-3.1 (-5.5 to -0.7)	0.012	0.010	-2.5 (-4.9 to -0.1)	0.046	0.037
<b>24h-DBP</b>	-1.3 (-3.0 to 0.4)	NS	NS	-0.7 (-2.3 to 0.9)	NS	NS
<b>Daytime-SBP</b>	-3.2 (-5.8 to -0.7)	0.013	0.011	-3.0 (-5.6 to -0.3)	0.028	0.023
<b>Daytime-DBP</b>	-2.2 (-4.1 to -0.3)	0.021	0.02	-1.1 (-2.9 to 0.6)	NS	NS
<b>Nighttime-SBP</b>	-1.0 (-4.4 to 2.4)	NS	NS	-0.2 (-3.5 to 3.1)	NS	NS
<b>Nighttime-DBP</b>	3.0 (0.7 to 5.3)	0.011	0.014	1.7 (-1.1 to 4.5)	NS	NS
<b>NORMOTENSIVES (n= 38)</b>						
<b>Office-SBP</b>	-9.7 (-12.7 to -6.6)	<0.001	<0.001	-10.2 (-13.3 to -7.0)	<0.001	<0.001
<b>Office-DBP</b>	-6.3 (-8.1 to -4.5)	<0.001	<0.001	-5.7 (-7.4 to -4.0)	<0.001	<0.001
<b>24h-SBP</b>	-5.2 (-7.7 to -2.7)	<0.001	<0.001	-4.8 (-7.1 to -2.5)	<0.001	<0.001
<b>24h-DBP</b>	-2.5 (-4.4 to -0.6)	0.009	0.008	-2.0 (-3.6 to -0.4)	0.014	0.013
<b>Daytime-SBP</b>	-5.6 (-8.2 to -3.0)	<0.001	<0.001	-5.4 (-7.8 to -2.9)	<0.001	<0.001
<b>Daytime-DBP</b>	-3.8 (-5.8 to -1.8)	<0.001	<0.001	-2.6 (-4.2 to -0.9)	0.002	0.002
<b>Nighttime-SBP</b>	-3.9 (-7.5 to -0.2)	0.037	0.041	-3.9 (-7.4 to -0.4)	0.029	0.032
<b>Nighttime-DBP</b>	1.6 (-1.1 to 4.4)	0.238	0.288	-0.7 (-4.2 to 2.9)	0.712	0.589
<b>HYPERTENSIVES (n= 24)</b>						
<b>Office-SBP</b>	-5.0 (-10.6 to 0.5)	0.083	0.076	-5.0 (-11.0 to 1.1)	0.105	0.096
<b>Office-DBP</b>	-2.9 (-7.7 to 1.8)	0.228	0.239	-3.1 (-8.2 to 2.0)	0.238	0.259

<b>24h-SBP</b>	-0.5 (-5.1 to 4.0)	0.818	0.818	-6.4 (-4.2 to 5.0)	0.864	0.860
<b>24h-DBP</b>	-0.3 (-2.9 to 3.5)	0.860	0.850	-0.8 (-2.3 to 4.0)	0.601	0.595
<b>Daytime-SBP</b>	-0.4 (-5.2 to 4.5)	0.886	0.874	-0.1 (-5.1 to 5.1)	0.989	0.985
<b>Daytime-DBP</b>	-0.3 (-3.8 to 3.3)	0.890	0.899	0.7 (-2.9 to 4.2)	0.721	0.716
<b>Nighttime-SBP</b>	2.9 (-3.1 to 8.9)	0.348	0.339	4.8 -1.1 to 10.7)	0.110	0.102
<b>Nighttime-DBP</b>	4.7 (0.8 to 8.6)	0.019	0.019	4.8 (0.5 to 9.1)	0.029	0.028

\* adjusted for age and gender

DBP = diastolic blood pressure; SBP = systolic blood pressure

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**Table 3.** Changes in both central and peripheral 24h-systolic blood pressure at different time points, **3A:** after adjusting by age, sex and basal hypertensive status; **3B:** after adjusting by age, sex, basal hypertensive status and body weight change.

