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Role of Echocardiography in Bariatric Surgery: Preoperative Assessment of Non-Cardiopathic Morbidly Obese Patients

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Abstract

Introduction: Morbidly obese patients are at risk of cardiovascular events and their optimal assessment before surgery is controversial. The aim of this study was to analyze the value of transthoracic echocardiography to detect structural and functional cardiac alterations and as a predictor of cardiovascular morbidity both in the operative period and in the immediate postoperative period in high risk but non-cardiopathic patients undergoing bariatric surgery.

Material and Methods: A transthoracic echocardiography was routinely performed in non-cardiopathic patients with OSA and/or hypertension treated with two or more drugs. Eighty seven patients aged 48.5 ± 9 years, BMI of 45.9 ± 7 kg/m^2 were studied. Cardiovascular risk factors, the anesthetic monitoring and the cardiovascular complications were evaluated.

Results: An abnormal indexed left ventricular mass (LVMi) was observed in 12.6 % of patients, septum hypertrophy in 24%. Diastolic dysfunction (DD) was diagnosed in 32.7% of subjects (74% with a mild alteration). No significant differences were found in the echocardiography findings in patients with severe obesity (BMI > 50 kg/m^2) compared with the rest of the subjects. In the linear regression analysis age (β=0.256, P=0.021) was found to be the main determinant of LVMi (R^2 for the model= 0.314). Regarding intraoperative monitorization, two patients required special measures due to surgical complications and one patient suffered from a cardiovascular complication but in neither case did the echocardiografic findings prompt a change in the anesthesic management.

Conclusions: The echocardiographic alterations found in a Mediterranean population of non-cardiopathic morbidly obese patients as candidates for bariatric surgery, were low. The echocardiographic findings did not change the anesthetic management of these patients and the cardiovascular events observed in the operative and immediate postoperative period were very scarce. This indicates that this test is not useful when routinely performed.

Keywords: Morbid obesity; Bariatric Surgery; Echocardiography; Anesthesia; Preoperative risk

Introduction

Morbid obesity is often associated with conditions that increase cardiovascular risk including dyslipidemia, systemic arterial hypertension (SHT), type 2 diabetes mellitus (DM2), metabolic syndrome and obstructive sleep apnea (OSA) [1,2]. Obesity is associated with increased blood volume and cardiac output [3-5]. This pre-load condition increases ventricle size, wall stress and left ventricular mass, leading to the development of eccentric ventricular hypertrophy [3,4], systolic dysfunction and ventricular dilation, that leads to the development of left ventricular failure (LVF) [6-8]. These alterations in cardiac structure can be present without any clinical signs, representing a subclinical manifestation of obesity cardiomyopathy [9,10].

Bariatric surgery is the most effective procedure for weight loss maintenance [11]. It is associated with a substantial reduction in obesity-related comorbidities and the incidence of cardiovascular events [12-14]. However, morbidly obese patients undergoing bariatric surgery are at an increased risk of perioperative cardiorespiratory complications, such as, intra-operative ventricular dysfunction precipitated by rapid fluid administration and by the negative inotropic effects of anaesthetic agents, pulmonary hypertension, pulmonary embolism and myocardial ischemia [15,16]. Before considering surgery, a careful preoperative evaluation must be performed in these patients in order to assess their cardiovascular risk that can condition the anesthetic management and recovery.

Regarding this, the preoperative assessment using transthoracic echocardiography in morbidly obese patients even without known heart disease could be useful. However, at present there are no established recommendations for its use [17].

The aim of our study was to analyze the value of transthoracic echocardiography to detect structural and functional cardiac alterations and as a predictor of cardiovascular morbidity both in the operative period and in the immediate postoperative period in high risk but non-cardiopathic patients undergoing bariatric surgery.

Methods

We conducted a retrospective observational study of 99 consecutive morbidly obese outpatients undergoing preoperative cardiac assessment over the period from January 2011 to January 2013. According to our Unit´s protocol, a transthoracic echocardiography was performed in all non-cardiopathic morbidly obese patients with the presence of OSA and/or hypertension treated with two or more drugs, before bariatric surgery. The exclusion criteria was the presence of a very poor acoustic window (n:9) or a history of cardiomyopathy (n:3), defined as the presence of atrial fibrillation, ventricular systolic dysfunction, myocardial ischemia and severe valvular disease. Finally 87 patients were included in the study.

