The Research Landscape of Autonomic Dysreflexia (AD)

1. Executive Summary

As we advance through 2026, the scientific and clinical understanding of Autonomic Dysreflexia (AD) is undergoing a fundamental paradigm shift. Historically, AD has been managed as a reactive, acute hypertensive crisis—a "symptom" of spinal cord injury (SCI) to be suppressed pharmacologically or mechanically once it manifests. The current research landscape, however, reveals a transition toward **precision neuro-autonomic modulation** and **predictive monitoring**.

The "State of the Science" is no longer defined merely by the search for a better antihypertensive agent. Instead, it is characterized by the convergence of bioelectronic medicine, implantable biotechnology, and molecular phenotyping. The most significant breakthrough in the last 24 months has been the elucidation of the specific "neuronal architecture" underlying AD, a discovery that has enabled the development of Targeted Epidural Spinal Stimulation (TESS) protocols capable of stabilizing hemodynamics in real-time. This moves AD management from the era of blunt pharmacology into the era of closed-loop neuromodulation.

Simultaneously, the "invisible" nature of AD—particularly "silent" dysreflexia—is being illuminated by novel wireless bladder sensors (UroMonitor) and AI-driven wearable algorithms that detect autonomic events via skin nerve activity (SKNA) before systemic hypertension reaches crisis levels. Pharmacologically, the field is pivoting from non-specific anticholinergics to targeted beta-3 adrenoceptor agonists (mirabegron) and onabotulinumtoxinA, viewing them not just as bladder therapies, but as prophylactic cardiovascular protectants.

This report provides an exhaustive analysis of these developments, categorizing active clinical trials, emerging technologies, and mechanistic breakthroughs. It serves as a roadmap for navigating the transition from the current standard of care to a future defined by predictive monitoring and automated therapeutic loops.

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2. Clinical Context: The Baseline and The Gap

To appreciate the trajectory of current research, one must first establish the clinical baseline against which these innovations are measured.

2.1 The Current Standard of Care (Status Quo)

Autonomic Dysreflexia is conventionally defined as an acute, uninhibited sympathetic discharge in individuals with SCI at or above the T6 level, though recent evidence suggests susceptibility in lesions as low as T8 or T10.[1] It is clinically diagnosed by a rise in systolic blood pressure (SBP) >20 mmHg above the patient's baseline.[1] In the tetraplegic population, where baseline SBP may hover around 90-110 mmHg, a rise to 130-140 mmHg constitutes a hypertensive emergency potentially leading to intracranial hemorrhage, retinal detachment, or seizure.[1]

The current management algorithm, as detailed by the Consortium for Spinal Cord Medicine and Paralyzed Veterans of America (PVA) guidelines, remains largely mechanical and reactive [4]:

1. **Orthostatic Maneuver:** Immediately sit the patient upright to induce orthostatic pooling and lower intracranial pressure.[1]
2. **Stimulus Removal:** Loosen constrictive clothing and check for the most common triggers: bladder distension (blocked catheter) or bowel impaction.[10]
3. **Pharmacological Intervention:** If SBP remains >150 mmHg, rapid-acting, short-duration antihypertensives are administered. The primary agents in the US remain nitroglycerin paste (easier to wipe off if hypotension occurs) and immediate-release nifedipine (bite and swallow).[5] In the UK and Canada, nifedipine and captopril are more commonly utilized due to the limited availability of nitropaste.[5]

2.2 The Gap Analysis: Why Innovation is Urgent

Despite these guidelines, the "gap" between current care and optimal outcomes is widening, driven by four critical deficiencies that recent research aims to address:

- **Reactive vs. Preventative:** The standard of care is inherently reactive. Interventions are deployed *after* the hypertensive crisis has begun. This "wait for the headache" approach exposes the patient to repeated bouts of sheer stress on the cerebral vasculature, contributing to the high incidence of stroke and cardiovascular disease in the SCI population.[13]
- **The "Silent" Threat:** A significant proportion of AD events are asymptomatic ("silent AD"). Research utilizing 24-hour ambulatory blood pressure monitoring (ABPM) has revealed that patients often endure severe hypertensive surges during sleep or routine bladder management without perceiving symptoms.[3] Current clinical protocols, which rely on symptom reporting, completely miss this burden.
- **Iatrogenic Instability:** The pharmacological tools are blunt instruments. Nifedipine, while effective, causes systemic vasodilation. Once the noxious stimulus (e.g., a blocked catheter) is resolved, the persistence of the drug can lead to severe "rebound hypotension," a dangerous state for SCI patients who already suffer from impaired sympathetic vasoconstriction.[5]
- **Maladaptive Plasticity:** Current treatments address the *trigger* but ignore the *substrate*. They do nothing to mitigate the aberrant sprouting of C-fiber afferents and sympathetic

preganglionic neurons that heighten susceptibility over time. The neuroplasticity that makes the cord "hyperexcitable" remains untreated by nifedipine or positioning.[16]