**3A**

	Change post-BS Mean (95% CI)	<i>P</i>	Change post-BS Mean (95% CI)	<i>P</i>
	<b>CENTRAL</b>		<b>PERIPHERAL</b>	
<b>1 month</b>	-6.2 (-8.7 to -3.7)	<0.001	-5.7 (-8.2 to -3.3)	<0.001
<b>3 months</b>	- 1.9 (-4.4 to 0.6)	0.133	-2.1 (-4.6 to 0.3)	0.09
<b>6 months</b>	-4.0 (-6.6 to -1.5)	0.002	-3.9 (-6.4 to -1.3)	0.003
<b>12 months</b>	-3.2 (-5.7 to -0.8)	0.010	-2.6 (-5.0 to -0.2)	0.037
<b>Normotensives</b>	-9.8 (-14.9 to -4.8)	<0.001	-9.9 (-15.1 to -4.8)	<0.001
<b>Women</b>	2.7 (-3.0 to 8.5)	NS	1.8 (-4.1 to 7.7)	NS
<b>Age</b>	-0.2 (-0.2 to 0.3)	NS	0.0 (-0.3 to 0.3)	NS

**3B**

	<b>Change post-BS Mean (95% CI)</b>	<b><i>P</i></b>	<b>Change post-BS Mean (95% CI)</b>	<b><i>P</i></b>
	<b>CENTRAL</b>		<b>PERIPHERAL</b>	
<b>1 month</b>	1.1 (-2.9 to 5.1)	NS	1.0 (-3.0 to 5.0)	NS
<b>3 months</b>	7.4 (2.7 to 12.2)	0.002	6.5 (1.8 to 11.2)	0.007
<b>6 months</b>	6.8 (1.5 to 12.2)	0.012	6.2 (0.8 to 11.5)	0.024
<b>12 months</b>	8.7 (3.0 to 14.4)	0.003	8.4 (2.7 to 14.2)	0.004
<b>Normotensives</b>	-9.5 (-14.2 to -4.7)	<0.001	-9.6 (-14.5 to -4.7)	<0.001
<b>Body weight variation</b>	-0.2 (-0.3 to -0.1)	<0.001	-0.2 (-0.3 to -0.1)	<0.001
<b>Women</b>	5.2 (-0.4 to 10.8)	0.066	4.1 (-1.6 to 9.8)	NS
<b>Age</b>	0.04 (-0.2 to 0.3)	NS	0.02 (-0.2 to 0.3)	NS

