Sustained Accelerated Idioventricular Rhythm in a Centrifuge-Simulated Suborbital Spaceflight

Rahul Suresh; Rebecca S. Blue; Charles Mathers; Tarah L. Castleberry; James M. Vanderploeg

INTRODUCTION: Hypergravitational exposures during human centrifugation are known to provoke dysrhythmias, including sinus dysrhythmias/tachycardias, premature atrial/ventricular contractions, and even atrial fibrillations or flutter patterns. However, events are generally short-lived and resolve rapidly after cessation of acceleration. This case report describes a prolonged ectopic ventricular rhythm in response to high G exposure.

CASE REPORT: A previously healthy 30-yr-old man voluntarily participated in centrifuge trials as a part of a larger study, experiencing a total of 7 centrifuge runs over 48 h. Day 1 consisted of two $+G_z$ runs (peak +3.5 G_z, run 2) and two $+G_x$ runs (peak +6.0 G_x, run 4). Day 2 consisted of three runs approximating suborbital spaceflight profiles (combined $+G_x$ and $+G_z$). Hemodynamic data collected included blood pressure, heart rate, and continuous three-lead electrocardiogram. Following the final acceleration exposure of the last Day 2 run (peak +4.5 G_x and +4.0 G_z combined, resultant +6.0 G), during a period of idle resting centrifuge activity (resultant vector +1.4 G), the subject demonstrated a marked change in his three-lead electrocardiogram from normal sinus rhythm to a wide-complex ectopic ventricular rhythm at a rate of 91–95 bpm, consistent with an accelerated idioventricular rhythm (AIVR). This rhythm was sustained for 2 m, 24 s before reversion to normal sinus. The subject reported no adverse symptoms during this time.

DISCUSSION: While prolonged, the dysrhythmia was asymptomatic and self-limited. AIVR is likely a physiological response to acceleration and can be managed conservatively. Vigilance is needed to ensure that AIVR is correctly distinguished from other, malignant rhythms to avoid inappropriate treatment and negative operational impacts.

KEYWORDS: accelerated idioventricular rhythm, acceleration, commercial spaceflight, spaceflight participant.

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Hypergravity exposures are known to induce cardiac dysrhythmias in healthy, asymptomatic subjects undergoing centrifuge training and aerobatic flight.[7] The most common aberrant rhythms documented during G exposure include sinus dysrhythmias, sinus bradycardias, and premature atrial and ventricular complexes.[5,7,11] Findings from primarily centrifuge studies suggest that these dysrhythmias represent a physiological response to $+G_z$ (head-to-toe) stress as they rarely reduce a pilot's tolerance of acceleration exposures, are short-lived, and resolve once acceleration exposures cease.[5] However, 3–5% of subjects develop more concerning dysrhythmias such as paroxysmal supraventricular tachycardia, ventricular bigeminy and trigeminy, and ventricular tachycardia, each of which have a physiological potential for progression to unstable or malignant rhythms.[5,7] In theory, such progression could cause incapacitation secondary to reduced cardiac output and a corresponding decrease in cerebral perfusion.[5,7] Even so, such rhythms, when documented during centrifuge training, rarely lead to termination of training or intervention—most are observed and rarely, if ever, do they progress.[5,7]

The majority of ventricular dysrhythmias noted during centrifugation have been single or paired premature ventricular complexes (PVCs, 68%), while significantly fewer are characterized as triplet PVCs or ventricular tachycardia (5%).[5,7] While ventricular tachycardia in most medical scenarios would be highly concerning, episodes of ventricular tachycardia during

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centrifuge exposure are nearly all short-lived and resolve with cessation of G exposure.[5,7] One large retrospective study of over 1100 human centrifuge exposures identified 24 cases of ventricular tachycardia, all of which spontaneously resolved, generally in under 30 s, during or after termination of acceleration.[7] Overall, ventricular dysrhythmias documented during hypergravity exposure to date have been short, rarely symptomatic, and self-limited.