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3. Deliverable 1: The Research Tables

Table A: Active & Recruiting Clinical Trials (2024-2026)

Trial Name / ID	Phase	Intervention	Primary Outcome	Status	Location / Lead
HEMO / HemON (NCT05044923, NCT05111093)	Phase 1/2	**Epidural Electrical Stimulation (EES)** via ARC-IM Therapy; Targeted Epidural Spinal Stimulation (TESS).	Restoration of hemodynamic stability; Reduction of orthostatic hypotension and AD severity.	Active / Completed (Results Pub 2025)	Calgary (A. Phillips) / Lausanne (G. Courtine) [18]
MACHINE (NCT04726059)	Phase 2	**Transcutaneous Spinal Cord Stimulation (tSCS)** + Activity Based Therapy.	Motor function; Autonomic functions (BP instability, bladder/bowel scores).	Active, Not Recruiting	UBC / ICORD (Canada); Krassioukov [21]
UroMonitor Trial (NCT04800523)	Feasibility	**UroMonitor Device** (Wireless catheter-free bladder pressure sensor).	Safety, feasibility, and tolerability of insertion/removal; Detection	Active / Completed	UBC / Cleveland Clinic (Krassiouko v/Damaser) [22]

			of AD events.		
Mirabegron vs. Oxybutynin (NCT03187795)	Phase 4	**Mirabegron** (Beta-3 agonist) vs. Oxybutynin.	Effectiveness/safety for NDO; Impact on AD severity/BP variability.	Active, Not Recruiting	USA / Canada [24]
Prazosin Prophylaxis (NCT00175682)	Phase 2	**Prazosin** (Alpha-blocker) vs. Placebo.	Reduction of AD signs/symptoms during sperm retrieval (vibrostimulation).	Active, Not Recruiting	Vancouver Coastal Health [25]
Deciphering Autonomic Function (NCT07012135)	Observational	**Diagnostic Testing** (Cold pressor, Valsalva) & Wearables.	Characterization of autonomic dysfunction gradients; Correlation with secondary complications.	Active, Not Recruiting	Mayo Clinic (USA) [26]
RECOVER-AUTONOMIC (NCT06305780)	Phase 2	**IVIG / Ivabradine**.	Autonomic dysfunction symptoms (POTS/Dysautonomia) in Long COVID (Mechanism relevance	Recruiting	USA (Multi-center) [27]

			to SCI).		
DPAF-MS (NCT07012135)	Observational	**Autonomic Testing** (Valsalva, Cold Pressor).	Characterize gradients of autonomic dysfunction (AD/OH) in MS vs. SCI.	Active, Not Recruiting	Mayo Clinic [26]
Botox for NDO/AD (NCT02298660)	Phase 4	**Onabotulinumtoxin A** (Intradetrusor).	Reduction of AD severity (SBP rise) during urodynamics.	Active, Not Recruiting	Rick Hansen Institute [29]

Table B: Emerging Technology & Wearables

Technology	Type	Purpose	Development Stage	Source
UroMonitor	Implantable Sensor	**Prevention/Monitoring:** Wireless, catheter-free monitoring of bladder pressure to detect AD triggers (NDO) in real-time without inducing AD via catheterization.	Clinical Validation / Pivotal Trials	[22]

Purdue AD Monitor	Wearable (Sensors + Controller)	**Detection:** AI-driven analysis of Skin Nerve Activity (SKNA), ECG, and GSR to detect AD onset with high accuracy (>93%).	Prototype / Validation (Start-up: Neuro Vigor)	[31]
Swiss Multimodal Watch	Wearable (Smartwatch)	**Detection:** Uses PPG, Heart Rate, and EDA (Electrodermal Activity) to classify AD events vs. exercise.	Clinical Research (Swiss Paraplegic Centre)	[34]
Closed-Loop EES	Implanted Neuromodulation	**Treatment:** "Smart" spinal stimulator that detects hemodynamic instability and auto-adjusts stimulation to stabilize BP (Hemodynamic Hotspot targeting).	Clinical Trials (HEMO/HemON)	[37]
ANSiscope	Clinical Monitor	**Monitoring:** Non-invasive assessment of Autonomic Nervous System (ANS)	Commercially Available / Research Use	[40]

		dysfunction and sympathovagal balance via HRV.		
VitalScan ANS+	Diagnostic System	**Assessment:** Comprehensive autonomic testing (Valsalva, Tilt, HRV) to stratify risk.	Commercially Available	[42]

