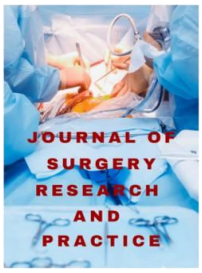

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Case Report

Low Mass Low Beat: The Effect of Bariatric Surgery on Cardiac Rhythm

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Abstract

Obesity is a global health problem, associated with significant morbidity and mortality. It is a multifactorial disorder associated with serious complications including diabetes mellitus, dyslipidemia and Cardiovascular Disease (CVD). Its prevalence has been increasing over the last few decades and has become a modern-day epidemic.

The management and the treatment of obesity include lifestyle modifications and increased physical activity. Patients with a BMI of greater than 35 kg/m^2 with other comorbidities such as type two diabetes are considered eligible for bariatric surgery.

There is a plethora of evidence about the effect of bariatric surgery on medical outcomes including reversal of metabolic disease and reduction of negative cardiovascular outcome. Based on the few reported cases in the medical literature, increased cholinergic tone manifesting as bradycardia and delayed chronotropic response on stress test have been reported in post-bariatric surgery patients with significant weight loss.

We reported three cases of patients that underwent bariatric surgery and present with brady-arrythmias. While considering other complications of bariatric surgery, it is important to consider cardiac related sequalae such as bradyarrhythmias like sinus bradycardia, atrioventricular block, sick sinus syndrome due to delayed chronotropic response and irreversible atrioventricular defect could be helpful in clinical decision making. We recommend that an EKG and echocardiogram should be a performed as part as of a routine analysis in patients who had a bariatric surgery.

Keywords: Obesity; Bariatric Surgery; Cardiovascular Disease; Arrythmias-Bradycardia-Metabolic Equivalents; Heart Rate; Internal Medicine

Abbreviation

CAD: Coronary Artery Disease; HF: Heart Failure; CVD: Cardiovascular Disease; MI: Myocardial Infarction; RYGB: Roux-en-Y Gastric Bypass; GB: Gastric Banding; MACE: Adverse Cardiovascular Events; AVB: Atrioventricular Block; METs: Metabolic Equivalents; EKG: Electrocardiogram EKG; LV: Left Ventricle; SSS: Sick Sinun Syndrome; HR-Heart Rate

Introduction

Obesity is a major contributor to cardiovascular risk factors including hypertension, hyperlipidemia, Coronary Artery Disease (CAD), Heart Failure (HF), stroke, sleep apnea and arrhythmias. Its pathogenesis is linked to proinflammatory factors and vessel wall remodeling, among others [1]. Obesity accelerates atherosclerosis by promoting lipid deposition and atherothrombosis formation. It further activates the cytokines and interleukins causing endothelial dysfunction and vascular remodeling [1]. This translates into Cardiovascular Disease (CVD) events including CAD, Myocardial Infarction (MI) and stroke. Excess visceral adiposity leads to the activation of renin-angiotensin-aldosterone system, cytokine gene expression and increased systemic

circulation of proatherogenic factors [1]. This in turn leads to myocardial fat accumulation, increased stroke volume, cardiac wall remodeling and fibrosis manifesting as heart failure. Similar mechanisms lead to left atrial enlargement and fibrosis contributing to arrhythmogenesis [1]. Similar mechanisms lead to left atrial enlargement and fibrosis contributing to arrhythmogenesis [2].

The etiologies leading to obesity could be biological, psychosocial, socioeconomic and environmental factors. Although unhealthy dietary habits play a major role, socioeconomic factors also play a major role in the high prevalence of obesity and its complications, particularity among minority populations [3,4]. A higher BMI was strongly associated with higher comorbid cardiovascular risk factors of the BMI-related deaths, 41% were notably due to cardiovascular diseases [1,5].

Lifestyle modifications and increased physical activity are the initial modalities recommended in the management of obesity. Patients with a BMI 35 kg/m^2 or greater with serious obesity-related comorbidities are considered eligible for bariatric surgery [6]. The commonly performed bariatric surgeries include sleeve gastrectomy, Roux-en-Y Gastric Bypass (RYGB) and Gastric Banding (GB) [6]. Sleeve gastrectomy is currently the most common performed owing to lower risk of complications. The benefits of bariatric surgery include greater long-term weight loss, reduction of Major Adverse Cardiovascular Events (MACE) and cardiovascular mortality [7]. In our article, we present several cases in which the surgery caused significant arrythmias like severe bradycardia and even AVB.

