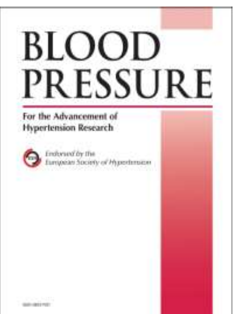

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RESEARCH ARTICLE

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Weight loss following gastric bypass increases aldosterone reactivity to orthostatic stress in patients with obesity

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ABSTRACT

Background: Weight loss after gastric bypass is associated with blood pressure (BP) reduction. However, the precise role of the sympathetic nervous system (SNS) and renin-angiotensin-aldosterone system (RAAS) in this reduction remains unclear. We investigated the effect of RYGB-induced weight loss on the hemodynamic, hormonal and renal responses to an orthostatic stress induced by lower body negative pressure (LBNP).
Methods: We conducted a monocentric study comparing response to LBNP (−30 mbar) in two groups of obese participants the first group underwent RYGB surgery (RYGB group) and the second group received lifestyle counselling (control group). We studied urinary sodium excretion, hemodynamic and hormonal responses before, during and 1 h after orthostatic stress induced by LBNP at three timelines: one month before planned RYGB (intervention group), and 3 and 12 months after the intervention.
Results: Thirty-seven adult participants were enrolled: 25 patients (72% women, age: 42.1 ± 10.5 years old, BMI 43.0 ± 5.1 kg/m^2) in the RYGB group and 12 in control group (58% women, age: 44.8 ± 13.6 years old, BMI 43.3 ± 5.3 kg/m^2). At 12 months, mean weight decreased from 126.3 ± 23.2 kg to 116 ± 20.7 kg in the control group and from 120.9 ± 19.4 kg to 78.6 ± 14.0 kg in the RYGB group (p value < 0.01 between groups). During LBNP, the reduction in urinary sodium excretion (−1.98 mmol/h; CI95%: −3.72 to −0.30, p value = 0.02) and the increase in plasma aldosterone concentration (PAC; +9.94 pg/ml, CI 95%: 0.317–19.569, p value = 0.043) were more pronounced in the RYGB group.
Conclusions: Our study suggests that weight loss induced by RYGB increases aldosterone responsiveness to orthostatic stress and enhances the sodium tubular response during orthostatic stress.
ClinicalTrials.gov ID: NCT02218112

PLAIN LANGUAGE SUMMARY

What is the context?

- Weight loss after gastric bypass surgery is known to lower blood pressure (BP).
- The precise role of the different hormonal systems involved is still unclear.

How did we proceed?

- In this study, we looked at how weight loss caused by Roux-en-Y gastric bypass (RYGB) surgery affects certain hormones and kidney responses during a posture change, a type of stress on the body called orthostatic stress.
  - To simulate this stress, we used a machine called lower body negative pressure (LBNP) that pulls blood toward the legs, mimicking the effects of standing up.
- We compared two groups of obese participants (37 adults participants):
  - The first group underwent RYGB surgery (surgery group, 25 patients),
  - The second group received lifestyle counselling (control group, 12 patients).

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Hypertension; lower body negative pressure; aldosterone; obesity; Roux-en-Y gastric bypass

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- We measured hormone levels, kidney sodium (salt) handling, heart rate (HR) and BP before, during and one hour after the orthostatic stress induced by LBNP at three time points: one month before surgery (surgery group), and 3 and 12 months later.

What is new?

- At 12 months, weight loss was much greater in the surgery group.
- During orthostatic stress, this group also showed a greater increase in aldosterone, a hormone that increases BP, and a stronger reduction in sodium loss in urine. Both of these results were statistically significant.

What is the impact?

- Our study suggests that weight loss after RYGB increases aldosterone responsiveness to orthostatic stress and enhances the kidney's ability to retain sodium during orthostatic stress, restoring the response seen in healthy patients.

