CLINICAL ARRHYTHMIA
Case Report
https://doi.org/10.24207/jca.v38i1.3570

Syncope due to Recurrent Asystole in the Recovery Phase of Exercise Testing

Caio Vittor Roque de Almeida Reis[1], Israel Guilharde Maynarde[2], Luciana Fernandes Balestra[3,4], Giulliano Gardenghi[3,5*]

ORCID ID

Reis CVRA https://orcid.org/0009-0006-3971-9671
Maynarde IG https://orcid.org/0009-0003-6846-8670
Balestra LF https://orcid.org/0009-0006-0123-357X
Gardenghi G https://orcid.org/0000-0002-8763-561X

ABSTRACT

We describe a clinical case of a 20-year-old male who was referred for an exercise stress test, undergoing cardiologic follow-up for 11 years due to episodes of syncope. Shortly after the end of the exercise phase, he presented recurrent episodes of asystole, associated with loss of consciousness, followed by spontaneous recovery.

KEYWORDS: Syncope, vasovagal; Heart Arrest; Ergometry; Unconsciousness.

INTRODUCTION

The basic disorder that causes syncope is a sudden reduction in cerebral oxygenation due to decreased blood flow to the region. The mechanisms involved in this decreased flow are:

- Sudden drop in cardiac output;
- Sudden drop in blood pressure;
- Sudden increase in cerebral vascular resistance[1,2].

Vasovagal reaction, also known as neurally mediated syncope, is the most common cause of syncope in young people with a structurally normal heart[1]. In the post-exertion phase of the exercise test, such a reaction may manifest, resulting in hypotension and, in occasional cases, a cardioinhibitory response, which can lead to asystole.

From a physiological perspective, during exercise, blood is mobilized to the working muscles, with greater blood sequestration as the intensity of the effort increases. Combined with decreased venous return during the recovery phase, this can lead to a decrease in cardiac preload, a time when the heart may beat more empty and with excessive vigor. This vigor acts as the primary stimulus that triggers the vagal response by activating intracardiac sensory receptors called

1. Pontifícia Universidade Católica de Goiás – Medical School – Goiânia (GO), Brazil.
2. Hospital do Coração de Goiás – Department of Ergometry – Goiânia (GO), Brazil.
3. Hospital do Coração Anis Rassi – Department of Clinical Arrhythmology – Goiânia (GO), Brazil.
4. Hospital de Urgências de Goiás – Department of Clinical Cardiology – Goiânia (GO), Brazil.
5. Hospital Encore – Department of Scientific Coordination – Aparecida de Goiânia (GO), Brazil.
***Correspondence author:** coordenacao.cientifica@ceafi.edu.br
Received: 10 jan. 2025 | **Accepted:** 15 set. 2025
Section Editor: Juan Carlos Zerpa Acosta

mechanoreceptors or C fibers, located primarily in the inferolateral wall of the left ventricle. These mechanoreceptors are stimulated in situations where venous return is decreased (upright position) or in hypovolemia, triggering intense vagal activity (Bezold-Jarisch reflex)[1,2].

Hyperventilation may also occur during exertion. The resulting drop in carbon dioxide causes cerebral vasoconstriction, increasing cerebrovascular resistance and, in some situations, decreasing brain perfusion, which can lead to syncope. Secher et al.[3] demonstrated that, during intense exercise, sympathetic vasoconstrictor tone can overwhelm the metabolic vasodilatory stimulus in the brain, resulting in decreased cerebral blood flow and syncope[2,3].

All the mechanisms described in the previous paragraph can exacerbate inadequate cardiovascular responses in susceptible individuals during the exercise test, particularly during the post-exercise recovery phase.

In this report, we describe a clinical case of a 20-year-old male patient who was referred for an exercise stress test, having been under cardiological follow-up for 11 years due to episodes of syncope, who presented significant asystole and loss of consciousness after the effort phase of the exercise stress test.

CASE REPORT

A 20-year-old man was referred for an exercise stress test to adjust his physical training. He had been followed by a cardiologist for 11 years due to episodes of syncope, primarily related to venipuncture. He had previously performed a sensitized tilt test with 6-second asystole, and had also presented two episodes of syncope per year after physical exertion and venipuncture.