**Table 4.** Changes in arterial stiffness and hemodynamic parameters 12 months after bariatric surgery.

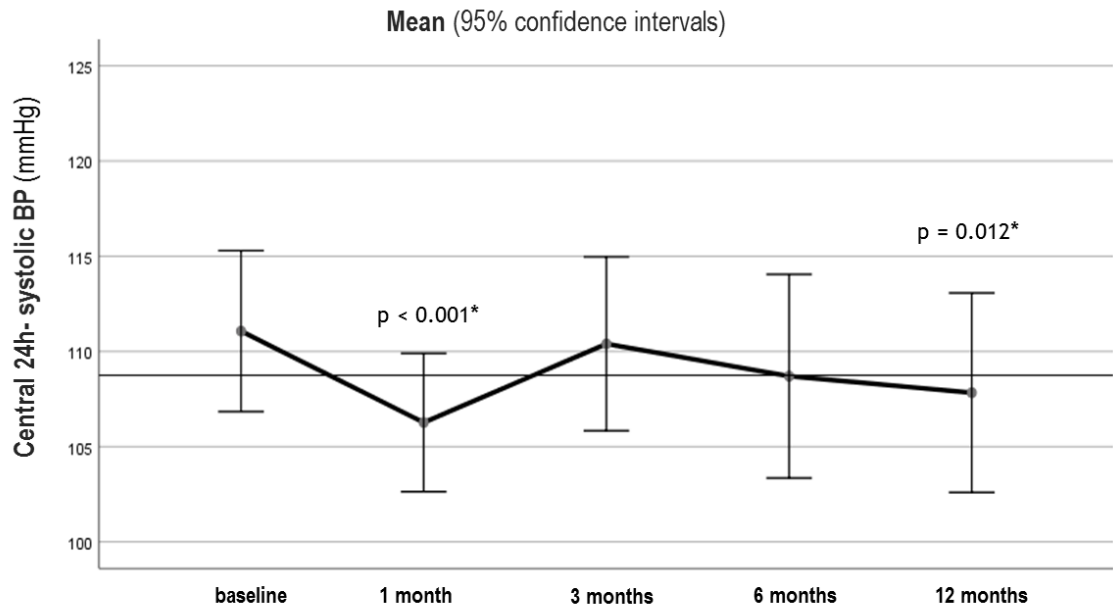
	Baseline	12 months post-BS	Change at 12 months post-BS Mean (95% CI)	<i>P</i>
<b>24-h PWV (m/s)</b>	6.69 ± 1.0	6.63 ± 1.1	-0.06 (-0.1 to 0.22)	0.441
<b>AIx@75 (%)</b>	25.9 [20.4; 25.3]*	25.1 [19.2; 31.0]*	-1.2 (-1.4 to 3.7)	0.055
<b>Cardiac output (L/min)</b>	4.66 ± 0.59	4.50 ± 0.55	-0.17 (0.03 to 0.30)	0.015
<b>Total peripheral resistance (s*mmHg/mL)</b>	1.27 ± 0.14	1.28 ± 0.15	0.01 (-0.05 to 0.02)	0.425

AIx@75 = Augmentation Index corrected at 75 beats per minute; PWV = pulse wave velocity

\* median (p25<sup>th</sup>; p75<sup>th</sup>)