Table C: Key Papers (Last 5 Years)

Title	Author / Group	Year	Key Finding	Source
A neuronal architecture underlying autonomic dysreflexia	**Soriano, Phillips, Courtine et al.** (Nature)	2025	Identified specific spinal interneurons and circuits responsible for AD, enabling targeted neuromodulation.	[37]
An implantable system to restore hemodynamic stability after spinal cord injury	**Phillips, Courtine et al.** (Nature Medicine)	2025	Demonstrated that closed-loop EES can effectively manage both orthostatic hypotension and autonomic dysreflexia.	[37]

Transcutaneous spinal cord stimulation...	**Flett / Krassioukov**	2023	Highlighted potential risks: tSCS may exacerbate sympathoexcitation in some cases, contrasting with EES specificity.	[43]
Reduced Reflex Autonomic Responses following Intradetrusor OnabotulinumtoxinA	**Fougere / Krassioukov**	2021	Confirmed Botox significantly ameliorates AD severity during urodynamics, validating it as a dual-purpose therapy.	[45]
Automatic Detection... of Autonomic Dysreflexia Using Multi-Modal Non-Invasive Sensing	**Suresh / Duerstock**	2022	Validated that Skin Nerve Activity (SKNA) combined with ML can accurately detect AD non-invasively.	[34]
Vagus Nerve Stimulation Reduces Neuroinflammation...	**Frontiers Neuroscience**	2022	VNS promotes microglial M2 polarization and reduces neuroinflammation, potentially mitigating AD plasticity.	[48]

4. The "State of the Science" Report: Deep Dive

4.1 The "Hot" Topics: Where is the Funding Going?

The research landscape in 2025-2026 is dominated by a shift from pharmaceutical management to **bioelectronic control**. While pharmacological trials are refining the use of existing agents (repurposing mirabegron and botox), the "heavy lifting" in terms of funding and high-impact publications is occurring in **neuromodulation**.

Specifically, the field is focused on "closing the loop"—creating systems that sense autonomic instability and automatically adjust stimulation to correct it. This is driven by major grants from bodies like the US Department of Defense (CDMRP), NIH (RECOVER initiative), and substantial philanthropic support (Wings for Life, Spinal Research UK).[31]

Two distinct "camps" of innovation have emerged:

1. **The "Fix the Circuit" Camp (Neuromodulation):** Using EES to physically override the aberrant sympathetic signaling.
2. **The "Early Warning" Camp (Wearables/Tech):** Using AI and novel biomarkers (SKNA) to detect the onset of AD before clinical symptoms manifest, allowing for behavioral intervention.

4.2 Detailed Trial Analysis: Neuromodulation

The most consequential development in AD research is the application of Epidural Electrical Stimulation (EES) for hemodynamic control.

A. Epidural Electrical Stimulation (EES): The HEMO & HemON Trials

Led by the collaborative "RESTORE Network" (Dr. Aaron Phillips at UCalgary and Dr. Gregoire Courtine at EPFL), these trials [18] have fundamentally altered the physiological understanding of spinal autonomic control.

- **The Innovation:** Unlike previous EES applications that targeted motor pools to facilitate walking, the HEMO trials target **"hemodynamic hotspots"** in the lumbosacral spinal cord. These are anatomically distinct dorsal root entry zones enriched with neurons that regulate the splanchnic vascular bed—the body's primary reservoir for blood pressure control.
- **Mechanism of Action:** Research published in *Nature* (2025) [37] demonstrated that stimulation of these hotspots does not merely "squeeze" blood vessels. Instead, it recruits specific proprioceptive afferents that synapse onto the sympathetic preganglionic neurons (SPNs). By modulating the excitability of these SPNs, the stimulation can be "tuned." High-frequency stimulation can drive vasoconstriction to treat orthostatic hypotension, while specific parameters can **dampen** the hyper-reflexive response to noxious stimuli, effectively "clamping" the blood pressure and preventing

AD.[39]

- **Trial Outcomes:** Participants implanted with these systems showed immediate stabilization of hemodynamics. Long-term use was associated with a reduction in the severity of AD episodes, suggesting that chronic stimulation may induce beneficial plastic changes in the spinal circuitry.[18]

B. Transcutaneous Stimulation (tSCS): A Cautionary Tale

While invasive EES is precise, non-invasive tSCS is being explored as a more accessible alternative (e.g., the MACHINE trial [21]). However, recent data has introduced significant complexity.

