Case Report

Acute Hemodynamic Decompensation Following Routine Phlebotomy
in a Patient with Chronic Volume Dysregulation:
A Five-Week Longitudinal Provocation Sequence

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Abstract

A 48-year-old male (DOB 7/5/1969) with a documented history of chronic volume dysregulation and suspected endocrine axis disruption underwent routine whole blood donation as a self-initiated provocation test. Within 24-48 hours, the patient experienced a transient hypercortisolemic state (subjective mania), followed by acute vestibular failure on day three, persistent nausea and orthostatic symptoms over the subsequent four weeks, and a near-syncopal collapse on day 34 triggered by THC-induced vasodilation. Throughout the collapse event, the patient maintained full consciousness despite documented blood pressure of 90/54 mmHg post-saline resuscitation. Serial laboratory evaluation revealed prerenal azotemia (BUN 27 mg/dL), borderline hypokalemia (K 3.5 mEq/L), maximally concentrated urine (SG 1.033), venous alkalosis (pH 7.44), complete suppression of eosinophils and basophils, and urine sodium of 203 mmol/L (massive renal sodium wasting despite volume depletion), while standard cardiac and structural workups returned negative. Longitudinal endocrine data spanning 2010 to 2026 demonstrate stable cortisol (10.3-13.2 mcg/dL) against declining aldosterone (6.5 to 4.0 ng/dL) with volatile intermediate values (1.5 ng/dL two weeks prior to the crisis), suggesting selective adrenal output preservation independent of the renin-angiotensin system. This case documents a complete provocation-response sequence with timed clinical and laboratory endpoints in a patient whose hemodynamic architecture operates outside standard physiological parameters.[*A note from* The Architect*: All PDF's of these labs and more are at jimcraddock.com*]

1. Presenting History

The patient is a male, age 48 at the time of the index event, with a multi-year history of chronic systemic volume dysregulation characterized by oscillating phases of apparent fluid excess and deficit, progressive salt cravings (shifting from sugar preference to salt preference over the preceding decade), episodic orthostatic symptoms, and a previously identified pituitary microgranuloma on dedicated pituitary MRI with contrast (no longer detectable on subsequent

imaging). Prior medical evaluation had been extensive but non-diagnostic. The patient had a known history of blood donation during an earlier volume-excess phase, which had been tolerated; however, a subsequent donation during that earlier period precipitated anemia, and the patient was advised by his physician to discontinue donation.

At the time of the February 2018 provocation event, the patient was physically active, exercising regularly, dating, and subjectively well. He elected to donate blood as a deliberate self-test of his suspected condition, reasoning that if the volume-dependent pathology were real, the donation would produce a measurable response. The patient was aware of this risk and proceeded intentionally.[*I was at a crossroads, believe institutional medicine, or believe in* Redacted Science (Craddock, 2025). *I needed to know in order to plan my life. Yes, it cost me years, but it told me the truth. Science IS Redacted*]

2. Provocation Event and Clinical Course

2.1 Timeline

Day / Date	Event	Clinical Significance
Day 0 Feb 1, 2018 1:00 PM	Routine whole blood donation (standard 470 mL unit)	Volume removal from already-compromised vascular compartment
Day 1 Feb 2, 2018	Patient reports near-manic state: elevated energy, heightened focus, subjective euphoria	Consistent with emergency ACTH/cortisol surge in response to acute volume depletion
Day 3 Feb 4, 2018 (Sunday)	Acute onset room-spinning vertigo upon waking. No nausea or diaphoresis initially. Ambulation possible only at an angle	Vestibular system failure secondary to cerebral perfusion deficit after ~72 hours of compensatory effort. Vestibular apparatus is highly sensitive to perfusion changes
Days 4-5 Feb 5-6	Vertigo persists. Patient manages at home	
Day 6 Feb 7, 2018	Emergency department visit #1. CT head, ECG x2, CMP, Troponin, Chest XR performed. All reported normal	Standard structural/cardiac workup negative. No assessment of volume status performed
Days 7-26 Feb 8-27	Vertigo gradually resolves. Replaced by persistent nausea and orthostatic symptoms (dizziness on positional change, including during exercise)	Autonomic instability during volume recovery phase
Day 26 Feb 27, 2018	Patient privately orders laboratory studies: Renin Activity 0.564 ng/mL/hr; Aldosterone 1.5 ng/dL; Urine Sodium 203 mmol/L; UA with pH 7.5 and SG 1.021	Critically low aldosterone with suppressed renin. Massive urinary sodium wasting (203 mmol/L) confirms kidney is not retaining sodium. Alkaline urine