Preoperative anthropometric measurements were collected. Height and weight were measured with the patient standing in light clothes and without shoes. Body mass index (BMI) was calculated as body weight divided by height squared (kg/m^2). Obesity was classified according to the World Health Organization criteria [18]. The study protocol included an evaluation of cardiovascular risk factors such as SHT, DM2, dyslipidemia, OSA and their respective treatments. SHT was defined as systolic pressure ≥ 140 mmHg and/or diastolic pressure ≥ 90 mmHg or by the use of anti-hypertensive medication. DM was defined according to ADA consensus [19] and dyslipidemia according to ATPIII [20].

All transthoracic echocardiography (General Electric Vivid 7 Dimension) was performed by two experienced cardiologists during the preoperative period, using one-dimensional mode (Mode M), two dimension (2D) and Doppler (pulsed and color) echocardiography in the paraesternal and apical windows. Left ventricular mass (LVM) measured using Devereux Formula [21], and left ventricular end-diastolic diameter (LVEDD) were assessed and corrected by body surface area, resulting in indexed values, LVMi and LVEDDi, respectively. Other parameters evaluated were: interventricular septal wall thickness (SWT), left ventricular fractional shortening (LVFS), left ventricular ejection fraction (LVEEF) measured by the Teichholz method, anteroposterior diameter of the left atrium (DLA); aortic root diameter (ARD); pulmonary arterial pressure (PAP), peak velocities of the E and A waves, E/A ratio and E waves deceleration time (EDT). For the reference values the Images of Cardiology Handbook and the American Society of Echocardiography guidelines and standards were used [22,23].

The surgical technique and the intraoperative and postoperative (within one month) cardiovascular complications were also reported. Cardiovascular complications were defined as acute coronary syndrome, including myocardial infarction or unstable angina (using standard definitions and determined by electrocardiographic findings and cardiac biomarker measurement), congestive heart failure (determined by examination findings and confirmatory testing, including chest X-ray and biomarker measurement), and new onset of rhythm abnormality (atrial fibrillation/flutter and supraventricular and ventricular arrythmias).

All bariatric patients undergo general anesthesia in our center. Intraoperative hemodynamic values are computed by a Datex Ohmeda system monitor, heart rate from a single-channel electrocardiogram signal, blood pressure (BP) from a noninvasive and inflatable BP cuff every 5 minutes, continuous pulsioximetry and end-tidal CO_2. For the first 5 minutes, patients breathe oxygen by face mask (fresh gas flow 6 L min^{-1}), and general anesthesia is usually induced with propofol 2 mg kg^{-1} and fentanyl 3 µg kg^{-1} of ideal weight. Tracheal intubation is, then, facilitated with rocurorium 0.9 mg kg^{-1}. Anesthesia is maintained with sevoflurane (FISEV 2.1%) or desflurane (FISEV 6.6%), according to the preferences of anesthesiologists who perform the procedure. Isotonic fluids are usually used and the fluid therapy is a balanced balance sheet. For the standard, two venous accesses are used, without further or more specific venous access or monitoring. Deviation from standard intraoperative monitorization, such as the use of a central venous catheter (CVC), the prescription of vasoactive drugs, and the performance of invasive BP determinations is used in alteration of hemodynamic parameters and it was registered.

All participants had signed a written consent to be included in the study, which was approved by the research ethics board of our hospital.

Statistical analysis

Data were expressed as mean ± SD or percentage for parametric and categorical data, respectively. The bivariate comparisons were evaluated using the Chi-squared (categorical), t (parametric) or Mann–Whitney (nonparametric) unpaired tests. Univariate and multiple regression analysis were performed to identify independent factors affecting LVM and diastolic dysfunction (DD). A value of $p<0.05$ was considered to be statistically significant. All statistical analysis was performed using the Statistical Package for Social Sciences (SPSS/Windows version 18, SPSS Inc., Chicago, IL, USA).