Introduction

Obesity is a public health concern affecting about 23% of the European adult population and is associated with arterial hypertension (HTN) [1,2]. According to Framingham Heart Study results, 70% of the risk of developing HTN is due to overweight [3]. As expected, weight loss plays an important role in HTN control and is a cornerstone of HTN treatment in obese patients according to the European Society of Hypertension (ESH) guidelines [4–6]. When obesity is severe, Roux-en-Y Gastric Bypass (RYGB) is an effective yet invasive therapeutic option to achieve weight loss. It is associated with a reduction in blood pressure (BP) and reversal of glomerular hyperfiltration but its effects on renal physiology are still unclear [7–9]. The sympathetic nervous system (SNS) and the renin-angiotensin-aldosterone system (RAAS) are key regulators of BP also in obesity-related HTN, as they influence systemic hemodynamics, renal hemodynamics and sodium excretion [10,11].

Lower body negative pressure (LBNP) is a technique used to study renal hemodynamic and tubular changes, as well as systemic hemodynamic responses to orthostatic stress. By pooling blood in the lower body, venous return to the heart is reduced, decreasing cardiac output and eliciting a series of neuro-hormonal response depending on the level of negative pressure. In a previous study, we demonstrated that at −30 mbar, LBNP activated the SNS, the RAAS and reduced significantly urinary sodium excretion in healthy subjects [10]. Normotensive obese patients respond similarly as oppose to hypertensive obese patients who do not decrease urinary sodium excretion during the same exposure to LBNP suggesting a blunted urinary response in obese patients with HTN [12].

Currently, various weight loss strategies are available, including glucagon-like peptide-1 receptor agonists (GLP-1 RA) and RYGB. However, at the time this study was initiated, GLP-1 RA were considered an emerging therapy and were approved for the treatment of obsesity in 2022 in Switzerland. Therefore, our research question focused on whether weight loss induced by RYGB restores sensitivity to sodium reabsorption in relation to changes in the RAAS or SNS. Our aim was to compare the renal sodium handling in response to LBNP between obese patients treated with RYGB and those managed with lifestyle counselling alone.

Materials and methods

This study was a monocentric prospective study comparing the effect of orthostatic stress induced by LBNP in two weight loss groups of participants with obesity. The first group was planned for a RYGB (RYGB group) and the second group received lifestyle counselling only (control group).

The primary outcome was the change in urinary sodium excretion during LBNP. Secondary outcomes were the changes in the components of the RAAS and the SNS.

All patients were recruited from the obesity outpatient clinic of the Service of endocrinology and diabetes or the Service of nephrology and hypertension at Lausanne University Hospital in

Switzerland. Inclusion criteria were age between 18 and 65 years old, a body mass index (BMI) > 35 kg/m^2 and estimated glomerular filtration rate >60 mL/min/1.73 m^2. For the RYGB group, scheduled bariatric surgery was an additional inclusion criterion. Furthermore, participants were required to fully understand the protocol and sign the study consent form. Exclusion criteria included any acute disease, asthma or asthma history, participation in another study during the previous 30 days, pregnancy, blood donation in the past 2 months, use of non-steroidal anti-inflammatory drugs or any another drug that may influence kidney function. Documented renal stenosis, severe renal malformation or polycystic kidney disease, hepatic insufficiency with ascites, hip waist >130 cm, weight >220 kg, post-prandial glycaemia > 11 mmol/l and BP > 180/110 mmHg during consultation were also exclusion criteria.

Patients who fulfilled criteria for bypass surgery but declined the intervention were included in the control group and managed with conservative weight loss strategies. The RYGB surgery procedure was standardized as described in a previous publication [8]. Patients in the lifestyle group received dietary counselling, behavioural therapy, physical activity and motivational interventions. No patients received GLP-1 RA during this study.

This study (ClinicalTrials.gov ID: NCT02218112) received approbation by the local ethical committee (CER-VD) on 7 January 2014. The study protocol was in accordance with the Helsinki declaration. All patients gave their written informed consent. Data collection period was between May 2014 and June 2017.