The exercise stress test was performed according to the Ellestad protocol, reaching a maximum heart rate (HR) of 199 bpm. The patient was asymptomatic during the exercise phase. Initial blood pressure was 120/80 mmHg and reached 190/85 mmHg at peak exercise (SBP variation of 4.6 mmHg/MET). No arrhythmias were observed during the test. Within the first minute of recovery, he began feeling unwell, followed by syncope.

Fig. 1 shows the electrocardiographic tracing. In the first line, it can be seen that a reduction in HR and some artifacts precede the onset of the pause. The artifacts are related to the loss of postural tone and the resulting collapse of the patient, who, despite reporting discomfort, presented a sudden pause while in an orthostatic position.

It is worth noting that no electrode disconnection was observed during the event. The occurrence of myoclonus, along with the attending physician's verification of electrode positioning, may explain the artifacts presented. Isolated junctional escape beats during the pause and the behavior of attempting to restart electrical activity with junctional escape beats followed by a sinus beat with atrioventricular conduction and new sinus pauses, in the authors' opinion, reinforce the exacerbated parasympathetic activity, which, physiologically, may reinforce the occurrence of the recorded pause.

The 12-lead electrocardiogram also demonstrated a sinus pause of approximately 30 seconds, followed by junctional escape beats and a new episode of sinus pause of approximately 8 seconds, 9 minutes into recovery, during an attempt to stand (Fig. 2). Adding the total asystole time in the episodes documented after the exercise stress test, the total was 38 seconds. Blood pressure was measured during recovery, when the junctional beats began. It was approximately 70/30 mmHg—an expected decrease, considering the direct relationship between the significant reduction in cardiac output and the absence of beats and the probable decline in peripheral resistance (Fig. 3).

The patient was kept supine with his lower limbs elevated to improve venous return until complete and spontaneous recovery of consciousness, with no further cardiopulmonary resuscitation measures required. He was referred for follow-up with an attending cardiologist, where the initiation of an autonomic rehabilitation program was discussed, including moderate-intensity aerobic exercise and moderate-intensity resistance training, with an emphasis on the lower limbs. He was advised to adopt countermaneuvers in cases of prodromal events, as well as general and dietary measures, such as increased fluid intake and a three-hourly diet.

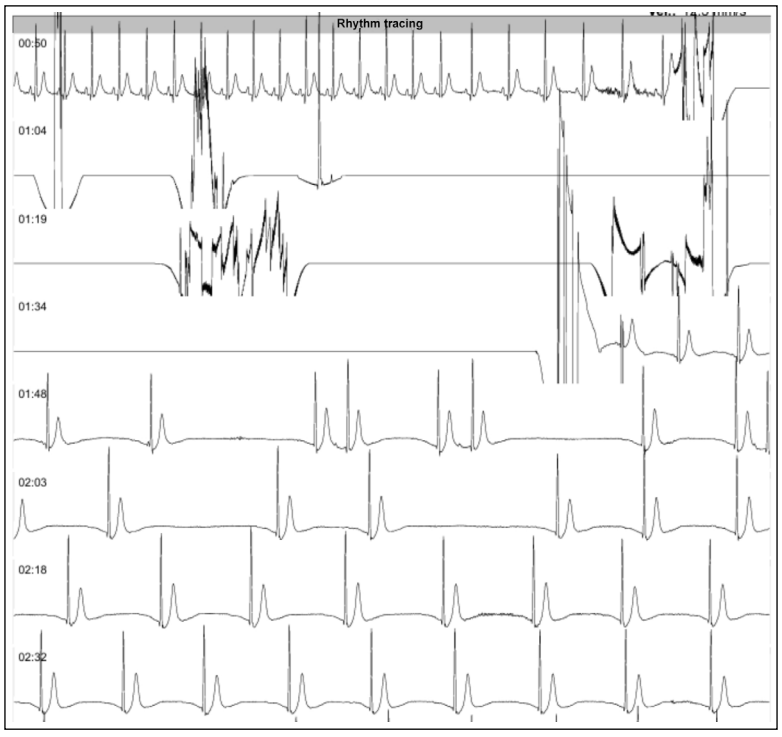

Figure 1. Electrocardiographic tracing showing the sinus pauses that occurred during the syncope event in the exercise test.