Figure 1

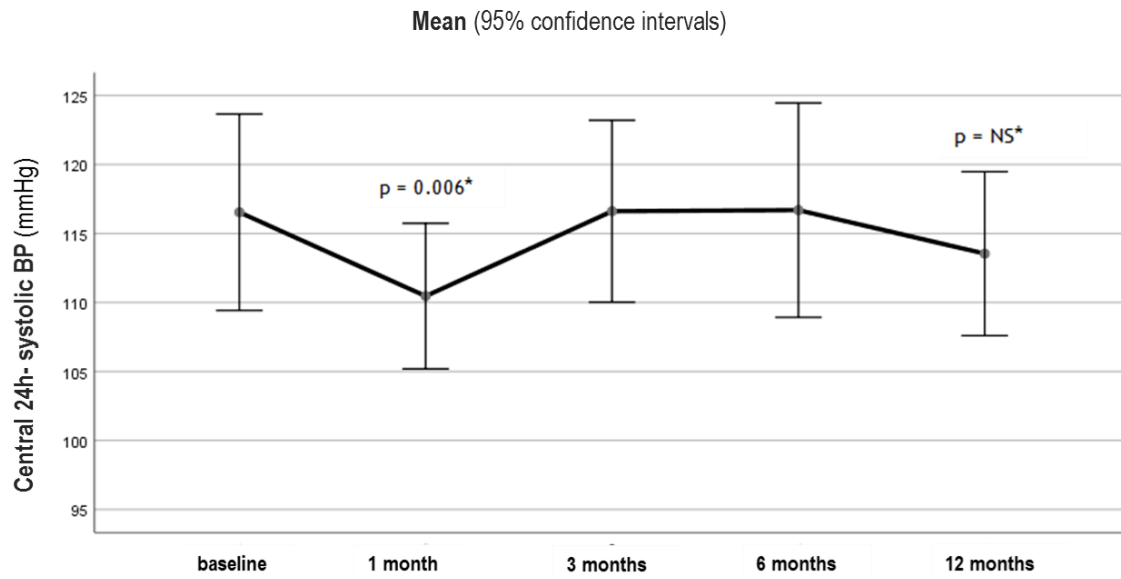
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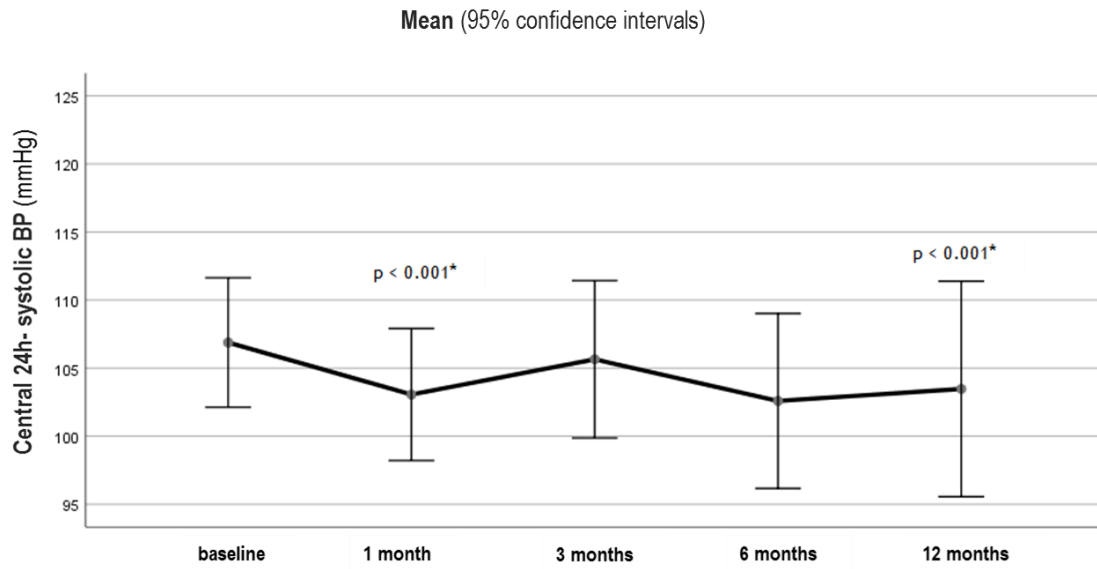
\* Unadjusted- $p$ , as compared to baseline value