Much of the human centrifuge experience to date has been in $+G_z$ accelerations, as these are the primary acceleration forces experienced by pilots in high-performance flight. However, with the development of the commercial spaceflight industry, there is increasing human exposure to $+G_x$ (chest-to-back) acceleration, as would be experienced during rocket-powered flight. While such exposure is well tolerated in career astronauts, less is known regarding how untrained laypersons may respond to hypergravitational force in any direction. Further, it is unclear whether $+G_x$ exposures may further place individuals at risk of aberrant cardiac rhythms. Recent studies have begun to test layperson subjects in centrifuge-simulated spaceflight, which has led to the collection of novel data regarding cardiovascular responses of laypersons exposed to such acceleration.[1]

Here we describe the development of a sustained ventricular dysrhythmia, consistent with accelerated idioventricular rhythm (AIVR), in a healthy, young male subject without history of cardiac disease exposed to centrifuge acceleration simulating suborbital spaceflight. His participation was part of a larger trial that has been previously published.[1]

CASE REPORT

A 30-yr-old Caucasian man with no significant medical history volunteered for participation in the ongoing centrifuge trial.[1] He completed all screening for participation, including documentation of current physical and medical status, with no significant medical history or physical limitations reported. He also submitted a standard 12-lead electrocardiogram (ECG) that demonstrated normal sinus rhythm at 79 bpm with normal intervals, early repolarization, and an otherwise normal ECG.

The subject underwent seven centrifuge runs over 2 d at the National Aerospace Training and Research Center centrifuge (Environmental Tectonics Corp., Southampton, PA) as part of the larger trial.[1] Day 1 consisted of two $+G_z$ runs (peak +3.5 G_z, run 2) and two $+G_x$ runs (peak +6.0 G_x, run 4). Day 2 consisted of three runs approximating suborbital spaceflight profiles (combined $+G_x$ and $+G_z$).[1] A high-fidelity multimedia system within the passenger gondola was used to provide audiovisual simulation to enhance the realism of the spaceflight experience. Audiovisual simulation consisted of a projected monitor screen with cockpit-simulated view of Earth and space images imitating real-time views throughout the flight profile, with matching flight-related sounds such as rocket engine firing. Audio communication with the participants was available via gondola speakers and a live microphone, and real-time video images of the subject's face and torso were projected in real time to a test operator and medical monitors while subjects were in the gondola. The subject was monitored with a three-lead ECG throughout the G exposure, with continual telemetered streaming of data to the medical monitor.

Prior to initiation of centrifuge runs, the subject's resting blood pressure (BP) and heart rate (HR) were recorded. The subject was taught basic anti-G straining (AGSM) and the "hook" (L-1 closed-glottis variant) maneuver. He was advised to use muscular strain during the $+G_z$ exposure, but to use the hook maneuver only in the event of grey-out or light-headedness. He was also cautioned against sudden head movements during centrifuge trials to avoid triggering Coriolis symptoms.

The subject's baseline hemodynamics, including a BP of 110/70 mmHg and HR of 74 bpm, were not significantly different from the average for his age and sex, as presented in **Table I**. He completed all Day 1 training profiles without incidence. During the first of the two full-acceleration simulated suborbital spaceflights on Day 2, he demonstrated occasional PVCs following $+G_x$ exposures during both boost and reentry acceleration profiles (boost: combined $+G_x$ and $+G_z$, peak +4.0 G_z and +3.5 G_x; respectively; reentry: $+G_x$ exposure only, peak +6.0 G_x). He described no palpitations or other complaints related to the PVCs.