Case Report

Case 1

50 years old female, with a history of obesity and BMI 42 underwent gastric bypass surgery. Four months after her surgery, she was admitted due to dizziness and severe nausea, with no other complaints. She had lost 25 kg in the interim period between his surgery and his presentation to the Emergency Room. At admission, she was found to have bradycardia (45 bpm) on EKG. No fever, blood pressure in the normal range and normal nutrition.

A head CT scan was performed with no evidence of abnormal findings. She was examined by a neurologist without any abnormal neurological findings, including a lack of explanation for her dizziness. She was then examined by an otolaryngologist and again, there were no abnormal findings to explain her dizziness. She was hospitalized in an Internal Medicine ward with cardiac monitoring and underwent the following investigation:

Echocardiogram - systolic function of the heart is normal, with no evidence of a valvular problem.

Holter ECG for 24 hours: sinus rhythm, heart rate average for 24 hours 40-45, without evidence of electrical conduction disorder except for bradycardia. A series of laboratory tests were performed including: complete blood count, chemistry, immune serology (C3/C4/ANA and ANCA), ferritin, folic acid and vitamin B12, all of which were found to be normal (Fig. 1).

A stress test was performed in order to rule out a chronotropic incompetence condition. The patient's resting HR was 48. At stage I of exercise, the patient's heart rate was 61. The patient did not experience a significant rise in heart rate until the later part of stage III at 10.4 Metabolic Equivalents (METs) where her rate increased to 133 beats per minute. The patient did not achieve target HR until stage IV of exercise when her HR did increase to 148 which was 88% of age-predicted HR. During the course of her care, the patient's pulse improved to between 60 and 70 bpm without specific intervention. The patient was followed up by the family doctor and all symptoms had resolved.

Case 2

47 years old male, with morbid obesity, BMI of about 42 kg/m^2. There was a recent diagnosis of OSA with no specific treatment. He underwent SLEEVE gastrectomy 4 months ago and lost about 30 kg of his weight. He also has a background hyperlipidemia treated with statins.

For about 10 days, he felt weakness and intermittent presyncope. At the reception to the Emergency Room, he had normal saturation, no fever and blood pressure 120/70. An ECG was performed and the patient was found to have bradycardia 35 beats per min. Second degree type Mobitz type one. A head CT scan was performed with no abnormal finding. CTA of lung was normal with no signs of pulmonary emboli. He was next examined by a neurologist. There were no central signs found to explain the dizziness (Fig. 2).

The patient was transferred to the Cardiology Department. He underwent several investigations that included echocardiogram and cardiac catheterization. Both tests were negative for any abnormal findings. Laboratory tests were performed and included C3/C4/ANA/ANCA, hemoglobin, ferritin, folic acid and vitamin B12, all of which was in the normal range.

During his hospitalization in the ward for cardiac monitoring, several episodes of AVB were observed during sleep together with the appearance of an episode of OSA. The patient was connected to a CPAP machine and the disorder disappeared. After discharge and follow up in the community, the patient's symptoms did not return.

Case 3

40 years old female, known to have type 2 diabetes, treated with oral medications, had gastric bypass surgery 4 months before admission and lost 30 kg. She was admitted to the hospital due to a feeling of palpitations for 5 minutes which passed spontaneously.

On admission to the emergency room, pulse was 60 beats per minute, no fever, normal saturation and blood pressure was in the normal range. An EKG was performed and they were found to have sinus rhythm, 40 per minute without additional conduction disturbances.

While waiting in the emergency department she complained of palpitations, an EKG was performed again and this time it demonstrated atrial flutter. A few seconds later the patient developed sinus bradycardia. An echocardiogram was performed and had no abnormal findings. Blood tests were taken including C3/C4/ANA/ANCA, hemoglobin, ferritin, folic acid and vitamin B12. All tests were within the normal range. The patient was eventually found to have sick sinus syndrome and they were admitted to ICU and a permanent pacemaker was implanted. After treatment and follow up in the community, the patient was found to be stable and symptom free (Fig. 3).