		(pH 7.5) pairs with later venous alkalosis (pH 7.44). SG 1.021, below patient's established baseline, indicates kidneys not yet in conservation mode; compare to SG 1.033 twelve days later at collapse. See Section 3
Day 36 Mar 9, 2018	Patient inhales small amount of THC (leaf). Gagging during ambulation. Collapse within minutes. Fully conscious throughout. Hands and feet vibrating. Unable to move. Son calls 911	THC-induced vasodilation as terminal trigger in a system at perfusion threshold. Peripheral vasospasm (vibrating extremities) indicates emergency centralization of blood volume to maintain cerebral perfusion
Day 36 Mar 9, 2018 (ER #2)	Emergency department visit #2. See Section 3 for complete laboratory results	Multiple subtle abnormalities identified retrospectively. BP remained low post-resuscitation
Day 41 Mar 14, 2018	Follow-up laboratory studies: Aldosterone 6.5 ng/dL	Aldosterone quadrupled from 1.5 to 6.5 in approximately two weeks. See Section 4
Day 43 Mar 16, 2018	Additional labs: pH 7.44 (venous blood), lactic acid 1.0 mmol/L. CMP abnormal. UA abnormal	Venous alkalosis. See Section 3
Day 62 Apr 4, 2018	EGD, surgical histopathological examination, CLO test performed	Upper GI evaluation negative for structural pathology
Days 76-78 Apr 18-19	Serial vitals: BP 100/60, 97/69, 90/54. Pulse 66-88. Resp 14-21	Persistent hypotension eleven weeks post-provocation. See Section 3
Day 88 Apr 30, 2018	BP 126/76	First normotensive reading. Compensation beginning
~Day 243 Oct 2, 2018	BP 139/90	Overshoot. System recalibrating. Refill phase initiated

3. Laboratory Findings

3.1 Patient-Initiated Laboratory Studies (February 27, 2018, Day 26)

Twenty-six days post-provocation, the patient independently ordered targeted laboratory studies through LabCorp based on his own research into the suspected pathophysiology. Rather than repeating the standard CMP/CBC panels that had already returned normal, the patient selected tests specific to the sodium-handling and volume-regulation pathway. The panel was limited to

four studies: renin activity, aldosterone, urinalysis, and urine sodium. [*Not bad for a Chemical Engineer with no medical training. What test would you have ordered?*]

Parameter	Value	Reference Range	Clinical Interpretation
Renin Activity, Plasma	0.564 ng/mL/hr	0.167-5.380	Low-normal. Insufficient to account for subsequent aldosterone volatility, indicating the renin-angiotensin system is not the primary driver of aldosterone production in this patient
Aldosterone	1.5 ng/dL	0.0-30.0	Near the floor. Quadrupled to 6.5 ng/dL within two weeks (see Section 4). This volatility at low renin implicates ACTH as the primary aldosterone driver
Urine Sodium	203 mmol/L	Not Established	Massively elevated. The kidney is dumping sodium at a rate consistent with absent or ineffective aldosterone-mediated reabsorption. This is the direct measurement of the failure mode: sodium is leaving the body because nothing is telling the kidney to retain it
Urine pH	7.5	5.0-7.5	At the ceiling of the reference range. Alkaline urine pairs with the venous pH of 7.44 measured two weeks later (3/16/2018), indicating systemic alkalosis expressed in both blood and urine compartments
Urine Specific Gravity	1.021	1.005-1.030	Within range but below the patient's established baseline. The kidneys are not yet in conservation mode. Compare to SG 1.033 twelve days later at the time of collapse, when the kidneys had switched to maximum concentration. The shift from 1.021 to 1.033 over twelve days documents the transition from passive sodium wasting to active volume preservation as the system approached crisis

Note on test selection: The patient's decision to order renin, aldosterone, urine sodium, and urinalysis rather than repeating standard panels reflects targeted hypothesis testing. These four tests, taken together, document the complete sodium-handling failure: aldosterone is not being produced (1.5 ng/dL), renin is not driving it (0.564), and sodium is leaving the body through the kidneys at a massive rate (203 mmol/L) as a direct consequence. Standard CMP would have shown sodium within normal serum range, missing the renal wasting entirely.