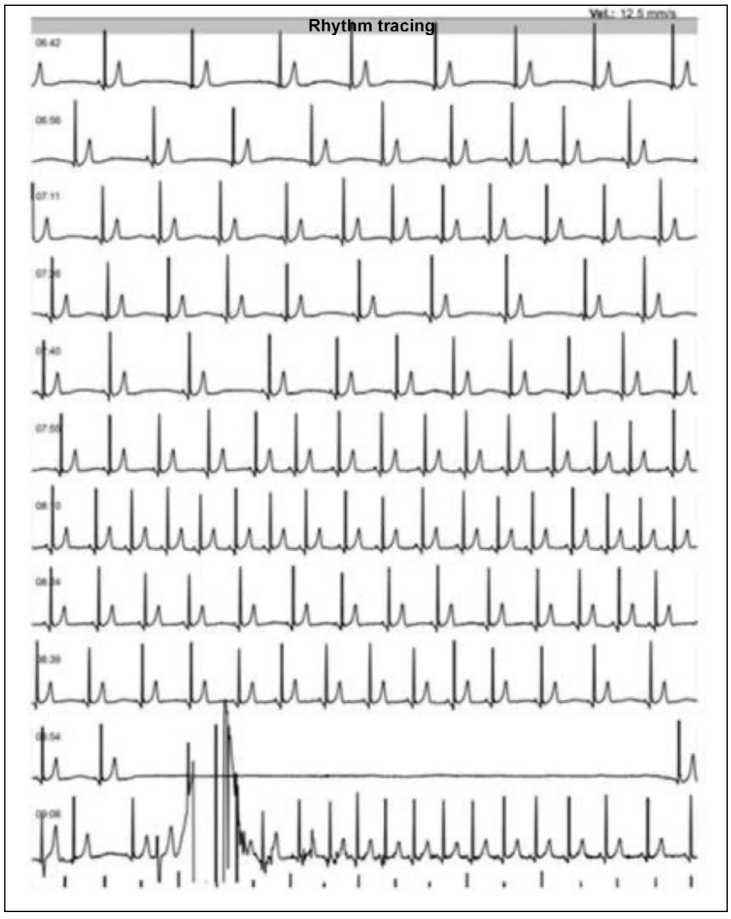

Figure 2. New sinus pause documented in the electrocardiographic record, during the recovery phase of the exercise test, after an attempt at orthostasis.

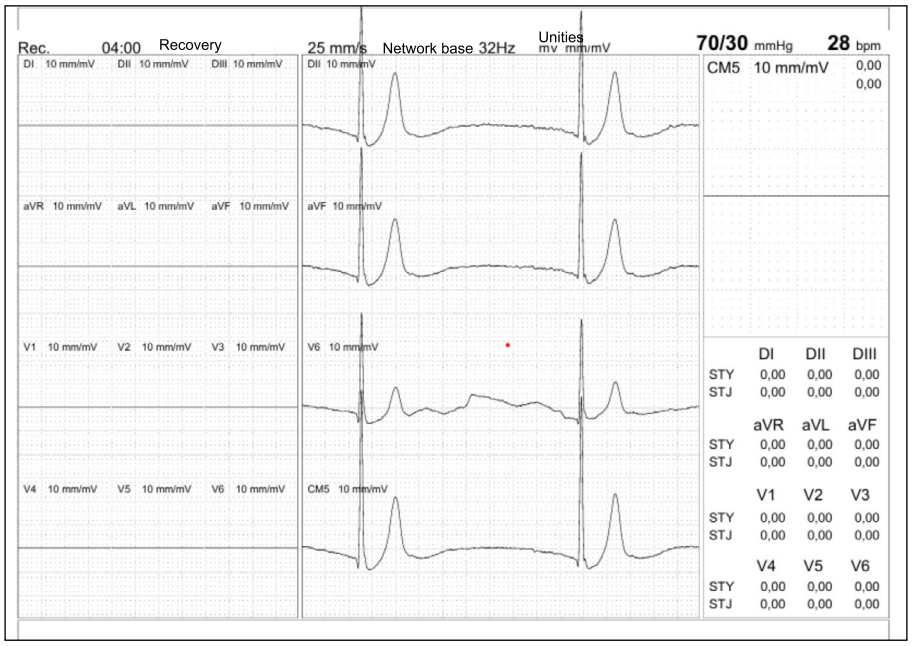

Figure 3. Measurement of blood pressure at the onset of junctional escape beats and placement of the patient in the supine position.

DISCUSSION

Exercise stress testing can be indicated in the initial evaluation of athletes of all ages, allowing for the identification and analysis of clinical responses, as well as hemodynamic and electrocardiographic changes, and the early detection of cardiovascular disease. Thus, it contributes to prognosis in both asymptomatic and non-symptomatic individuals[4]. Its indication also contributes to the assessment of cardiorespiratory fitness, whether during the examination or in monitoring the patient's progress over time[4].