Following the final acceleration exposure of the last Day 2 run (combined reentry, peak +4.5 G_x and +4.0 G_z, resultant vector +6.0 G), during a period of idle resting centrifuge activity (resultant vector +1.4 G), the patient demonstrated a marked change in his three-lead ECG from normal sinus rhythm to a wide-complex ectopic ventricular rhythm at a rate of 91–95 bpm, consistent with an accelerated idioventricular rhythm (AIVR; **Fig. 1**; **Table II**). This rhythm was sustained for 2 min and 24 s before reversion to normal sinus rhythm at a rate of 84–89 bpm for the remainder of his idle spin and gondola stop, a period of greater than 3 min of observation. At this time the subject was removed from the gondola and queried regarding symptoms. He denied any symptoms of palpitations, lightheadedness, malaise, chest discomfort, or any other indication of symptomatology related to the change in cardiac rhythm. Repeat 12-lead ECG demonstrated normal sinus rhythm at a rate of 88 with normal intervals, without ST elevation or depression, and without evidence of PVCs or ectopic ventricular

Table I. Baseline and Prespin Hemodynamic Responses by Flight Phase for Case Subject Compared to Cohort Mean Response.

FLIGHT PHASE	SUBJECT	CONTROL MEAN
Baseline systolic (mmHg)	110	122.3 ± 12.8
Baseline diastolic (mmHg)	70	75.7 ± 9.5
Baseline heart rate (bpm)	74	68 ± 10.4
Prespin systolic (mmHg)	141.2 ± 4.2	136.1 ± 13.9
Prespin diastolic (mmHg)	87.8 ± 4.8	99.7 ± 27.8
Postspin systolic (mmHg)	131.8 ± 6.4	143.3 ± 14.2
Postspin diastolic (mmHg)	83.8 ± 2.6	84.9 ± 9.5
Prespin heart rate (bpm)	88.5 ± 10.8	72.1 ± 12.7
Postspin heart rate (bpm)	81.0 ± 13.3	74.6 ± 14.4

Control means are from subjects from the larger study population[1] matched by age and sex to our case subject (men ages 25–35, no medical disease, $N = 48$). BP: Blood pressure, HR: heart rate, bpm: beats per minute.

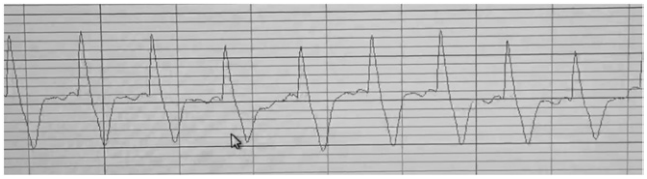

Fig. 1. The subject's 3-lead electrocardiogram demonstrated a wide-complex ectopic ventricular rhythm, consistent with an accelerated idioventricular rhythm, or AIVR.

rhythm. The subject was observed for an additional 1 h without any further symptoms or complaints.

DISCUSSION

The incidence of cardiac dysrhythmias during hypergravity exposures is well documented.[5,7,8] Dysrhythmias arising from the ventricle are predominantly single or couplet PVCs; a small but significant subset includes triplet PVCs and ventricular tachycardia.[5,7] Historically, ventricular dysrhythmias are usually short lived and resolve with cessation of G exposure.[7] We highlight here a case of a 30-yr-old male volunteer who developed sustained AIVR following centrifuge-simulated G forces like those anticipated during the launch and reentry profiles of short-duration, suborbital, commercial spaceflight. Despite experiencing sustained AIVR for more than 140 s, the patient remained entirely asymptomatic. Moreover, the dysrhythmia occurred following G exposure rather during G exposure.

AIVR is a wide-complex ventricular dysrhythmia with a heart rate above the ventricular escape rhythm of 30–40 bpm and less than 100 bpm.[8] Onset of AIVR is gradual and not paroxysmal, and can present as a regular or irregular rhythm, with or without atrioventricular dissociation.[8,12] AIVR manifests when ventricular pacemakers have an automaticity that is greater than the sinus rate (usually due to sinus bradycardia), but can also occur as an escape rhythm in the setting of atrioventricular block.[8,12] AIVR is rare in the general population. A review of 24-h ambulatory ECG monitoring of healthy volunteers with normal screening ECG obtained from 21 large clinical trials found incidence of spontaneous AIVR to be 0.3% during the monitoring period.[6] AIVR is most commonly seen during cardiac rhythm monitoring after myocardial ischemia, often suggesting reperfusion of previously ischemic tissue, with a prevalence between 10–15%.[2,3] Other factors known to predispose to AIVR include digoxin toxicity, metabolic disturbances, and hypoxemia.[12]