- **The Conflict:** While some studies suggest tSCS can improve orthostatic tolerance, rigorous physiological assessments [43] have raised safety signals. In well-controlled crossover studies, tSCS was found to **exacerbate** sympathoexcitation in some participants.
- **The "Non-Specific" Problem:** Unlike EES, which targets specific roots, tSCS applies a broad electrical field. This may indiscriminately activate the aberrant sympathetic sprouts that drive AD. In one study, tSCS normalized Valsalva responses (good) but induced dysreflexic responses to cold pressor tests in patients who were previously stable (bad).[43] This suggests that tSCS may lower the threshold for AD in susceptible individuals, necessitating careful patient selection and monitoring.

C. Vagus Nerve Stimulation (VNS)

VNS is emerging as a "disease-modifying" strategy rather than an acute treatment.

- **The Inflammatory Reflex:** Research [48] posits that chronic neuroinflammation (elevated TNF-alpha, IL-6) drives the maladaptive synaptic sprouting that causes AD. VNS activates the cholinergic anti-inflammatory pathway, suppressing this systemic inflammation.
- **Safety Profile:** Preclinical and early clinical data [52] confirm that VNS is safe in high-level SCI and does not trigger AD events—a critical safety hurdle. The current hypothesis is that chronic VNS may reduce the *susceptibility* to AD over months by dampening the inflammatory drive for synaptic reorganization.

4.3 Detailed Trial Analysis: Pharmacological Refinement

The search for a "magic bullet" pill has largely been replaced by the strategic refinement of urological drugs to serve cardiovascular ends.

A. Mirabegron (Beta-3 Agonist)

The shift from anticholinergics (oxybutynin) to mirabegron is gaining robust evidentiary support.[24]

- **Mechanism:** Mirabegron relaxes the detrusor muscle via beta-3 adrenergic receptors

during the storage phase. Unlike anticholinergics, it does not block the parasympathetic voiding contraction as aggressively, nor does it carry the cognitive/dry mouth burden.
- **AD Prevention:** Crucially, studies [56] show that mirabegron significantly lowers **detrusor pressure at end-filling**. Since high intravesical pressure is the primary afferent trigger for AD, mirabegron acts as a specific prophylactic agent. Patients switched to mirabegron demonstrated reduced AD frequency and increased bladder capacity without the rebound hypotension associated with systemic alpha-blockers.

B. OnabotulinumtoxinA (Botox)

Botox is now viewed as a "dual-purpose" therapy.

- **Validation:** Trials [46] have quantified that intradetrusor Botox does more than prevent incontinence; it significantly blunts the hypertensive surge during urodynamics. By chemically denervating the afferent arm of the reflex loop, Botox effectively "silences" the bladder as a trigger source for 6-9 months.

C. Prazosin for Prophylaxis

For predictable triggers (sexual activity, sperm retrieval, bowel programs), Prazosin is being validated as a superior prophylactic to nifedipine.[15]

- **Advantage:** As a selective alpha-1 blocker, Prazosin blunts the sympathetic surge without obliterating resting blood pressure. This avoids the "crash" often seen when nifedipine is taken proactively.

4.4 Mechanistic Updates: The "Neuronal Architecture"

The most profound scientific advancement has been the detailed mapping of the neural circuits that drive AD.

- **The Circuit Revealed:** A landmark 2025 *Nature* paper [37] identified a specific population of **propriospinal neurons** (V2a interneurons) that connect lumbosacral sensory inputs to thoracic sympathetic preganglionic neurons.
- **Aberrant Sprouting:** Following SCI, these neurons undergo massive, NGF-driven sprouting, creating a "short circuit" that bypasses supraspinal control. This sprouting is not random; it follows a specific molecular architecture.
- **Implication:** This discovery validates the "hemodynamic hotspot" theory for EES. It also suggests that future therapies could use viral vectors or molecular inhibitors to selectively target these specific interneurons, potentially "pruning" the dysreflexic circuit at the molecular level.

4.5 Technology & Monitoring Updates

The inability to monitor AD in the home environment has historically hindered research. New tech is solving this "data gap."

A. The UroMonitor (Wireless Urodynamics)

Developed at UBC/Cleveland Clinic, the UroMonitor [22] is a catheter-free, wireless intravesical pressure sensor.