Results

Clinical data n=87	
Age (years)	48.5 ± 9.3
Gender (M/F)	35/52
Weight (kg)	125.91 ± 23.90
BMI (kg/m^2)	45.9 ± 7.6
Hypertension	53 (60.9%)
Dyslipidemia	38 (43.7%)
Type 2 Diabetes	30 (34.5%)
OSA	77 (88.5%)
Roux-en-Y gastric bypass	19 (21%)
Sleeve gastrectomy	39 (44.8%)
Gastric plication	11(12%)
Duodenal switch	18 (20%)

Table 1: General characteristics of the study population; All data are expressed as mean ± standard deviation (SD). OSA: obstructive sleep apnea.

Eighty-seven non-cardiopathic morbidly obese patients (35 men, 52 women), 48.5 ± 9 years old and with initial BMI of 45.9 ± 7 kg/m^2 were studied before undergoing bariatric surgery. Twenty (22.9%) had a BMI above 50 kg/m^2. Anthropometric and clinical characteristics of all patients are summarized in Table 1.

Fifty three patients (60.9%) had SHT, and 23 (41.8%) were using two or more anti-hypertensive medication. Thirty eight patients (43.7%) had dyslipidemia and twenty six of them were under treatment. Thirty patients (34.5%) had DM2 thirteen (41.9%) were treated with one or more doses of insulin. Seventy-seven patients (88.5%) had OSA and 80% of them were under continuous positive airway pressure (CPAP) treatment.

Different laparoscopic bariatric surgical techniques were used: Roux-en-Y gastric bypass was performed in 19 patients, sleeve gastrectomy in 39, gastric plication in 11 and duodenal switch in 18.

The echocardiography findings are exposed in Table 2 and 3. An abnormal LVMi was observed in 9 patients (12.6 %). Patients with altered LVMi were significantly older (56 ± 5 vs. 48 ± 9 years, p= 0.044), and had a higher prevalence of dyslipidemia (83% vs. 40%, p=0.041) compared to patients with normal values. No patient presented an altered LVEDDi. Transthoracic echocardiography showed hypertrophy of the septum in 21 patients (24%). Patients with hypertrophy of the septum were significantly older (51 ± 6 vs. 47 ± 9 years, p=0.025) compared to patients with a septum in the reference range but showed similar BMI and similar percentages of SHT, DM2, dyslipidemia and OSA. Regarding systolic function, 3 patients (3.4%) had a LVEEF under 55%. DD was evaluated in 58 patients out of 87. In the rest of the participants it could not be assessed due to a poor acoustic window. Nineteen subjects (32.7%) were diagnosed with DD, and among them, 74% presented a mild alteration (grade I dysfunction) and 26% grade III dysfunction. Patients with DD were older without reaching statistical significance (51 ± 7.3 vs. 46.2 ± 10 p=0.053) but no differences were found in the percentages of SHT, DM2, dyslipidemia and OSA compared to subjects with normal diastolic function. In 28 patients the PAP was measured and in 8 (28.5%) it was elevated.

	WOMEN		MEN	
	Mean results	**Moderately abnormal**	**Mean results**	**Moderately abnormal**
LVMi, g/m^2	81.73 ± 23.04	101-112	89.99 ± 18.31	117-130
LVEDDi ,mm/m^2	23.25 ± 2.58	35-37	21.76 ± 2.58	35-36
SWT, mm	10.85 ± 1.7	13-15	11.9 ± 2.06	14-16
LVFS (%)	40.42 ± 4.62	17-21	39.29 ± 5.94	17-21
LVEEF (%)	64.66 ± 5.96	30-44	63.40 ± 7.15	30-44
DLA (mm)	40.06 ± 5.31	43-46	43.0 ± 8.16	47-52
ARD (mm)	29.15 ± 3.16	20-32	33.63 ± 3.63	20-32
PAP (mm)	32.52 ± 7.89	35-37	31.08 ± 5.08	35-37

Table 2: Results and reference range of echocardiography findings according to sex; All data are expressed as mean ± standard deviation (SD). LVMi: left ventricular mass indexed by body surface area; LVEDDi: left ventricular end diastolic diameter index by body surface area; SWT: interventricular septal wall thickness; LVFS: left ventricular fractional shortening; LVEEF: left ventricular ejection fraction; DLA: diameter of the left atrium; ARD: aortic root diameter; PAP: pulmonary artery pressure. Moderately abnormal values according to the American Society of Echocardiography guidelines and standards.