Sustained ventricular dysrhythmias are generally concerning for clinically significant hemodynamic instability and incapacitation. However, due to the relatively slow rate and nonparoxysmal onset and resolution of AIVR, precipitation of more rapid ventricular arrhythmias is rarely observed, and the rhythm does not generally precipitate hemodynamic compromise.[2,8] A recent study of patients with first-time myocardial infarction found that there was no difference in mortality at 12 mo between those that developed AIVR and those that did not.[2] Further, AIVR in young, healthy individuals without structural heart disease is considered benign, is self-limited, and does not require intervention.[12]

Several features of the dysrhythmia experienced by this subject support a benign etiology. First, the patient had no previous history of medical conditions or cardiovascular disease, and both pre- and postspin ECGs were normal. Further, the patient remained clinically asymptomatic for the entirety of the dysrhythmic event; the event itself was self-limited and did not require any intervention or treatment. There was no evidence of any hemodynamic instability at any time.

Typical and more concerning ventricular dysrhythmias, including ventricular tachycardia, are thought to be precipitated in part by increased sympathetic tone.[9] In contrast, onset of dysrhythmia in this case occurred during recovery from peak G exposure, a period of time when parasympathetic activity typically predominates. The timing of the AIVR following G exposure may be related to modulation of the autonomic nervous system during recovery. Previous studies have noted increased parasympathetic tone during recovery from acceleration, often indicated by frequent incidence of sinus bradycardia.[7] Decreased sympathetic tone and increased parasympathetic activity have been shown to correlate with AIVR incidence.[2] In the presence of frequent ectopy, increased parasympathetic tone provides an ideal electrical environment for the development of AIVR and mitigates predisposition to developing ventricular tachycardia.[2] As PVCs are well known to occur during centrifugation[7] and, in fact, were observed in this subject during other centrifuge runs and particularly $+G_x$ exposure, it

Table II. Mean Heart Rate Responses by Flight Phase for Case Subject Compared to Cohort Mean Response.

SPIN PROFILE	EXPOSURE	DURATION AT PEAK G	SUBJECT HR	COHORT MEAN HR
$+G_z$ Familiarization (+3.5 G_z)	Peak $+G_z$	15 s	122	131.3 ± 19.3
$+G_x$ Familiarization (+6.0 G_x)	Peak $+G_x$	15 s	95	92.4 ± 30.7
100% Integrated Spaceflight Simulation	Launch Peak ($+G_x/+G_z$)	5 s	157	139.7 ± 16.8
- Boost Peak +3.5 G_z, +4.0 G_x				
- Reentry Peak +6.0 G_x	Reentry Peak ($+G_x$)	5 s	80	96.7 ± 25.4
100% Integrated Spaceflight Simulation	Launch Peak ($+G_x/+G_z$)	5 s	162	140.9 ± 18.5
- Boost Peak +3.5 G_z, +4.0 G_x				
- Reentry Peak +4.0 G_z/+4.5 G_x	Reentry Peak ($+G_x/+G_z$)	5 s	153	134.0 ± 18.5
- Resultant +6.0 G				

Control means and SDs are from subjects from the larger study population[1] matched by age and sex to our case subject (men ages 25–35, no medical disease, $N = 48$). HR: Heart rate, presented in beats per minute (bpm).

seems likely that ectopic beats and a milieu of increasing parasympathetic tone may have precipitated a parasympathetically driven ectopy.