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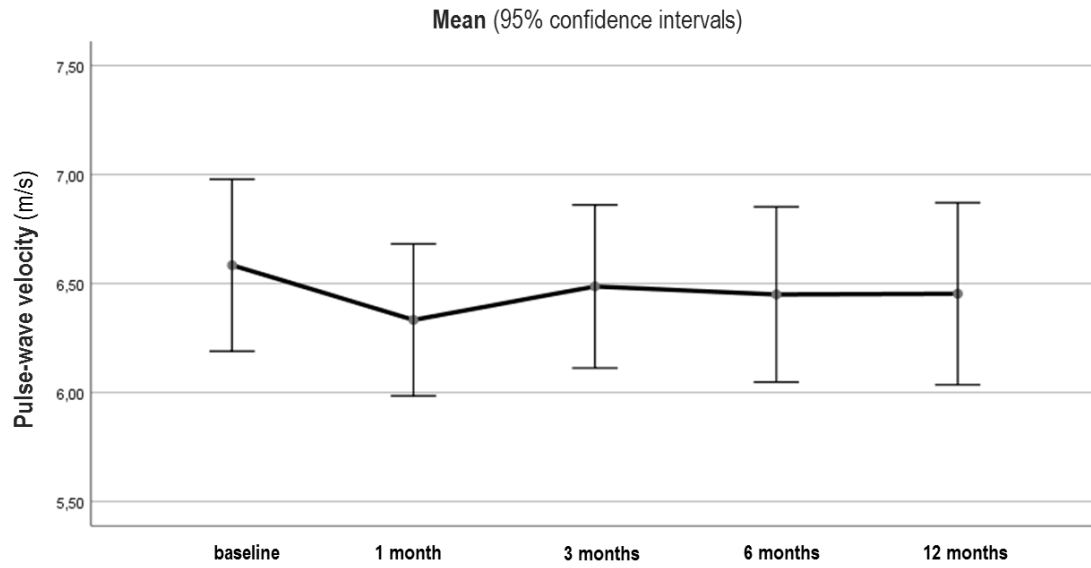
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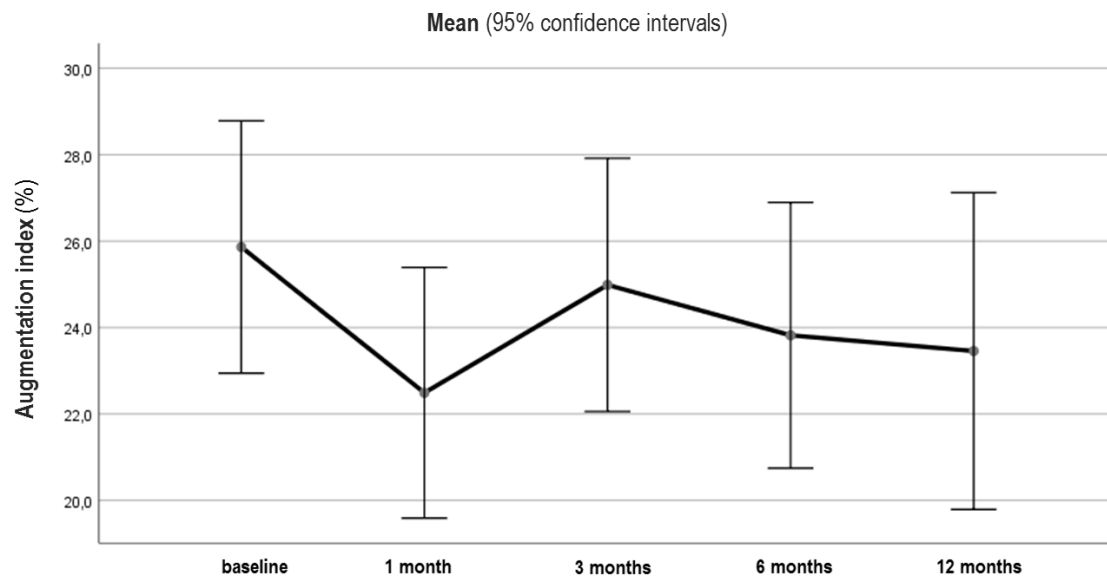
\* Unadjusted- $p$ , as compared to baseline value

Figure 2.

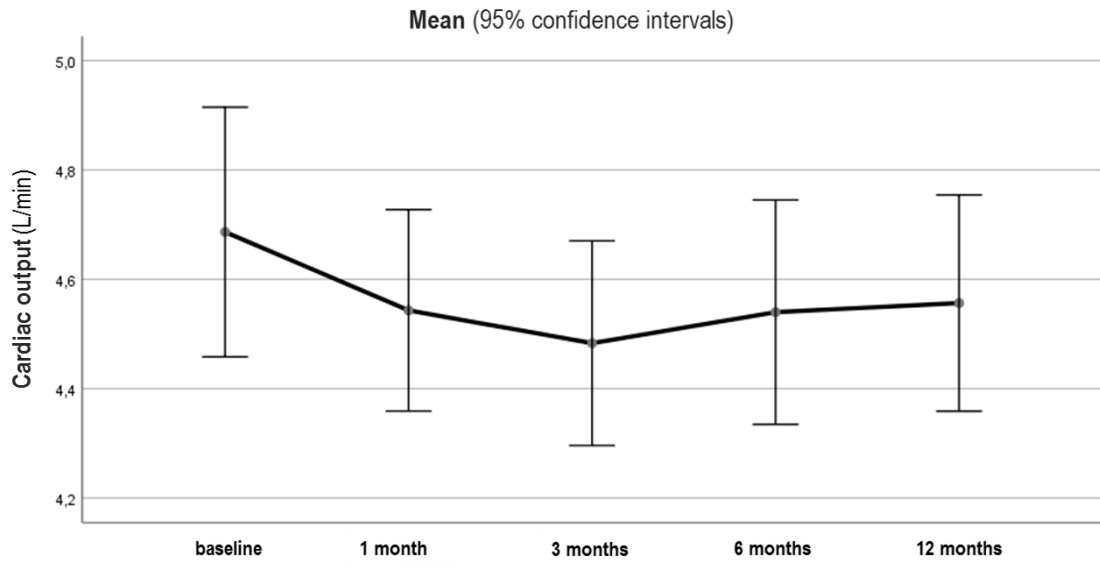
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## Online/Data Supplement