Data collection and procedure during study days

Upon arrival, patients consumed an initial oral water load of 400 mL, followed by 150 mL of water every hour until the procedure's completion to maintain stable urine output. An Inulin perfusion, to measure GFR, was started at this time. BP and heart rate (HR) were measured each 15 minutes during the whole study day (Omron® HEM-907 model). Participants remained in the supine position throughout the study except for voiding. Anthropomorphic data were also collected at each visit. Urine volume was measured hourly. After 4 hours in supine position, the patient's lower extremities were placed in a Plexiglas box tightly closed at the level of the hips and LBNP was started for 1 hour at −30 mbar. Blood and urine samples were collected hourly after 3 hours in the supine position. Three time points were recorded: the pre-LBNP phase (0–60 min), the LBNP phase (60–120 min) and the post-LBNP phase (120–180 min). Blood analysis included sodium, creatinine, epinephrine (E), norepinephrine (NE), plasma aldosterone concentration (PAC), plasma renin activity (PRA) and inulin. Urine analysis included sodium, creatinine and inulin.

This procedure was repeated during three different visits: 1 month before intervention (visit 1), and then 3 and 12 months after intervention for the RYGB group and 4 and 13 months after the initial visit for the control group (visit 2 and 3, respectively). Antihypertensive medication was stopped 48 hours before each visit except for calcium channel blockers in patients taking ≥2 antihypertensive drugs. Spironolactone was withdrawn 6 weeks before the study days (1 participant).

Analytical method

NE and E were measured using ultra-high-performance liquid chromatography-tandem mass spectrometry [13]. PRA and PAC were determined by a radioimmunoassay kit for the quantitative determination of Angiotensin I in human plasma (REN-CT2, Cis-bio bioassays, Codolet, France) and by a commercial RIA kit (Aldo-Riact, CIS Bio International, Yvette, Cedex, Saclay, France), respectively.

The inulin clearance (mL/min) was calculated using the formula $Cl_{In} = U_{In} \times V/P_{In}$, where U_{In} and P_{In} are the urine and plasma concentrations of inulin and V the urine flow rate (ml/min). Sodium excretion was calculated with the following formula $V \times U_{Na}$, where U_{Na} (mmol/L) is the urinary sodium concentration. Fractional excretion of sodium (FE_{Na} in %) was calculated using $100 \times U_{Na} \times P_{In}/U_{In} \times P_{Na}$, where P_{Na} refers to plasma sodium concentration.

Statistical analysis

The primary outcome was the change from baseline in urinary sodium excretion during LBNP. Secondary outcomes are changes in measured glomerular filtration rate (mGFR), NE, PRA and PAC before, during and after LBNP protocol in the two groups.

Descriptive statistics are expressed as mean ± standard deviation (SD). We used Stata software for statistical analysis (StataCorp. 2023. Stata Statistical Software: Release 18: StataCorp LLC, College Station, TX) and R software version 4.3.2 (Vienna, Austria). We used a two-sample *t*-test to compare baseline characteristics between the two groups. To study the effect of LBNP, we performed robust linear mixed-effects models. Robust methods were used due to the presence of outliers. To reduce complexity, separate models were run for the change from baseline to LBNP and to post-LBNP. Each model was adjusted for the change at visit 1, age at visit 1, sex, group and the interaction between the group and the visit. A patient-specific random effect was considered to account for within-patient variability. Contrast estimates with 95% confidence intervals and *p* values were reported at each visit for the changes from baseline to LBNP and to post-LBNP. If any data were missing, a missing at random (MAR) mechanism was assumed and treated with maximum likelihood-based methods.

Results

Thirty-seven participants were enrolled in this study: 15 were included in the control group and 22 in the RYGB group. Three patients initially assigned to the control group, switched to the RYGB group. Their last study day before the surgical intervention was used as the baseline visit. In the final analysis 12 patients remained in the control group (58.3% women, age: 44.8 ± 13.6 years) and 25 in the RYGB group (72% women, age: 42.1 ± 10.5 years). All patients completed the three visits, except three in the control group who completed only visits 1 and 2. In the RYGB group, two urine collections were discarded by participants before collection (one during visit 1 in the pre-LBNP phase, one during visit 2 at LBNP phase).