	Mean results		**Reference values of diastolic dysfunction**		
	Women n=34	Male n=24	Grade I	Grade II	Grade III
Wave E	0.84 ± 0.20	0.75 ± 0.16	≤ 0.5	0.5-1.2	≥1,2
Wave A	0.77 ± 0.22	0.67 ± 0.18	≥0.8	0.3-0.8	≤0.3
E/A ratio	1.15 ± 0.44	1.19 ± 0.43	<1	1-1.5	≥2
EDT (msc)	203.9 ± 35.7	204.25 ± 34.98	≥240	160-240	≤150

Table 3: Results and reference range of diastolic function findings; All data are expressed as mean ± standard deviation (SD). EDT: E waves deceleration time. Diastolic dysfunction values according to the Images of Cardiology Handbook.

We selected patients with severe obesity (BMI>50 kg/m^2) and compared the echocardiography findings with the rest of the subjects. No significant differences were found in LVMi, LVEDDi, SWT, LVFS, LVEEF, DLA, ARD and PAP in both groups (Table 4).

	BMI<50	**BMI>50**
	n:67	**n:20**
LVMi, g/m^2	86.29 ± 22.13	80.96 ± 19.48
LVEDDi, mm/m^2	23.11 ± 2.72	21.10 ± 1.80
SWT, mm	11.40 ± 2.00	11.05 ± 1.71
LVFS (%)	63.7 ± 6.6	65.65 ± 5.6
LVEEF (%)	39.7 ± 5.32	40.75 ± 4.78
DLA (mm)	41.25 ± 5.73	41.05 ± 9.40
ARD (mm)	31.15 ± 3.99	30.45 ± 3.96
PAP (mm)	31.42 ± 7.34	34.4 ± 2.19

Table 4: Results of echocardiography findings according to body mass index; All data are expressed as mean ± standard deviation (SD). BMI: body mass index; LVMi: left ventricular mass indexed by body surface area; LVEDDi: left ventricular end diastolic diameter index by body surface area; SWT: interventricular septal wall thickness; LVFS: left ventricular fractional shortening; LVEEF: left ventricular ejection fraction; DLA: diameter of the left atrium; ARD: aortic root diameter; PAP: pulmonary artery pressure.

A univariate correlation analysis was performed, and showed that LVMi correlated positively with age (r=0.29 p=0.007), but not with BMI. No significant correlation was found between DD and age or BMI. In the linear regression analysis age (β=0.256, P=0.021) was found to be the main determinant of LVMi (R^2 for the model= 0.314). No association was found with BMI and the presence of SHT, DM2, dyslipidemia or OSA.

Regarding intraoperative monitorization, only two patients required the use of CVC not because of ecocardiographic findings but due to surgical complications. Both had intraoperative bleeding that required a CVC for a possible initiation of vasoactive drugs and to facilitate the extraction of blood samples. Only one patient (1.1%) suffered from a cardiovascular complication in the early postoperative period within 6 hours of the intervention. The patient was a 50-year-old woman and had a BMI of 54 kg/m^2 a history of DM2, SHT and chronic alveolar hypoventilation syndrome treated with invasive ventilation (IV). She was admitted to hospital a month before the intervention due to a respiratory failure in the context of an acute bronchitis. When her respiratory problem was stabilized she was prepared for surgery. The echocardiogram prior to surgery showed lower–lateral-posterior akinesia, LVEEF of 47%, LVMi 967 g/m^2 SWT 12 mm, a normal PVC and DD grade I. The anesthesia induction was performed with propofol (300 mg), fentanyl (150 mg) and rocuronium (70 mg), the mean blood pressure during surgery was 65 mmHg, and a total of 1040 cc of fluid was administered in two hours. During the postoperative period she suffered from an acute heart failure that was resolved with diuretics within 12 hours.