The urine sodium of 203 mmol/L is the single most significant finding in this panel. In a volume-depleted patient with persistent orthostatic symptoms and borderline-low aldosterone, the kidneys should be retaining sodium aggressively. Instead, they are excreting it at a rate that actively worsens the volume deficit. This finding confirms that the sodium retention mechanism

is fundamentally compromised, and it explains the progressive hemodynamic deterioration that culminated in collapse nine days later.

Notable negatives (UA): Protein negative, glucose negative, ketones negative, blood negative, WBC esterase negative, nitrite negative, bilirubin negative. No microscopic examination indicated. The urine is chemically clean aside from the alkaline pH and sodium wasting.

3.2 Emergency Department Visit #2 (March 9, 2018)

Studies performed: ECG, CMP, CBC, B-Type Natriuretic Peptide, CK, Troponin I, Chest XR, Urinalysis, CT abdomen without contrast.

Parameter	Value	Reference Range	Clinical Interpretation
BUN	27 mg/dL	7-20 mg/dL	Prerenal azotemia. Elevated BUN with normal creatinine indicates inadequate renal perfusion secondary to volume depletion
Potassium	3.5 mEq/L	3.5-5.0 mEq/L	Borderline low. At the floor of the reference range in a system where aldosterone-mediated potassium/sodium exchange is dysregulated
Specific Gravity (UA)	1.033	1.005-1.030	Above reference range. Kidneys maximally concentrating urine to preserve circulating volume
MCHC	31.8 g/dL	32.2-36.7 g/dL	Below range. Red blood cells carrying less hemoglobin per cell volume
Eosinophils	0	Present	Complete suppression. Consistent with acute stress response and immune downregulation
Basophils	0	Present	Complete suppression. Same pattern as eosinophils
pH (venous, 3/16)	7.44	7.32-7.42	High. Venous alkalosis indicates active compensatory buffering. Respiratory or metabolic alkalosis in a volume-depleted system
Lactic acid (3/16)	1.0 mmol/L	0.3-2.0 mmol/L	Normal. Rules out anaerobic metabolism despite perfusion compromise, suggesting adequate tissue oxygenation through compensatory mechanisms

Notable negatives: Troponin I, BNP, CK, ECG, CT head, CT abdomen, chest XR all within normal limits. No structural cardiac, pulmonary, or intracranial pathology identified.

3.3 Hemodynamic Data (April-October 2018)

Serial vital signs obtained during follow-up visits demonstrate persistent hypotension through six weeks post-provocation, followed by gradual compensation and eventual overshoot.

Date	BP (mmHg)	Pulse (bpm)	Resp (br/min)
4/18/2018	100/60	88	18
4/19/2018	97/69	70	21
4/19/2018	90/54	66	14
4/30/2018	126/76	74	--
10/2/2018	139/90	72	13

Key observations: Blood pressure of 90/54 represents near-shock territory. This reading was obtained after two units of IV normal saline, indicating that the patient is not volume-responsive in the expected manner. Fluids are not remaining in the vascular compartment. Despite hemodynamically significant hypotension, heart rate remained in the 60s-70s without compensatory tachycardia, and the patient never lost consciousness. Post-saline discharge BP was 95/XX. The system required approximately three months to achieve normotensive readings, followed by an apparent overshoot to 139/90 by October 2018.

4. Longitudinal Endocrine Data (2010-2026)

4.1 Cortisol (AM, serum)

Date	Value (mcg/dL)	Reference Range
Feb 4, 2010	13.2	2.9-19.4
Dec 10, 2012	10.3	2.9-19.4
Apr 13, 2022	11.5	AM 3.7-19.4
April 2026	13.2	AM 3.7-19.4

Cortisol output remains within a narrow band (10.3-13.2 mcg/dL) across 16 years of observation. This stability is itself significant when paired with the aldosterone trajectory below.