There were no significant difference in systolic (SBP) and diastolic BP (DBP), age and BMI at the inclusion visit between the two study groups. Antihypertensive medications were used by 6 patients in the RYGB group and 4 patients in the control group. The median number of antihypertensive drugs used by the RYGB group taking medications was 2.5, decreasing to 1.0 after 12 months. In the control group, the median number of antihypertensive drugs used was 1.0, and this number remained stable throughout the study. Participants' characteristics at baseline and after 3 and 12 months are shown in Table 1. The detailed descriptive statistics for the variables of interest, categorized by visit, timepoint and group, are presented in Supplementary Table C.

Table 1. Characteristics of the study participants at visits 1, 2 and 3.

	Visit	Control ($n = 12$)	RYGB ($n = 25$)	*p*-value
Female sex (%)		58	72	0.41
Age (years)		44.8 (± 13.6)	42.1 (± 10.5)	0.50
BMI (kg/m^2)	1	43.3 (± 5.3)	43 (± 5.1)	0.88
	2	41.57 (± 4.84)	34.67 (± 4.53)	**< 0.01**
	3	39.27 (± 5.83)	28.0 (± 4.17)	***< 0.01***
Weight (kg)	1	126.3 (± 23.2)	120.9 (± 19.4)	0.46
	2	121.1 (± 21.0)	97.2 (± 14.8)	**< 0.01**
	3	116.0 (± 20.7)	78.6 (± 14.0)	**< 0.01**
Mean weight loss* (%)	2	− 3.76 (±5.92)	− 19.36 (±5.28)	**< 0.01**
	3	− 8.33 (±13.63)	− 34.83 (±13.63)	**< 0.01**
SBP (mmHg)	1	122.5 (±11.8)	117.1 (± 16.1)	0.31
	2	117.5 (± 15.1)	116.4 (± 12.8)	0.81
	3	116.1 (± 14.8)	110.5 (± 10.5)	0.22
DBP (mmHg)	1	78.5 (±9.4)	74.6 (±10.3)	0.27
	2	76.16 (± 10.43)	74.55 (± 11.54)	0.68
	3	74.9 (± 12.53)	66.14 (± 9.34)	***0.04***
HR (/minute)	1	70.2 (±11.6)	65.6 (±10.1)	0.22
	2	67.66 (± 10.68)	58.52 (± 7.12)	***< 0.01***
	3	67.28 (± 11.41)	56.24 (± 9.18)	***< 0.01***

Results are presented as mean (± standard deviation) or percentage. p-values < 0.05 are considered as statistically significant and are written in bold.

*Mean weight loss is expressed as percentage, with visit 1 serving as the baseline.

Three months after the initial visit, the mean weight decreased from 126.3 ± 23.2 kg to 121.1 ± 21kg in control group and from 120.9 ± 19.4 kg to 97.2 ± 14.8 kg in RYGB group (p value < 0.01 between groups). BMI was also lower in the RYGB group (34.7 ± 4.5 kg/m^2) than in the control group (41.6 ± 4.8 kg/m^2, p value < 0.01). SBP and DBP in pre-LBNP phase was similar between the two groups for SBP and DBP. The HR was lower in RYGB group (58.5 ± 7.1/min, *vs.* 67.7 ± 10.7/min in control group, p value < 0.01).

At 12 months, the mean weight further decreased to 116 ± 20.7 kg in the control group and to 78.6 ± 14.0 kg in the RYGB group (p value < 0.01 between groups). BMI was lower in the RYGB group (28.0 ± 4.2 kg/m^2) than in the control group (39.3 ± 5.8 kg/m^2, p value <0.01). DBP in pre-LBNP phase was also lower in the RYGB group (66.1 ± 9.3 mmHg *vs.* 74.9 ± 12.5 mmHg, respectively, p value < 0.04). SBP in the pre-LBNP phase followed the same trend as DBP but was not statistically significant (p value = 0.22). The HR was also lower in bypass group (56.2 ± 9.2/min, *vs.* 67.3 ± 11.4/min in control group, p value < 0.01).

Effect of lower body negative pressure on the first visit

LBNP increased NE and E in both groups (p value = 0.02 and p value < 0.01, respectively) with no difference between groups. This response was independent of age or sex, when those two variables were included in the mixed-effect model (Supplementary Table A). PAC decreased in response to LBNP in both groups (p value = 0.02). The effects on NE and PAC persist during post-LBNP phase. We did not observe a significant change in PRA. DBP increased in response to LBNP and remained higher during post-LBNP phase (p value < 0.01). However, we did not show any difference in time for SBP and HR. Measured GFR was lower during LBNP (p value < 0.01). Sodium excretion did not change at the various time points. PRA was lower in RYGB group than in control group (p value = 0.03).