**Table S1.** Comparison of central and peripheral blood pressure (BP) parameters at baseline.

Baseline blood pressure	Central	Peripheral	<i>p</i> -value
<b>24 hour-SBP</b>	111.7 ± 10.5	120.6 ± 10.7	<0.001
<b>24 hour-DBP</b>	74.9 ± 8.4	73.3 ± 8.1	0.001
<b>Daytime-SBP</b>	114.2 ± 10.3	123.5 ± 10.8	<0.001
<b>Daytime-DBP</b>	78.9 ± 8.5	76.5 ± 8.5	<0.001
<b>Nighttime-SBP</b>	105.6 ± 12.6	112.2 ± 11.6	<0.001
<b>Nighttime-DBP</b>	65.2 ± 9.1	64.2 ± 8.6	0.077

DBP = diastolic blood pressure; SBP = systolic blood pressure

**Table S2.** Changes in office and ambulatory blood pressure, both central and peripheral, 1-month after bariatric surgery, in normotensives and hypertensives separately.

	Change at 1 month post-BS Mean (95% CI)	<i>p</i>	Change at 1 month post-BS Mean (95% CI)	<i>p</i>
	<b>CENTRAL</b>		<b>PERIPHERAL</b>	
<b>NORMOTENSIVES (n= 38)</b>				
24h-SBP	-5.9 (-9.0 to -2.7)	0.001	-5.3 (-7.7 to -2.9)	<0.001
24h-DBP	-2.5 (-4.7 to -0.3)	0.029	-2.8 (-4.4 to 1.2)	0.002
Daytime-SBP	-5.9 (-8.9 to -2.8)	0.001	-5.4 (-8.0 to -2.7)	<0.001
Daytime-DBP	-2.9 (-4.9 to -0.9)	0.006	-3.1 (-4.6 to -1.63)	<0.001
Nighttime-SBP	-4.1 (-8.7 to 0.6)	NS	-2.3 (-6.2 to 1.6)	NS
Nighttime-DBP	0.6 (-2.1 to 3.3)	NS	0.5 (-1.6 to 2.6)	NS
<b>HYPERTENSIVES (n= 24)</b>				
24h-SBP	-7.5 (-12.3 to -2.7)	0.004	-7.6 (-12.6 to -2.6)	0.005
24h-DBP	-4.2 (-7.6 to -0.8)	0.019	-4.3 (-7.6 to -0.9)	0.016
Daytime-SBP	-10.6 (-14.0 to -4.2)	0.001	-9.2 (-14.4 to -3.9)	0.002
Daytime-DBP	-5.2 (-8.9 to -1.4)	0.010	-4.8 (-8.3 to -1.2)	0.012
Nighttime-SBP	-2.6 (-9.7 to 4.4)	NS	-1.6 (-8.2 to 5.1)	NS
Nighttime-DBP	-0.6 (-5.3 to 4.2)	NS	0.1 (-4.6 to 4.9)	NS

DBP = diastolic blood pressure; SBP = systolic blood pressure

**Table S3.** Changes in office and ambulatory blood pressure, both central and peripheral, 12-months after bariatric surgery, according to surgical technique.