In this study, G exposure was designed to simulate suborbital spaceflight profiles that include a combination of $+G_z$ and $+G_x$ acceleration. Exposure to $+G_x$ acceleration is less well studied than $+G_z$ acceleration, particularly in layperson populations. However, another potential etiology leading to the development of the aberrant rhythm is one of mechanical stretch. Mechanoelectric feedback is a well-established mechanism that describes electrophysiological changes caused by changes in myocardial segment length; mechanical stretch of cardiac muscle can precipitate ectopic beats and occasional dysrhythmias.[4] Benign ventricular dysrhythmias, such as AIVR, can be triggered by ectopic beats, the risk of which increases with increasing ectopy.[10] Exposure to high $+G_x$ acceleration would result in cardiac mechanical deformation; it is possible that activation of stretch receptors during centrifugation may have led to increasing ectopy and the AIVR event.

Regardless of etiology, this event has raised the question of whether or not such aberrant rhythms, particularly when sustained, should prompt observation, intervention, or even termination of training or flight activities. Management of dysrhythmias including AIVR in the operational environment is dictated by risk tolerance, which itself is dependent on the risk for impairment, need for and extent of acute intervention, and the operational impact of any potential incapacitation. Our observations suggest that in young, otherwise healthy individuals experiencing combined $+G_z$ and $+G_x$ exposures, the development of AIVR is likely physiological rather than pathological. The likelihood of development of significant hemodynamic instability or conversion to unstable rhythms is low given the rhythm's parasympathetic drive and low risk when AIVR occurs in nonoperational circumstances. As in the general population, we believe that in such individuals without cardiac disease, AIVR in response to G exposure can be managed with simple observation. These principles should apply equally to anyone exposed to high G force, including both flight crew and participants. However, vigilance will be required to avoid confusing AIVR with ventricular tachycardia, as both dysrhythmias are uncommon, can appear similar on electrocardiographic tracings, and the management of the two conditions is quite different. Incorrect diagnosis, therefore, can have serious negative operational repercussions.

AIVR in an individual with prior cardiac history, including history of ischemic heart disease with percutaneous coronary intervention, myocardial infarction, dysrhythmias, implanted cardiac defibrillators, or cardiomyopathy, must be interpreted with caution. In those with ischemic heart disease, data suggest that AIVR noted in the postinfarct period does not portend increased adverse outcomes.[2] Even in those individuals who have a cardiac disease burden, intervention for AIVR is not indicated unless hemodynamic instability or unstable rhythms develop.[12] Commercial spaceflight is unique in that individuals with previously mentioned cardiac history may be cleared to fly. However, in such individuals with cardiac disease or with a high cardiac risk factor burden, development of AIVR during acceleration exposure must be managed cautiously as the prognostic implications are still unknown.

Dysrhythmias during and after acceleration exposure, including those of ventricular origin, are common, but generally short lived. In a healthy subject without heart disease, AIVR is likely a benign physiological response to high $+G_x$ acceleration or, alternatively, the combination of $+G_z$ and $+G_x$. We believe that this rhythm can be managed conservatively with observation, without need for termination of training, intervention, or restriction of operational capabilities. Due to the infrequent occurrence of this rhythm in aviation and spaceflight populations, increased vigilance is needed to ensure that AIVR and ventricular tachycardia are correctly distinguished to avoid inappropriate treatment and negative operational impacts. The incidence or consequence of AIVR in an individual with known significant cardiac disease is unknown and should be approached with caution.

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Erratum

Senese AL, Regis DP, Hall AA, Mahon RT, Cronin WA. *Propranolol effects on decompression sickness in a simulated DISSUB rescue in swine.* Aerosp Med Hum Perform. 2017; 88(4):385–391.

The authors of this article recently notified us of an error in reference 27. The correct reference should be as follows:

27. U.S. Navy Submarine Rescue System (SRS) Decompression Plan. Panama City (FL): U.S. Navy; 2014. Revision, 8 May 2014.

The online versions of the article have been corrected. We apologize for this error and any inconvenience.