Changes induced by LBNP 3 months after surgery

At 3 months, DBP, SBP and HR did not differ between the two groups in response to and after LBNP. We did not find any difference in renal response to LBNP. Inulin clearance, urinary output, FE_{Na} and sodium excretion were similar between groups. The hormonal response (PRA, PAC, NE and E) to LBNP did not vary significantly between the two groups. The results are presented in Table 2 and in Supplementary Table B.

Changes induced by LBNP 12 months after surgery

Systemic hemodynamics showed no significant differences during or after LBNP stimulation. The renal response to LBNP was not significant for urinary output, FE_{Na} and inulin clearance. However, we found a significant difference in sodium handling. LBNP decreased urinary sodium excretion in the RYGB group (contrast estimate −33.40 umol/min CI 95%; −62.03 to −4.78, p value = 0.02 compared to control group). This response persisted into the post-LBNP phase (contrast estimate −41.32 umol/min, CI95%; −78.54 to −4.11, p value = 0.03) in the RYGB group. The PAC response to LBNP was more pronounced in the RYGB group (contrast estimate +9.94 pg/mL, CI 95%; 0.317–19.57, p value = 0.04 compared to control group). PAC remained higher in the post-LBNP period (contrast estimate +11.68 pg/mL, CI 95%; 2.38–20.99, p value = 0.02) in RYGB group (Figure 1). We did not observe any difference in PRA during LBNP and post-LBNP phases. The adrenosympathetic response to LBNP evaluated with plasma NE and E was not different between groups. Complete results are presented in Table 2 and in Supplementary Table B.

Discussion

The main finding of this study is that one year after RYBG, the antinatriuretic response to an orthostatic stress is increased compared to a control group. This increased renal response is associated with an increased PAC response to LBNP in the RYGB group.

Table 2. Differences between the RYGB and control groups in their response to orthostatic stress (during the baseline and LBNP phases) at visits 2 and 3 was assessed using contrast estimates that compared change scores from robust linear mixed-effects models. A positive estimate indicates a larger outcome for the RYGB group, whereas a negative estimate indicates a larger outcome in the control group.

	Visit 2		Visit 3	
RYGB-Control	Estimate (2.5-97.5%)	*p*-value	Estimate (2.5-97.5%)	*p*-value
Mean SBP (mmHg)	−3.21 (-7.88 ; 1.47)	0.17	−4.64 (−9.80 ; 0.52)	0.08
Mean DBP (mmHg)	−3.07 (-7.01 ; 0.88)	0.13	−0.952 (−5.32 ; 3.41)	0.66
Mean HR (/min)	−0.532 (-3.41 ; 2.35)	0.71	0.649 (−2.5 ; 3.8)	0.68
NE (nmol/l)	−0.008 (-0.331 ; 0.315)	0.96	−0.095 (−0.456 ; 0.265)	0.60
E (nmol/l)	−0.005 (-0.028 ; 0.017)	0.63	−0.010 (−0.035 ; 0.015)	0.43
PAC (pg/mL)	5.77 (-3.34 ; 14.88)	0.21	9.94 (0.317 ; 19.57)	**0.04**
PRA (ng/mL/h)	−0.032 (-0.119 ; 0.054)	0.46	−0.009 (−0.105 ; 0.088)	0.86
Urinary output (ml/min)	−0.262 (-1.28 ; 0.755)	0.61	−0.671 (−1.791 ; 0.45)	0.24
Urinary sodium excretion (umol/min)	−8.96 (-36.06 ; 18.134)	0.51	−33.40 (−62.03 ; −4.78)	**0.02**
Inulin clearance (ml/min)	−2.24 (-14.80 ; 10.33)	0.72	−3.41 (−16.82 ; 10.01)	0.61
FE_{Na} (%)	−0.14 (-0.391 ; 0.112)	0.27	−0.210 (−0.478 ; 0.059)	0.12

LBNP = Lower body negative pressure, RYGB = Roux-en-Y gastric bybass, SBP = Systolic Blood Pressure, DBP = Diastolic Blood Pressure, HR = Heart Rate, NE = Norepinephrine, E = Epinephrine, PRA = Plamatic Renin Activity, PAC = Plasmatic aldosterone concentration, FE_{Na} = Fractional excretion of sodium. Bold values are statistically significant (*p*-value < 0.05).