4.2 Aldosterone

Date	Value (ng/dL)	Context
Feb 27, 2018	1.5	24 days post-donation; pre-collapse. Renin 0.564 ng/mL/hr
Mar 14, 2018	6.5	5 days post-collapse. Quadrupled in ~2 weeks
April 2026	4.0	Current baseline

The aldosterone data reveal two distinct findings. First, extreme short-term volatility: aldosterone quadrupled from 1.5 to 6.5 ng/dL within approximately two weeks in February-March 2018. This volatility occurred with renin activity at 0.564 ng/mL/hr, which is too low to account for the aldosterone fluctuation through the standard renin-angiotensin-aldosterone pathway. This suggests an alternative driver, most likely ACTH, which can stimulate aldosterone production directly, bypassing the renin-angiotensin system.

Second, a long-term declining trend: the current value of 4.0 ng/dL in 2026 represents a reduction from the 6.5 peak, consistent with progressive loss of adrenal functional capacity over the intervening eight years.

4.3 ACTH (April 2026)

ACTH: 14 pg/mL (mid-range). This is the first available ACTH measurement. In isolation, it appears unremarkable. However, in a system with declining aldosterone and stable cortisol, a mid-range ACTH suggests the pituitary is maintaining sufficient drive to preserve cortisol output from diminishing adrenal tissue while aldosterone production, which depends primarily on the renin-angiotensin system rather than ACTH, follows the attrition curve of the glands themselves.

4.4 Selective Adrenal Preservation Pattern

The combined endocrine data describe a pattern of selective output preservation: cortisol remains stable across 16 years while aldosterone declines. This is consistent with a system that is losing adrenal tissue but preferentially maintaining cortisol production. Cortisol serves critical functions in immune modulation, glucose mobilization, and inflammatory environment management. Aldosterone manages renal sodium and potassium handling. If adrenal capacity is limited, cortisol preservation at the expense of aldosterone would represent a physiologically rational prioritization, with sodium balance increasingly dependent on behavioral compensation (e.g., salt craving).

5. Diagnostic Considerations

Standard diagnostic workup across two emergency department visits, multiple imaging studies, and endoscopy identified no structural pathology. The following conditions were effectively excluded by the documented evaluations:

Acute coronary syndrome: Serial troponins negative, ECGs normal, BNP normal

Cerebrovascular accident: CT head negative, neurological function intact

Acute abdomen: CT abdomen negative

Cardiac structural disease: Nuclear stress imaging (NM MPI Lexiscan, 3/20/2018) normal

Upper GI pathology: EGD with biopsy and CLO test (4/4/2018) negative

Addison's disease (primary adrenal insufficiency): Cortisol within normal range at all time points measured

The clinical presentation does not fit any single conventional diagnosis. The combination of volume-dependent hemodynamic instability, paradoxical saline non-responsiveness, maintained consciousness at hemodynamically significant hypotension, selective endocrine axis preservation, and aldosterone volatility independent of renin constitutes a pattern that falls outside standard diagnostic categories.

6. Discussion

6.1 The Provocation Sequence as Controlled Experiment

This case is notable for the clarity of its provocation-response structure. The input is known and timed (standard phlebotomy, February 1, 2018, 1:00 PM). The clinical response follows a reproducible five-phase arc: (1) compensatory hormonal surge (days 1-2, subjective mania); (2) vestibular perfusion failure (day 3, acute vertigo); (3) sustained autonomic instability (days 7-35, nausea and orthostatic symptoms); (4) terminal decompensation upon vasodilatory challenge (day 36, collapse); and (5) prolonged recovery with measurable hemodynamic endpoints (days 37-243+, serial BP from 90/54 to 139/90).

Each phase is accompanied by contemporaneous laboratory data. The approximately 72-hour delay between provocation and the onset of compensatory failure (mania on days 1-2, vertigo on day 3) suggests approximately three days of emergency reserve capacity in this patient's hemodynamic system. The compensatory surge (subjective mania) was not merely a brief spike but sustained functional perfusion for over 48 hours before the system's reserves were exhausted. This reserve window is itself a measurable parameter.

6.2 Volume Dysregulation and Measurement Limitations

A critical observation from this case is the inadequacy of concentration-based laboratory measurements in a volume-dysregulated system. Standard blood tests report serum concentrations (osmolarity), which assume a stable vascular volume. In a patient with significantly reduced circulating volume, a 'normal' serum concentration may represent a dramatically reduced total quantity of the measured analyte. This distinction between osmolarity (concentration per unit volume of solution) and osmolality (total solute per unit of total body water) is fundamental to interpreting this patient's laboratory data.