- **The Problem it Solves:** Traditional urodynamics require a catheter, which is itself a noxious stimulus that can artificially trigger AD.
- **The Innovation:** Inserted into the bladder, the UroMonitor transmits pressure data wirelessly for days. This allows researchers to correlate "real world" AD events (e.g., hitting a bump in a wheelchair, a leg spasm) with bladder pressure spikes, proving that many "spontaneous" AD events are actually bladder-driven.

B. AI & Skin Nerve Activity (SKNA)

Purdue University researchers [31] have developed a wearable that measures **Skin Nerve Activity (SKNA)**—a high-frequency component of the ECG signal that serves as a direct proxy for sympathetic tone.

- **AI Detection:** Machine learning algorithms can analyze SKNA, heart rate, and electrodermal activity to detect the onset of AD with >93% accuracy.[34]
- **Clinical Utility:** This acts as a "Check Engine Light" for the autonomic nervous system, alerting the patient to a rising sympathetic surge *before* the pounding headache begins, allowing for early intervention.

4.6 Leading Research Hubs

- **University of Calgary (Canada) & EPFL (Switzerland):** The epicenter of hemodynamic neuromodulation. PIs **Aaron Phillips** and **Gregoire Courtine** are leading the HEMO/HemON trials and publishing foundational mechanistic papers.[37]
- **ICORD (Vancouver, Canada):** PI **Andrei Krassioukov** remains the global authority on clinical management, guidelines, and non-invasive cardiovascular assessment.[44]
- **Purdue University (USA):** PI **Bradley Duerstock** is leading the engineering front, specifically AI detection and SKNA wearables.[31]
- **Stoke Mandeville Spinal Research (UK):** A critical funding hub for pragmatic, QoL-focused research (UTIs, pain, upper limb) that translates directly to the UK patient population.[62]
- **Swiss Paraplegic Centre (Nottwil):** Leading large-scale biomarker and autonomic phenotyping studies.[26]

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5. The "Gap" Remains: Unanswered Questions

Despite this progress, critical gaps persist:

1. **The tSCS Safety Paradox:** The conflicting data on whether transcutaneous stimulation stabilizes or exacerbates AD is a major safety hurdle.[43] Large, rigorous trials are needed

to define "safe" stimulation parameters before tSCS can be recommended for home use.
2. **Pediatric AD:** The research is overwhelmingly focused on adults. The developing spinal cord may respond differently to neuromodulation, yet pediatric-specific trials are virtually non-existent.
3. **Biomarker Validation:** While we have candidates (NF-L, GFAP, Cytokines) [67], we lack a validated clinical blood test that can stratify a patient's risk. We cannot yet tell a patient, "Your blood work suggests you are at high risk for severe AD this year."
4. **Availability of Therapeutics:** While UroMonitor and EES are promising, they are not yet commercially available standard-of-care devices. The regulatory pathway for "autonomic prosthetics" is complex and nascent.

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6. Researcher/Clinician "Watch List" (12-24 Months)

- **Late 2025 / Early 2026:** Watch for the publication of **long-term home-use data** from the **HEMO trial**.[20] Key question: Does the autonomic nervous system "habituate" to EES over time?
- **Commercial Launch:** Monitor the FDA/CE status of the **UroMonitor**.[22] Its approval would revolutionize the assessment of neurogenic bladder and AD risk.
- **Guideline Updates:** Anticipate potential updates to **PVA/NICE guidelines** [4] incorporating **Mirabegron** as a preferred first-line agent over anticholinergics due to its AD-protective profile.
- **Neuro Vigor / Purdue:** Keep an eye on the commercialization of the **SKNA wearable**.[31] If this reaches the market, it will be the first consumer device specifically for AD monitoring.
- **Nature/Nature Medicine Papers:** The 2025 papers by **Phillips and Courtine** [37] are the new "textbooks" for autonomic neuroanatomy. Clinicians should familiarize themselves with the concept of "hemodynamic hotspots."

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7. Conclusion

The research landscape of Autonomic Dysreflexia is transforming from a static field of symptom management to a dynamic arena of **preventative bioengineering**. The identification of the neural circuits driving AD has unlocked the potential for targeted neuromodulation, while advances in sensor technology are making the invisible visible.

For the clinician, this means the future of care will likely involve not just prescribing nifedipine, but programming a spinal stimulator to "clamp" blood pressure and reviewing data from a bladder sensor to preempt triggers. For the researcher, the frontier lies in refining these technologies, validating safety, and ensuring these sophisticated interventions are accessible to the global SCI population. The gap between "bench" and "bedside" is closing, promising a future where AD is no longer a life-threatening emergency, but a manageable, monitored

physiological state.

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