Discussion

To our knowledge, this study is the first to report echocardiographic alterations in a Spanish population of morbidly obese patients prior to bariatric surgery. The echocardiographic alterations found in our cohort of non-cardiopathic morbidly obese patients candidates for bariatric surgery, were low. From an echocardiographic perspective, the "cardiomyopathy of obesity" is mainly manifested by a LV remodeling and ventricular DD [24]. Our data showed a low prevalence of LV hypertrophy (12.6%) and ventricular DD (32.7%), even though the population studied had an elevated cardiovascular risk as most of them were hypertensive and/or with OSA. In contrast to our study, in a sample of 30 asymptomatic morbidly obese patients, Rocha et al., described prevalence of LV hypertrophy in 46.4% and of DD in 54.6% [10]. These discrepant results might be explained by the use in this latter study of other reference values instead of the American Society's Echocardiography standards. Also, in this former study, 83% of patients had SHT in comparison to 60% in our population. Using our same standards, Tavares et al., reported the presence of LV hypertrophy in 20% and DD in 53% in a cohort of 132 morbidly obese patients from Brazil without heart disease [25]. Comparing with our population, the prevalence of SHT was similar but dyslipidemia and DM2 was higher, and OSA was lower. However, almost 5% of patients consumed alcohol, and in our patients no alcohol consumption was allowed as it was a contraindication for bariatric surgery. Moreover, other factors such as ethnic differences and the use of tissue Doppler method to measure the DD could explain the differences in the results. Another important point to be considered is that our study was performed using a Mediterranean population which is known to have a lower risk of cardiovascular events, explained in part because of the dietary properties [26]. Nevertheless, we were unable to find other references regarding echocardiographic values of a population sharing similar characteristics to ours.

The analysis of the factors influencing LVMi and diastolic function showed that BMI and comorbidities were not determinants for ventricular hypertrophy or DD. Instead, older age was significantly associated with both parameters. The fact that SHT was not associated with LVMi could be explained since most patients had a mild SHT that was controlled with only two drugs.

In our study the pathological findings in the echocardiography did not correlate with an increased percentage of intraoperative or immediate postoperative complications. This might be partly explained by the very low rate of adverse cardiovascular events observed. Earlier studies have reported an increased mortality in bariatric surgery, and obesity appeared to be an independent risk factor of morbidity and mortality from cardiovascular and pulmonary complications [27-30]. According to Klasen et al., obstructive and restrictive pulmonary syndromes, silent coronary heart diseases, or subclinical heart failure could decompensate in the immediate postoperative period in bariatric patients [16]. Nevertheless, bariatric surgery has evolved in recent years and advances in anesthesiology, the use of new drugs, better monitoring procedures, and ventilators have improved the prognosis of these patients. Moreover, their greater level of surveillance probably explains the low incidence of fatal events. Indeed, recent studies analyzing cardiovascular complications during obesity surgery, even in patients with known heart disease, have shown a rate of non-fatal cardiovascular events ranging between only 5.8 to 3% [31, 32]. We have to take into account that our hospital is a referral hospital for bariatric surgery with experienced surgeons and anesthetists explaining the low prevalence of complications. As previously described, monitoring and anesthetic management were not conditioned by echocardiographic findings and only 2 patients required special intraoperative monitorization, secondary to a surgical complication and not to any cardiovascular events. Additionally, the echocardiographic findings didn't determine the place of post-surgical recovery. In our study, only one subject had an adverse cardiovascular event, an episode of acute pulmonary edema in the immediate postoperative period. This patient had an elevated surgical risk because of her severe respiratory insufficiency treated with IV and high BMI. But even in this case the echocardiographic assessment did not condition in her anesthetic management.

In the current literature, there are no specific guidelines for preoperative evaluation of morbidly obese candidates for bariatric surgery [17]. Our data show that routinely including echocardiography in the preoperative assessment of all candidates with SHT, BMI>50 kg/m^2 and with OSA is not useful. Although transthoracic echocardiography is a non-invasive diagnostic test, it has a cost and it can delay surgery. From a clinical point of view, the echocardiographic alterations found in our study had no consequences on the perioperative management, suggesting that its use should be restricted to higher risk patients. In fact, the results of our study have changed our protocol of preoperative assessment before bariatric surgery, confining the use of echocardiography to patients with known heart disease.