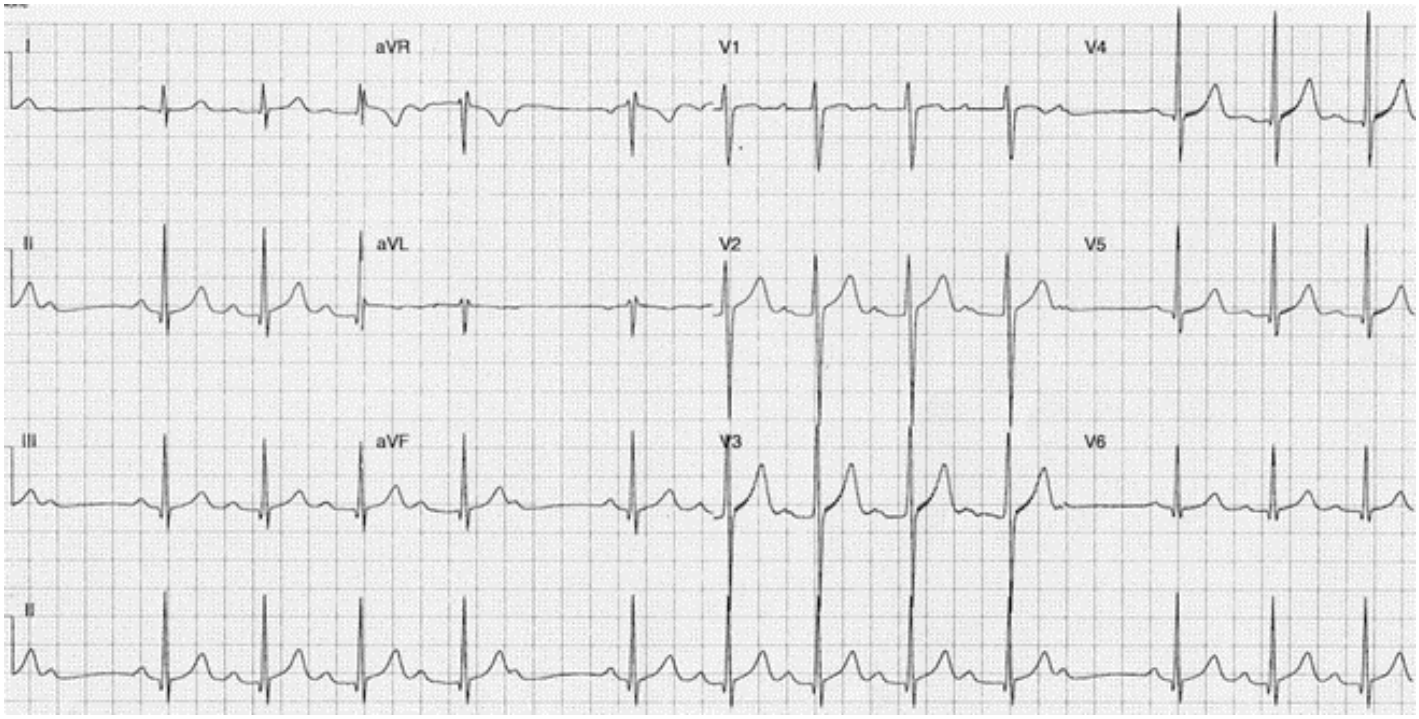

Figure 1: II Degree AV Block Mobitz I.