The occurrence of neurally mediated syncope after physical stress testing is estimated at 0.3 to 3% of patients undergoing the test. This event is generally considered benign and self-limited, with a poor prognosis when secondarily associated with heart disease[5,6]. The decrease in HR in the first minute of recovery, in relation to the HR reached during peak effort, allows for the evaluation and inference of cardiac vagal modulation[7].

Although the reflex response presents similarly among patients, current literature reveals significant variation in both the onset time and duration of asystolic episodes. This diversity in presentation may be explained by factors such as vagal modulation and the circulatory status of each individual, which causes interpersonal variation in the manifestation of the reaction[8].

In this report, no structural or functional cardiac alterations were identified. However, within the first minute of the recovery phase of the exercise stress test, the patient began to experience malaise followed by syncope. Fleg and Asante[9] described the case of a 52-year-old man, with no evidence of structural diseases, who presented an episode of asystole for 11 seconds, 10 minutes after reaching maximum effort in the exercise test.

The symptom of syncope alone does not establish the cause of global cerebral hypoflow, and syncopal events can occur in individuals with macroscopic or genetic heart disease, such as channelopathies, as well as in individuals without the disease, as in the case presented. The characteristics of the event are essential in this direction, so that syncope after, rather than during, intense exertion, not preceded by chest pain or followed by palpitations, usually reinforces the benign nature of the event. The neurally mediated response, even with such an exuberant cardioinhibitory component, is not associated with sudden death[9].

In general, cardioinhibitory responses are often reproduced during the tilt table test. This test is used to investigate syncope episodes in individuals predisposed to them through an orthostatic stimulus. In most patients, the cardioinhibitory response is the predominant reflex, despite the knowledge that a positive tilt table test indicates the presence of a vasodilatory component, regardless of the response.

Furthermore, it is common for hypotension to precede bradycardia in the mechanism of syncope[6]. As an example, Winker et al.[10] demonstrated, in a positive tilt table test, a 22-year-old patient with no previous medical conditions who presented a drop in blood pressure, followed by bradycardia, sinus pause, apnea, and syncope. The study demonstrates the utility of the test in identifying distinct patterns of autonomic responses that contribute to syncope in individuals without structural heart disease.

The above reinforces the understanding that the symptom syncope is not exclusively a manifestation of structural diseases, but also a mechanism triggered by a baromediated vagal hyperstimulation after the vigorous contraction of an "empty heart"[2]. Hypotension, for example, preceding bradycardia in the reported case, suggests that the initial stimulus may be related to peripheral venous sequestration and decreased venous return, which occurs in the orthostatic position, thereby corroborating the hypothesis that the vagal response is secondary[3,10].

In this sense, Hirata et al.[11] described a case of asystole and syncope triggered by strenuous exercise in a man without organic heart disease in their study. The observation of asystole in this case demonstrates a dysfunction in the autonomic regulation of the heart.

The patient in this report was 20 years old when he presented with asystole after exercise stress testing. Considering the possibility of pacemaker implantation in reflex asystole, the European Society of Cardiology (ESC) syncope and pacemaker guidelines, published in 2021, restrict the indication to those over 40 years of age with recurrent events, without prodromes, and refractory to established therapies. Cardioinhibitory predominance, with sinus or ventricular pauses documented in spontaneous episodes, using an event monitor, or during a tilt test in the syncope laboratory, associated with carotid sinus compression or a reflex event, achieves a grade II recommendation and a level of evidence of A or B, respectively. The ESC also emphasizes that the absence of a documented cardioinhibitory reflex contraindicates pacemaker implantation, which is reserved as a last-line therapy for a limited number of patients with severe reflex syncope that is refractory to other approaches[12].

Another therapeutic option has been proposed in recent years, involving autonomic neuromodulation through the isolation and ablation of areas within the intracardiac parasympathetic ganglionic plexus via endocardial ablation. This procedure, although widely discussed, has not yet been included in guidelines[13].

CONCLUSION

In this report, we hypothesized that asystolic syncope during the recovery phase of exercise testing was caused by an inadequate cardiac autonomic response, a consequence of vagal hyperstimulation. This mechanism highlights the role of the baromediated reflex in individuals without structural cardiac alterations.