Study limitations

A limitation of our study is that it is retrospective and no control group was examined for comparison. Also, the number of patients studied was small and we are aware that the sample should be enlarged in order to confirm our results. Patients with a very poor acoustic window were excluded, however, they were only nine with similar BMI compared to the rest of participants and none of them had operative complications. Nevertheless, some patients included in the study had a limited acoustic window because of their obesity and this might have interfered with the echocardiographic measurements. Regarding echocardiography, we did not use tissue doppler measurement which is more specific to quantify DD or ecocardiographic contrast that could have improved the image quality. The color- Doppler M-Mode of transmitral blood flow, which is another measure of DD, was neither used because in the routine evaluation of diastolic function we only use peak velocities of the E and A waves, E/A ratio and E waves deceleration time (EDT).

Conclusion

The echocardiographic alterations found in a Mediterranean population of non-cardiopathic morbidly obese patients as candidates for bariatric surgery, were low. The echocardiographic findings did not change the anesthetic management of these patients and the cardiovascular events observed in the operative and immediate postoperative period were very scarce. This indicates that this test is not useful when routinely performed.

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References

1. Lois K, Kumar S (2009) Obesity and diabetes. Endocrinol Nutr 56: 38-42.
2. Kannel WB, D'Agostino RB, Cobb JL (1996) Effect of weight on cardiovascular disease. Am J Clin Nutr 63: 419S-422S.
3. Alpert MA (2001) Obesity cardiomyopathy: pathophysiology and evolution of the clinical syndrome. Am J Med Sci 321: 225-236.
4. Benotti PN, Bistrain B, Benotti JR, Blackburn G, Forse RA (1992) Heart disease and hypertension in severe obesity: the benefits of weight reduction. Am J Clin Nutr 55: 586S-590S.
5. Iacobellis G, Ribaudo MC, Leto G, Zappaterreno A, Vecci E, et al. (2002) Influence of excess fat on cardiac morphology and function: study in uncomplicated obesity. Obes Res 10: 767-773.
6. Alpert MA, Terry BE, Lambert CR, Kelly DL, Panayiotou H, et al. (1993) Factors influencing left ventricular systolic function in nonhypertensive morbidly obese patients, and effect of weight loss induced by gastroplasty. Am J Cardiol 71: 733-737.
7. Zarich SW Kowalchuk GJ, McGuire MP, Benotti PN, Mascioli EA, et al. (1991) Left ventricular filling abnormalities in asymptomatic morbid obesity. Am J Cardiol 68: 377-381.
8. Iacobellis G Ribaudo MC, Zappaterreno A, Iannucci CV, Di Mario U, et al. (2004) Adapted changes in left ventricular structure and function in severe uncomplicated obesity. Obes Res 12: 1616-1621.
9. Kenchaiah S Evans JC, Levy D, Wilson PW, Benjamin EJ, et al. (2002) Obesity and the risk of heart failure. N Engl J Med 347: 305-313.
10. Rocha IE Victor EG, Braga MC, Barbosa e Silva O, Becker Mde M (2007) Echocardiography evaluation for asymptomatic patients with severe obesity. Arq Bras Cardiol 88: 52-58.
11. Sugerman HJ1 (2005) The pathophysiology of severe obesity and the effects of surgically induced weight loss. Surg Obes Relat Dis 1: 109-119.1
12. Sjstrm L, Rissanen A, Andersen T, Boldrin M, Golay A, et al. (1998) Randomized placebo-controlled trial of orlistat for weight loss and prevention of weight regain in obese patients. European Multicentre Orlistat Study Group. Lancet 352: 167-172.
13. Christou NV Sampalis JS, Liberman M, Look D, Auger S, et al. (2004) Surgery decreases long-term mortality, morbidity, and health care use in morbidly obese patients. Ann Surg 240: 416-423.
14. Adams TD Gress RE, Smith SC, Halverson RC, Simper SC, et al. (2007) Long-term mortality after gastric bypass surgery. N Engl J Med 357: 753-761.
15. O Neill, Allam J (2010) Anaesthetic considerations and management of the obese patient presenting for bariatric surgery. Current Anaesthesia & Critical Care 21: 16-23.