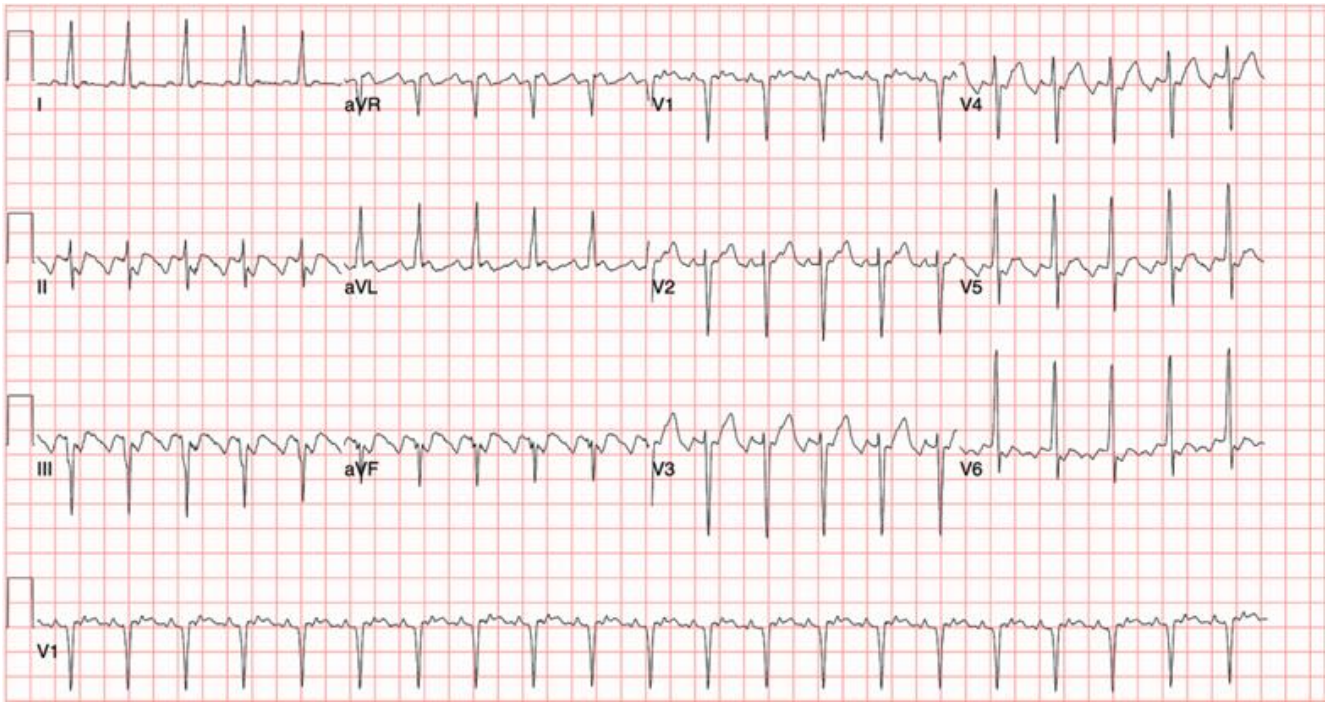

Figure 2: Atrial flutter.