Autonomic regulation plays a crucial role in the genesis of syncope. It should be considered by cardiologists and other healthcare professionals involved in exercise programs, particularly when there is a history of neurally mediated syncope.

CONFLICT OF INTEREST

Nothing to declare.

AUTHOR CONTRIBUTIONS

Conceptualization: Balestra LF, Gardenghi G; Supervision: Gardenghi G; **Investigation:** Balestra LF, Maynarde IG; **Writing – First Draft:** Reis CVRA, Maynarde IG, Gardenghi G; **Writing – Review and Editing:** Balestra LF, Gardenghi G; **Final approval:** Gardenghi G.

DATA AVAILABILITY STATEMENT

The data needed to reproduce the findings reported in this article cannot be shared at this time due to legal/ethical reasons that ensure the confidentiality of the individual described here.

FUNDING

Not applicable.

ACKNOWLEDGMENTS

Not applicable.

REFERENCES

1. Sutton R. Reflex syncope: Diagnosis and treatment. J Arrhythm. 2017;33(6):545-52. https://doi.org/10.1016/j.joa.2017.03.007
2. Gardenghi G, Balestra LF. Tratamento não-farmacológico da síncope neuromediada. J Card Arrhythm [Internet]. 2011 [cited Sep 19, 2025];24(3):145-50. Retrieved from https://jca.org.br/jca/article/view/2579
3. Secher NH, Clausen JP, Klausen K, Noer I, Trap-Jensen J. Central and regional circulatory effects of adding arm exercise to leg exercise. Acta Physiol Scand. 1977;100(3):288-97. https://doi.org/10.1111/j.1748-1716.1977.tb05952.x
4. Ghorayeb N, Costa RVC, Castro I, Daher DJ, Oliveira Filho JA, Oliveira MAB, et al. Diretriz em cardiologia do esporte e do exercício da Sociedade Brasileira de Cardiologia e da Sociedade Brasileira de Medicina do Esporte. Arq Bras Cardiol. 2013;100(1 Supl. 2):1-41. https://doi.org/10.5935/abc.2013S002
5. Ghorayeb N, Dioguardi GS. Tratado de cardiologia do exercício e do esporte. São Paulo: Atheneu; 2007.
6. Mallet ALR. Síncope: abordagem diagnóstica. Rev SOCERJ [Internet]. 1999 [cited Sep 19, 2025];12(1):422-9. Retrieved from http://sociedades.cardiol.br/socerj/revista/1999_01/a1999_v12_n01_art11.pdf
7. Ulucan Ş, Kaya Z, Keser A, Katlandur H, Karanfil M, Ateş İ. Deterioration of heart rate recovery index in patients with erectile dysfunction. Anatol J Cardiol. 2016;16(4):264-9. https://doi.org/10.5152/AnatolJCardiol.2015.6132
8. Tse HF, Lau CP. Exercise-associated cardiac asystole in persons without structural heart disease. Chest. 1995;107(2):572-6. https://doi.org/10.1378/chest.107.2.572
9. Fleg JL, Asante AVK. Asystole following treadmill exercise in a man without organic heart disease. Arch Intern Med. 1983;143(9):1821-2.
10. Winker R, Frühwirth M, Saul P, Rüdiger HW, Pezawas T, Schmidinger H, et al. Prolonged asystole provoked by head-up tilt testing. Clin Res Cardiol. 2006;95(1):42-7. https://doi.org/10.1007/s00392-006-0310-1
11. Hirata T, Yano K, Okui T, Mitsuoka T, Hashiba K. Asystole with syncope following strenuous exercise in a man without organic heart disease. J Electrocardiol. 1987;20(3):280-3. https://doi.org/10.1016/s0022-0736(87)80027-4
12. Glikson M, Nielsen JC, Kronborg MB, Michowitz Y, Auricchio A, Barbash IM, et al. 2021 ESC Guidelines on cardiac pacing and cardiac resynchronization therapy. Eur Heart J. 2021;42(35):3427-520. https://doi.org/10.1093/eurheartj/ehab364. Erratum in: Eur Heart J. 2022;43(17):1651. https://doi.org/10.1093/eurheartj/ehac075
13. Ali M, Pachon Maetos JC, Kichloo A, Masudi S, Grubb BP, Kanjwal K. Management strategies for vasovagal syncope. Pacing Clin Electrophysiol. 2021;44(12):2100-8. https://doi.org/10.1111/pace.14402