16. Klasen J Junger A, Hartmann B, Jost A, Benson M, et al. (2004) Increased body mass index and peri-operative risk in patients undergoing non-cardiac surgery. Obes Surg 14: 275-281.
17. Mechanick JI, Youdim A, Jones DB, Garvey WT, Hurley DL, et al. (2013). American Association of Clinical Endocrinologists; Obesity Society; American Society for Metabolic & Bariatric Surgery (2013) Clinical practice guidelines for the perioperative nutritional, metabolic, and nonsurgical support of the bariatric surgery patient--2013 update: cosponsored by American Association of Clinical Endocrinologists, The Obesity Society, and American Society for Metabolic & Bariatric Surgery. Obesity (Silver Spring) 21: S1-27.
18. World Health Organization (1997) Obesity: preventing and managing the global epidemic. In: Report on a WHO consultation on obesity. WHO/NUT/NCD/98.1. Geneva, Switzerland: World Health Organization.
19. American Diabetes Association (2012) Standards of medical care in diabetes--2012. Diabetes Care 35 Suppl 1: S11-63.
20. Grundy SM, Cleeman JI, Merz CN, Brewer HB Jr, Clark LT, et al. (2004) National Heart, Lung, and Blood Institute; American College of Cardiology Foundation; American Heart Association Implications of recent clinical trials for the National Cholesterol Education Program Adult Treatment Panel III guidelines. Circulation 110: 227-239.
21. Devereux RB, Casale PN, Kligfield P, Eisenberg RR, Miller D, et al. (1986) Performance of primary and derived M-mode echocardiographic measurements for detection of left ventricular hypertrophy in necropsied subjects and in patients with systemic hypertension, mitral regurgitation and dilated cardiomyopathy. Am J Cardiol 57: 1388-1393.
22. Solís J, Fernández L, Diano Cárdenas N (2011) Manual de imagen en cardiología. (1ra edición), Pulso Ediciones, Barcelona.
23. Lang RM, Bierig M, Devereux RB, Flachskampf FA, Foster E, et al; Chamber Quantification Writing Group; American Society of Echocardiography's Guidelines and Standards Committee; European Association of Echocardiography (2005) Recommendations for Chamber Quantification: A Report from the American Society of Echocardiography's Guidelines and Standards Committee and the Chamber Quantification Writing Group, Developed in Conjunction with the European Association of Echocardiography, a Branch of the European Society of Cardiology. J Am Soc Echocardiogr 18(12): 1440-1463.
24. Powell BD Redfield MM, Bybee KA, Freeman WK, Rihal CS (2006) Association of obesity with left ventricular remodeling and diastolic dysfunction in patients without coronary artery disease. Am J Cardiol 98: 116-120.
25. Tavares Ida S Sousa AC, Menezes Filho RS, Aguiar-Oliveira MH, Barreto-Filho JA, et al. (2012) Left ventricular diastolic function in morbidly obese patients in the preoperative for bariatric surgery. Arq Bras Cardiol 98: 300-306.
26. Estruch R Ros E, Salas-Salvadó J, Covas MI, Corella D, et al. (2013) Primary prevention of cardiovascular disease with a Mediterranean diet. N Engl J Med 368: 1279-1290.
27. Catheline JM Bihan H, Le Quang T, Sadoun D, Charniot JC, et al. (2008) Preoperative cardiac and pulmonary assessment in bariatric surgery. Obes Surg 18: 271-277.
28. Lam B Sam K, Mok WY, Cheung MT, Fong DY, et al. (2007) Randomised study of three non-surgical treatments in mild to moderate obstructive sleep apnoea. Thorax 62: 354-359.
29. Deutzer J1 (2005) Potential complications of obstructive sleep apnea in patients undergoing gastric bypass surgery. Crit Care Nurs Q 28: 293-299.
30. Mokdad AH Marks JS, Stroup DF, Gerberding JL (2004) Actual causes of death in the United States, 2000. JAMA 291: 1238-1245.

31. Alsabrook GD Goodman HR, Alexander JW (2006) Gastric bypass for morbidly obese patients with established cardiac disease. Obes Surg 16: 1272-1277.

32. Afolabi BA Novaro GM, Szomstein S, Rosenthal RJ, Asher CR (2009) Cardiovascular complications of obesity surgery in patients with increased preoperative cardiac risk. Surg Obes Relat Dis 5: 653-656.