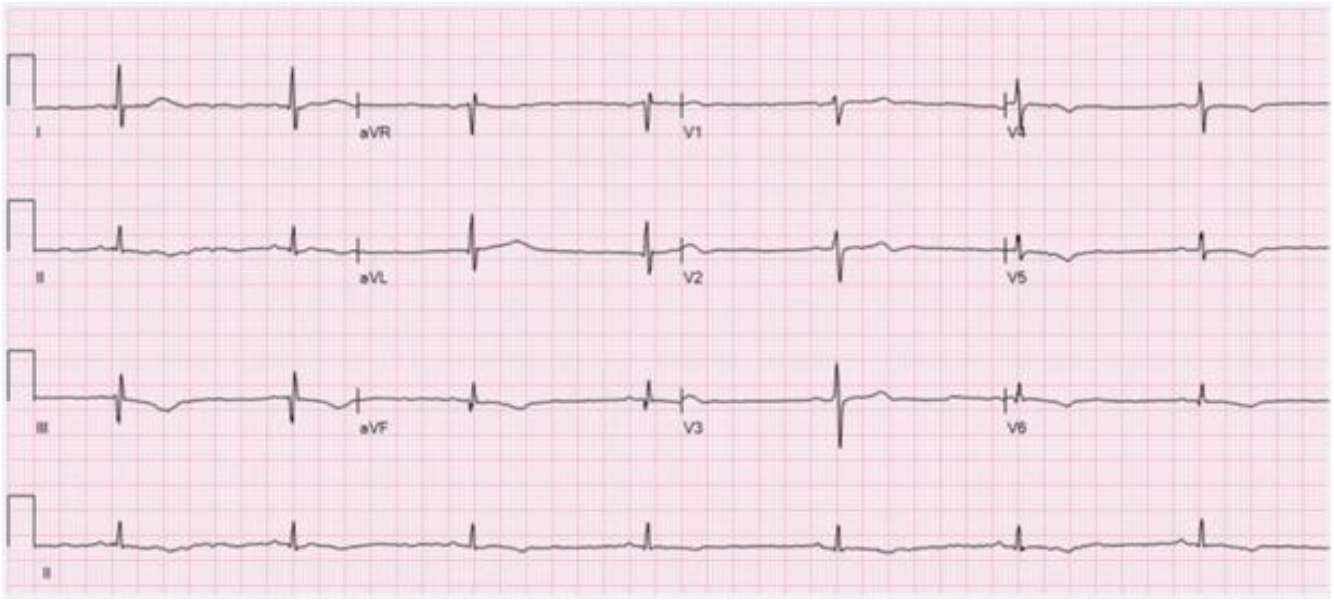

Figure 3: Sinus bradycardia.

Discussion

Patients have bariatric surgery with the desired end goals of weight reduction and/or improve metabolic function. However, it is not without physiological ramifications. Based on the few reported cases in the medical literature, increased cholinergic tone manifesting as bradycardia and delayed chronotropic response on stress test has been reported in post-bariatric surgery patients with significant weight loss [8-10].

Increased parasympathetic and reduced sympathetic activity after significant weight loss is well documented in the literature. A study by Malik, et al., were the first to formally document asymptomatic sinus bradycardia after bariatric surgery-induced weight loss [11]. Analyzing data for 24 months into the post-operative period, they showed an increased incidence of asymptomatic bradycardia in patients who underwent large volume weight loss associated with bariatric surgery.

In addition to the metabolic effect like decreasing insulin resistance, bariatric surgery weight loss is associated with a decrease in Left Ventricle (LV) mass, improvement in LV systolic function and diastolic dysfunction and overall improved cardiac function due to favorable effects of gut hormones such as Glucagon-Like Peptide-1 (GLP-1) and ghrelin [12]. On the other hand, massive weight loss comes with significant changes in hormone levels and alterations in autonomic function with a decrease in sympathetic tone and an increase in vagal tone.

Leptin has been shown to cause sympathetic overdrive in obese individuals through its sympatho-excitatory effect on the hypothalamus and its levels are directly proportional to BMI and heart rate [13]. The increase in leptin along with other endogenous factors such as insulin and cytokines contribute to the development of hypertension, diabetes mellitus and atherosclerosis in obese subjects. As leptin levels decrease with weight loss, vagal tone is augmented and sympathetic overtone on the sinus node is withdrawn, thus causing bradycardia [13]. The direct relationship between leptin and BMI also explains the reversal of metabolic diseases without treatment following weight loss, such as in this case.

The patient in case one presented with dizziness due to bradycardia at rest. She then performed a stress test which increased her pulse to within 88% of age-predicted HR. We hypnotized that the patient had cholinergic tone at baseline at rest and hence needed higher sympathetic system activation to overcome the cholinergic tone, which could only be achieved with high intensity and heavy physical activity.

The second patient presented with AVB due to OSA that worsened after bariatric surgery. Some studies reported high prevalence of OSA amongst patients undergoing bariatric surgery ranging between 77% and 88% [14,15]. Previous studies also have shown a high prevalence of OSA in patients with obesity despite the absence of patient-reported symptoms [16]. Patients' planning bariatric surgery may under-report symptoms for various reasons. Possible explanation for the low reports of functional impairment is the inability to recognize sleepiness. We think that the main treatment for patients with OSA should include starting the patient on a CPAP and not insertion of a pacemaker. Our patient was connected to a CPAP and showed significant improvement in his clinical condition and improvement in arrhythmia.

The third patient with sinus node dysfunction tachy-brady type which is characterized by bradycardia and tachycardia. We assume that obesity is associated with many electrophysiological and structural changes. From an electrophysiological point of view, obesity has been linked to dysfunction of the autonomic nervous system, which is manifested in increased sympathetic activity and its effect on the electrical conduction of the heart. On the other hand, obesity plays a major role in the remodeling process of the left atrium, which contributes to the appearance of SAN-related arrhythmias [17-19].