	CENTRAL					PERIPHERAL				
	LRYGB (n = 35)		L SG (n =27)		p*	LRYGB (n = 35)		LSG (n =27)		p*
	Change at 12 months post-BS Mean (95% CI)	p	Change at 12 months post-BS Mean (95% CI)	p		Change at 12 months post-BS Mean (95% CI)	p	Change at 12 months post-BS Mean (95% CI)	p	
Office-SBP (mmHg)	-7,4 (-11.8 to -3.1)	0.002	-6.9 (-12.7 to -1.1)	0.02	NS	-7.2 (-11.5 to -2.9)	0.002	-7.6 (-13.9 to -1.4)	0.02	NS
Office-DBP (mmHg)	-5.6 (-8.9 to -2.3)	0.002	-3.9 (-8.3 to 0.5)	0.08	NS	-5.1 (-8.3 to -1.9)	0.003	-4.2 (-8.9 to 0.6)	0.08	NS
24h-SBP (mmHg)	-5.3 (-9.3 to -1.3)	0.11	0.2 (-6.2 to 6.6)	NS	NS	-4.4 (-8.8 to 0.1)	0.05	0.2 (-6.0 to 6.3)	NS	NS
24h-DBP (mmHg)	-2.9 (-6.1 to 0.4)	0.08	0.7 (-3.4 to 4.8)	NS	NS	-2.3 (-5.6 to 0.9)	NS	0.7 (-2.7 to 4.2)	NS	NS
Daytime-SBP (mmHg)	-5.2 (-9.5 to -0.7)	0.018	-0.3 (-7.0 to 6.5)	NS	NS	-5.2 (-9.4 to -0.9)	0.02	-0.2 (-6.8 to 6.5)	NS	NS
Daytime-DBP (mmHg)	-4.2 (-7.8 to -0.7)	0.023	0.3 (-4.3 to 4.8)	NS	NS	-3.0 (-6.6 to 0.5)	0.09	0.7 (-3.5 to 4.8)	NS	NS
Nighttime-SBP (mmHg)	-3.0 (-9.2 to 3.3)	NS	1.9 (-5.7 to 9.5)	NS	NS	-1.8 (-8.5 to 5.0)	NS	1.8 (-5.3 to 9.0)	NS	NS
Nighttime-DBP (mmHg)	1.8 (-2.0 to 5.6)	NS	3.9 (-0.6 to 8.4)	NS	NS	-0.6 (-7.2 to 5.9)	NS	3.2 (-0.6 to 7.1)	NS	NS

\* for the comparison between LRGYB and SG of the changes in blood pressure parameters

DBP = diastolic blood pressure; LRYGB = laparoscopic Roux-in-Y gastric bypass;

LSG = laparoscopic sleeve gastrectomy; SBP = systolic blood pressure

**Table S4.** Comparison of arterial stiffness and systemic hemodynamic parameters between normotensives and hypertensives at baseline.

Parameter	Normotensives (n = 38)	Hypertensives (n = 24)	<i>p</i> - value
24-h PWV (m/s)	6.2 ± 1.1	6.9 ± 0.9	0.004
Alx@75 (%)	25.1 [20.9; 29.4]*	28.1 [18.0; 33.9]*	NS
Cardiac output (L/min)	4.68 ± 0.49	4.67 ± 0.66	NS
Vascular resistance (s*mmHg/mL)	1.21 ± 0.11	1.33 ± 0.15	0.001

Alx@75 = Augmentation Index corrected at 75 beats per minute; PWV = pulse wave velocity

\* median (p25<sup>th</sup>; p75<sup>th</sup>)

Figure S1.