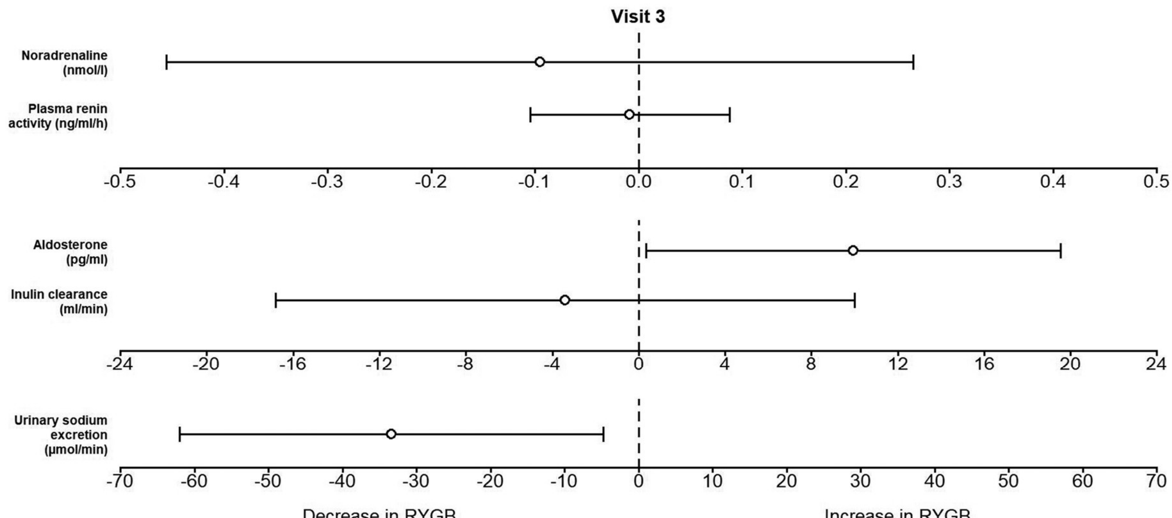

Figure 1. Differences in orthostatic stress responses between RYGB and control groups during LBNP phases at visit 3, displayed as a forest plot.

All groups responded to LBNP with increased NE and E during this orthostatic stress. This stimulation of the SNS is similar to our previous studies confirming the efficacy of the stimulation [10,12]. However, this sympathetic response did not appear to be affected by RYGB, as the response to LBNP remained unchanged in the month following the procedure. It can be argued that the sympathetic intrarenal response may differ from the plasma response, which is considered a suboptimal marker of SNS activity, however, we did not measure regional sympathetic nervous activity neither renal NE spill-over in our study [14].

At baseline, renal response was marked by a decrease in mGFR during LBNP as previously described in normotensive population with obesity [12]. However, as with the catecholaminergic response, this response did not change after weight loss. Despite the stability of the mGFR response in time, sodium retention during LBNP increased after surgery suggesting a new or possibly recovered responsiveness to an orthostatic stress restoring the response seen in healthy volunteers [10].

If the SNS did not seem to be involved in this restored response, the RAAS may have played a predominant role. As demonstrated in several articles, PAC is higher in patients with obesity [5,15–18]. It has been postulated that fat mass causes an increase in aldosterone *via* leptin secretion (leptin-aldosterone pathway) [19,20]. With weight loss, PAC often decreases [5,8,16,17,21,22]. We