Conclusion

Weight reduction after bariatric surgery can have an effect on sympathetic and parasympathetic stimulation. Leptin is an adipocytokine associated with tachycardia value that is significantly reduced after bariatric surgery. This combination may contribute to the development of or exacerbate sinus bradycardia and/or delayed chronotropic response. Bariatric surgeries have certain complications and one of the more important complications that should be taken into account are bradyarrhythmias like sinus bradycardia, AVB, SSS or irreversible atrioventricular block. We recommend that an EKG and echocardiogram should be a performed as part of a routine analysis in patients who have performed a bariatric surgery. This may be especially relevant in patients with bradycardia and other autonomic manifestations.

Conflict of Interests

The authors have no conflict of interest to declare.

References

1. Powell-Wiley TM, Poirier P, Burke LE, Després JP, Gordon-Larsen P, Lavie CJ, et al. Obesity and cardiovascular disease: a scientific statement from the American Heart Association. Circulation. 2021;143(21):e984-1010.
2. Lavie CJ, Pandey A, Lau DH, Alpert MA, Sanders P. Obesity and atrial fibrillation prevalence, pathogenesis and prognosis: effects of weight loss and exercise. J Am Coll Cardiol. 2017;70:2022-35.
3. Cozier YC, Yu J, Coogan PF, Bethea TN, Rosenberg L, Palmer JR. Am Racism, segregation and risk of obesity in the Black Women's Health Study. J Epidemiol. 2014;179:875-83.

4. Bell CN, Kerr J, Young JL. Associations between obesity, obesogenic environments and structural racism vary by county-level racial composition. Int J Environ Res Public Health. 2019;16:861.
5. Afshin A, Forouzanfar MH, Reitsma MB. Health effects of overweight and obesity in 195 countries over 25 years. N Engl J Med. 2017;377:13-27.
6. Arterburn DE, Telem DA, Kushner RF, Courcoulas AP. Benefits and risks of bariatric surgery in adults: a review. JAMA. 2020;324:879-87.
7. Bouchard P, Al-Masrouri S, Demyttenaere S, Court O, Andalib A. Long-term impact of bariatric surgery on major adverse cardiovascular events in patients with obesity, diabetes and hypertension: a population-level study. Obes Surg. 2022;32:771-8.
8. Shah R, Bansal N, Manocha D. A woman who lost weight at the cost of her heartbeat. Case Rep 2014;2014:bcr2014205359.
9. Sagheer S, Sheikh AB, Alkubeysi M. Delayed chronotropic response due to autonomic imbalance presenting as dyspnea on early exertion: a complication of bariatric surgery. Cureus 2018;10:e3212.
10. Block M, Jacobson LB, Rabkin RA. Heart block in patients after bariatric surgery accompanying sleep apnea. Obes Surg. 2001;11(5):627-30.
11. Malik MG, Franklin SM, Whigham LA, Castellanos A, Fontaine JM. Asymptomatic sinus bradycardia following bariatric surgery. Am J Cardiol. 2014;113:1049-53.
12. Ashrafian H, le Roux CW, Darzi A, Athanasiou T. Effects of bariatric surgery on cardiovascular function. Circulation. 2008;118:2091-102.
13. Lin YC, Huang J, Hileman S, Martin KH, Hull R, Davis M, et al. Leptin decreases heart rate associated with increased ventricular repolarization via its receptor. Am J Physiol Heart Circ Physiol. 2015;309:1731-9.
14. Frey WC, Pilcher J. Obstructive sleep-related breathing disorders in patients evaluated for bariatric surgery. Obes Surg. 2003;13:676-83.
15. O'Keeffe T, Patterson EJ. Evidence supporting routine polysomnography before bariatric surgery. Obes Surg. 2004;14:23-6.
16. Foster GD, Sanders MH, Millman R, Zammit G, Borradaile KE, Newman AB, et al. Obstructive sleep apnea among obese patients with type 2 diabetes. Diabetes Care. 2009;32:1017-9.
17. Tedrow UB, Conen D, Ridker PM. The long- and short-term impact of elevated body mass index on the risk of new atrial fibrillation the WHS (women's health study). J Am Coll Cardiol. 2010;55(21):2319-27.
18. Abed HS, Samuel CS, Lau DH. Obesity results in progressive atrial structural and electrical remodeling: implications for atrial fibrillation. Heart Rhythm. 2013;10(1):90-100.
19. Kindel TL, Strande JL. Bariatric surgery as a treatment for heart failure: review of the literature and potential mechanisms. Surg Obes Relat Dis. 2018;14(1):117-22.