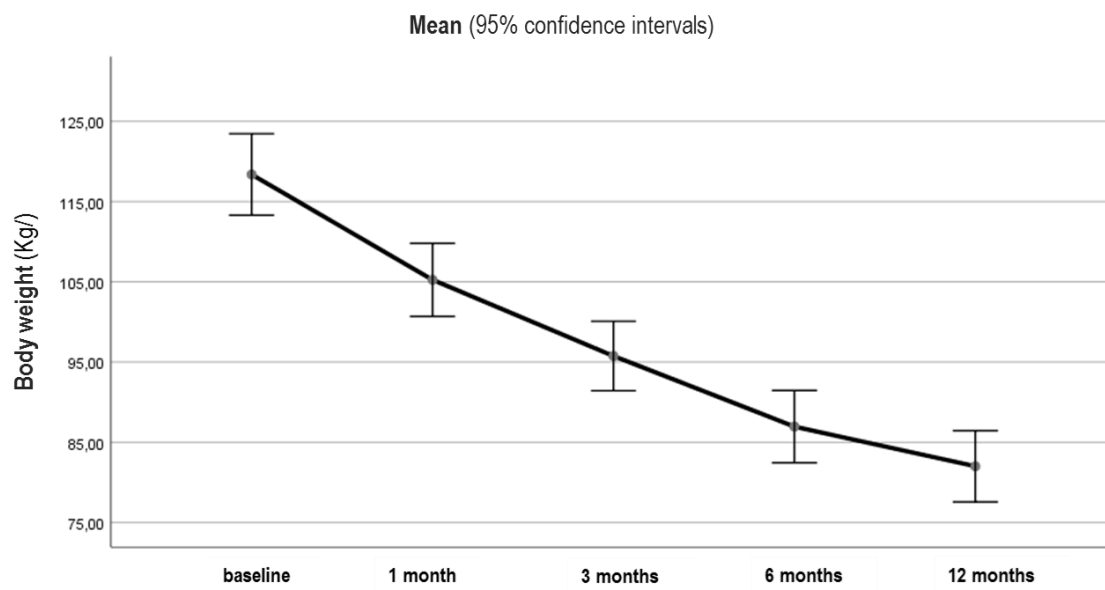
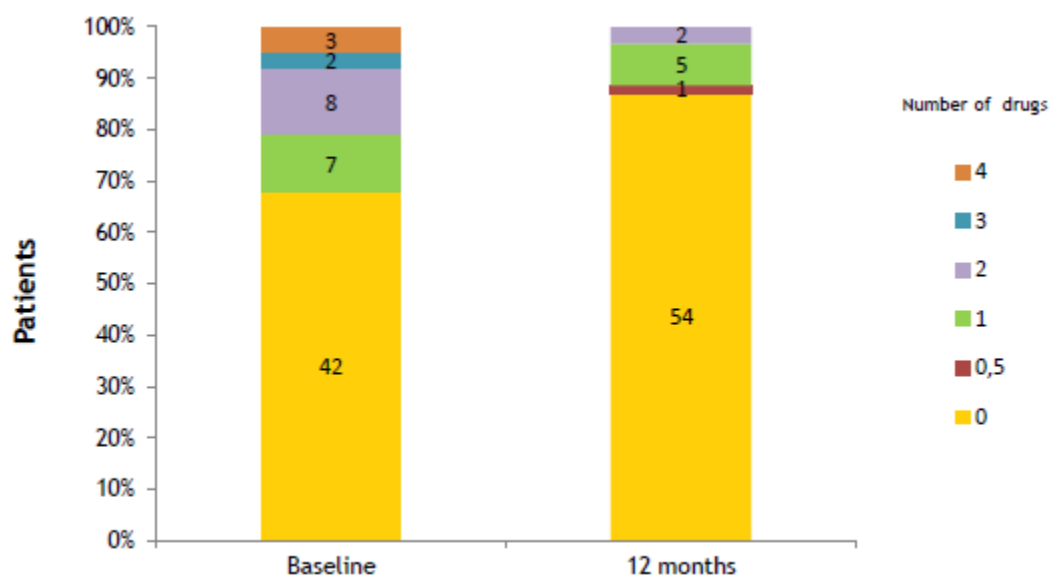


Figure S2.



Note: 0,5 drugs means medication in medium dosage. (reviewer #2, comment #1)