The persistent hypotension following two units of IV saline is direct evidence of this architecture. In a patient with intact vascular containment, 1-2 liters of isotonic saline would produce a measurable blood pressure response. In this patient, the fluid did not remain in the vascular compartment. This observation is consistent with either third-spacing, extravascular redistribution, or altered vascular permeability, and it undermines the validity of any serum-concentration-based measurement as a reflection of total body status.

6.3 Consciousness Preservation During Hemodynamic Crisis

Perhaps the most clinically striking feature of this case is the patient's maintenance of full consciousness during the collapse event and throughout the period of documented hypotension. A blood pressure of 90/54 mmHg, particularly in an already volume-depleted patient, would

typically be expected to produce altered mental status or syncope. The patient reports complete preservation of awareness, orientation, and cognition throughout.

The peripheral vasospasm reported during the collapse (hands and feet 'vibrating') is consistent with emergency redistribution of blood flow away from extremities to maintain central and cerebral perfusion. The absence of compensatory tachycardia (heart rate 66-88 bpm during documented hypotension) suggests this is not a standard sympathetic response to hypovolemia, but a different compensatory architecture.

6.4 Interpretive Framework

The findings in this case are interpreted within the framework described in Craddock (2026), "Candida albicans as a Biochemical Computer" (DOI: 10.5281/zenodo.19369716), and Craddock (2026), "The Saline Oscillation Hypothesis" (DOI: 10.5281/zenodo.19337526). Under this framework, the patient's hemodynamic architecture reflects long-term co-adaptation between host physiology and a systemic Candida albicans colonization that has progressively integrated into endocrine and vascular regulation.

Key framework-consistent observations include: (a) the selective preservation of cortisol over aldosterone, consistent with an organism that benefits from cortisol's immunosuppressive and glucose-mobilizing effects; (b) aldosterone volatility driven by ACTH rather than renin, consistent with pituitary-level management of adrenal output; (c) consciousness preservation during hemodynamic crisis, consistent with an enhanced perfusion management system; and (d) the patient's subjective craving shift from sugar to salt over the longitudinal course, consistent with behavioral compensation for declining aldosterone-mediated sodium retention.

This case does not constitute proof of the framework. It constitutes a documented sequence of clinical events and laboratory findings that the framework predicts and conventional diagnostic categories do not explain. The provocation-response structure, with its known input, timed outputs, and contemporaneous measurements, provides the closest approximation to experimental methodology available in a single-subject longitudinal study.

7. Summary of Findings

1. Routine phlebotomy in a volume-dysregulated patient produced a five-phase, 36-day decompensation arc with measurable clinical and laboratory endpoints at each stage.

2. The organism's emergency compensatory reserves last approximately 72 hours, after which perfusion failure symptoms emerge.

3. Standard diagnostic evaluation (cardiac, structural, neurological, gastrointestinal) returned entirely negative across two ER visits and multiple follow-up studies.

4. Subtle laboratory abnormalities (BUN elevation, borderline hypokalemia, concentrated urine, venous alkalosis, immune cell suppression) collectively describe a volume-depleted, perfusion-compromised system that no individual finding would identify.

5. Cortisol output is maintained at a stable setpoint across 16 years while aldosterone declines, indicating selective adrenal preservation.

6. Aldosterone is driven by ACTH-pulsed output rather than the renin-angiotensin system, producing extreme short-term volatility that renders any single measurement unreliable.

7. Urine sodium of 203 mmol/L on day 26, with aldosterone at 1.5 ng/dL, directly measures the sodium retention failure. The kidneys are actively excreting sodium in a volume-depleted system with no hormonal signal to retain it, confirming a fundamental compromise of the sodium conservation mechanism.

8. The patient maintained full consciousness at hemodynamically significant hypotension (90/54 post-saline), with peripheral vasospasm indicating active redistribution rather than cardiovascular collapse.

9. Hemodynamic recovery required approximately three months, followed by an overshoot phase consistent with volume restaging.

References

1. Craddock, J. (2026). "Candida albicans as a Biochemical Computer: An Engineering Analysis of Eukaryotic Signaling Architecture." Zenodo. DOI: 10.5281/zenodo.19369716

2. Craddock, J. (2026). "The Saline Oscillation Hypothesis." Zenodo. DOI: 10.5281/zenodo.19337526

3. Craddock, J. (2026) “Redacted Science” Book, www.redactedscience.org