recently demonstrated that this PAC reduction is independent of PRA and sodium excretion after RYGB [8]. These findings suggested a reduction of aldosterone secretion *via* reduction of leptin after weight loss. Hence, our hypothesis is that weight loss reduces leptin-aldosterone pathway thus restoring aldosterone reactivity to orthostatic stress. We confirmed these original observations after 12 months suggesting that a significant weight loss is required to achieve RAAS restoration. We believe that the observed effect is related to the weight loss induced by RYGB. However, whether RYGB *per se* is responsible for this effect cannot be answered with our study. The delayed time effect on PAC responsiveness suggests an effect associated with weight loss rather than RYGB. An effect of plasma volume status was unlikely since 24-hour urinary output was similar between the groups at 3 and 12 months. Sodium diet was not controlled during the study and may have impacted the results. However, we found a similar 24-hour sodium excretion between the two groups at baseline and at 12 months [8].

In the era of widespread use of GLP-1 RA, it would interesting to compare the effect of weight loss induced by GLP-1 RA to those of RYGB. To the best of our knowledge the chronic effect of GLP-1 RA on aldosterone reactivity has not been published. In healthy participants no chronic change on aldosterone after GLP-1 RA infusion were found [23,24]. If PAC response changed in time, we did not observe a concomitant increased in PRA. Several observations may explain this finding. First, the high-water load possibly blunted the RAAS response by inhibiting renin release. In fact, acute water loading is an important confounding factor reducing proximal sodium reabsorption [25,26]. Second, in healthy normotensive subjects with obesity, PRA is stimulated by LBNP at −30 mbar. This observation is not made in patients with obesity and HTN [10,12].

In the absence of PRA stimulation, the increase of aldosterone is intriguing. A stimulation of aldosterone secretion by adrenocorticotropic hormone (ACTH) cannot be excluded. This pathway seems unlikely as sodium retention induced by LBNP seems to predominantely enhance proximal tubule reabsorption and thus angiotensin 2 mediated [10]. However, indirect estimation of proximal and distal sodium reabsorption with endogenous lithium was not measured in our study.

PRA modifications after weight loss is not uniform across studies. Some have found no significant change [6,8,22,27], while others describe PRA decrease [16,21]. These discordant results can be explained by the multiple alterations and pathophysiological mechanisms involved, such as leptin-aldosterone pathway, a reduction in sodium intake or a reduced activity of the SNS. Thus, it is probable that different phenotypes exist in the obesity-induced HTN spectrum. In that way, visceral and subcutaneous obesity are susceptible to express different physiopathological patterns. Indeed, modifications of RAAS in patients with obesity may have a better correlation with visceral obesity [17,28], but many studies, including this study, do not distinguish fat distribution. This can explain a lack of significant PRA variation in our results.

Interestingly, a sex difference seems to exist in the pathophysiology of obesity-associated HTN [29]. In fact, according to some studies, premenopausal women seem to be more sensitive to leptin through the aldosterone pathway and men through the SNS pathway [17,29]. The sample size of our study did not enable us to perform sex-specific analysis.

The limitations of our study stem from the small sample size, which was a result of the complex and demanding design, where minor differences may have been overlooked. Second, the lack of analysis of endogenous lithium handling does not allow the characterization of proximal or distal renal sodium handling to untangle the tubular effect of aldosterone. Thirdly, we did not make a distinction for fat distribution (subcutaneous and visceral) despite the fact that HTN phenotype might be different [28].

To conclude, this study suggests a novel insight into the understanding of reversibility of obesity-induced HTN and sodium handling after weight loss. To the best of our knowledge, this is the first evidence of restitution of aldosterone response after RYGB weight loss. Further studies to evaluate the clinical response for instance in an acute stress need to be realized.

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Authors' contributions

JZ: Data analysis and interpretation, drafting of manuscript.
NV: Acquisition of data, reviewing the manuscript
NS: reviewing the manuscript
MHB: database preparation and cleaning, reviewing the manuscript
SM: reviewing the manuscript
MM & EG: Laboratory and blood samples analysis.
JS: Statistical analysis
LF: Participation to patient's recruitment, contribution to writing manuscript.
GW: Study concept and design, conducting and study supervision and reviewing the manuscript.

Disclosure statement

The authors have no conflicts of interest to declare in relation to this article.

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Data availability statement

The data underlying this article will be shared on reasonable request to the corresponding author.

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