Brief History and Current State of Autonomic Dysreflexia

Isaac Hernandez Jimenez, MD
November 19th, 2015
Goals and Objectives

• **Review** the history of autonomic dysreflexia (AD)
• **Define** autonomic dysreflexia (AD)
• **Understand** pathophysiology of AD
• **Identify**
  • characteristics of AD
  • current treatment strategies used to prevent AD
  • current strategies used to manage AD
• **Understand** challenges in the identification, prevention and management of AD
BRIEF HISTORY
Brief History

• Head and **Riddoch** first described autonomic dysreflexia in 1917

• Significance and dangers described by:
  • Whitteridge and **Guttman** 1946-1947
  • Thompson and Whitham 1948
  • Pollock et al. 1951
  • Bors and French 1952
  • Schreibert 1955
  • Ariefff et al. 1962
Dear Dr. Herrald,

I must apologise for the delay in submitting this report, but I have had some difficulty in getting my photographs ready in time.

I am enclosing the receipts for the apparatus purchased for the skin temperature test.

Brigadier Riddoch, who was here at the end of last month, has already seen the details of my research, and he has asked me to publish them. I shall certainly do so, with pleasure, when the study is completed. He also asked me to inform you that, at the moment, I am concerned with a research on healing of pressure sores in spinal injuries.

I take this opportunity to express my thanks to you for your kind interest in my work.

Yours sincerely,

Lynx

P.S. I should be glad if you would kindly return the photos when you have finished them.

Dr. F.J.C. Herrald, Secretary,
Medical Research Council, c/o London School of Hygiene, Keppel Street, London, W.C.1.
Brief History

Whitteridge and Guttman in 1947

- Work still considered seminal nowadays
- Recognized AD as being life-threatening
- Part of series of studies on sweating mechanisms and their control by the spinal cord
MEDICAL RESEARCH COUNCIL

FORM OF APPLICATION FOR A RESEARCH GRANT

N.B.—Sections A and B should be completed in every case, except that questions marked with an asterisk in Section A need not be answered by applicants already well known to the Council. One or more of Sections C, D, and E should be completed according to the nature of the grant sought.

A.—PERSONAL PARTICULARS

Full name .... Guttmann, Ludwig

Postal address .... 63, Londesdale Road, Oxford.

*Date of birth .... 3rd July, 1899.

*Degree, etc. (with dates and sources) .... M.D. Freiburg, 1924.

*Present position .... Research Assistant at the Hufriedt Department of Surgery, University, Oxford.

1926-1929 Assistant, Psych. Clinic, Univ., Hamburg.
1933-1939 Director, Neurology & Neurosurgery, Dept., of the Jewish Hospital. Since 1939 (April 1st) present position.

*Principal scientific publications .... Correlations between Sweating & Nervous System.
(10 papers published between 1920 - 1941).

Complete list available if desired.

*If desired, name of referee ....

B.—PARTICULARS OF THE INTENDED RESEARCH

Brief definition of the intended research

A general and comprehensive study of the physiology and pathology of the sweat glands with special reference to spinal cord and spinal root lesions.

Hand and sensibility

Centres or centres where arrangements have been made for the proposed work .... Spinal Cord Centre, Stoke Mandeville.

Name of director, if any, who has agreed to supervise the work ....

Probable duration of the proposed work .... One year and three months.

C.—PARTICULARS REQUIRED FROM APPLICANTS FOR PERSONAL GRANTS

Amount sought .... 500 rs.

Whether for whole-time work, or, if not, for what proportion of time ....

Appointments, if any, to be held concurrently with the grant. (Give particulars of salaries and conditions)

Whether applicant engages in private practice .... No.

D.—PARTICULARS REQUIRED FROM APPLICANTS FOR EXPENSES’ GRANTS

Amount sought ....

Indication of the nature of the expenses. (e.g. Materials, laboratory or clerical assistance, etc.)
Brief History

Guttman

- Cystometrogram 3/13/1944
- T4 complete SCI
- Bladder distension (no description of BP/HR)
- Initially did not recognize BP was being affected
- Study’s initial objectives were looking at effects of bladder distension on sweating, vasomotor function and skin temperature.
- He fortuitously eventually discovered the elevated BP
- This led to the beginnings of our understanding of AD
DATE: 18-23.99

Pain in abdomen
Pain increasing, spreading to chest, throat, neck

Heating, feeling nauseated

Inward wound may have entered upper abdomen

Distant of sound over anterior suprapubic area (A.S.)

Pain increasing. Feeling of burning

Knee jerks low tone

Loss of strength in upper limbs

Note: X-ray through suprapubic opening
December 1946

- Guttman described multiple pts with “complete upper cord lesions”
- Upon distending their bladders:
  - Patchy flushing of the face and neck
  - Profuse localized sweating over head and shoulders or at level of lesion
  - Blockage of nasal air passage and nasal voice
  - Slow pulse with occasional extrasystoles
  - Respiratory discomfort
  - Fullness of the head -> severe occipital or frontal headaches
- SBP: 190 – 260 mmHg   DBP: 125-135 mmHG
Brief History

Guttman

- Recognized sweating was just a manifestation of autonomic function
- Contraction of the bladder causing autonomic disturbance
- Further work done on blood flow control (between 1945 and 1947)
- 1946: realized rise in BP was the key element to AD
- 1947: published paper along with Whitteridge
INTRODUCTION
Introduction

Cardiovascular (CV) disease after SCI can be significantly **accelerated**

- Autonomic disturbances
- Cardiovascular dysfunction
- Lifestyle changes/issues
Introduction

The risk of heart disease is almost 3 times higher

The risk for stroke is almost 4 times higher
Introduction

• SCI leads to **neurological injury**
  • Including autonomic nervous system
    • Primarily sympathetic dysfunction

• **Unstable blood pressure**
  • Low resting blood pressure
  • Orthostatic hypotension
  • Autonomic dysreflexia
Introduction

CV dysfunction often more important than motor function recovery

Initially pt not aware of presence/significance
Introduction

• In acute and chronic SCI, *one of the most common causes of death*

• **Extremely** important to know and understand impact of autonomic/CV dysfunction

• International Standards for Autonomic Dysfunction *(ISAD)*
DEFINITION
Definition

- Acute elevation of SBP of at least 20 mmHg
- +/- bradycardia
- Triggered by nonnoxious or noxious visceral or somatic stimulation below the Level Of Injury (LOI)
PATHOPHYSIOLOGY
Pathophysiology

- Large sympathetic discharge triggered below LOI

- Norepinephrine and dopamine release ↑ regional vasoconstriction ↑ systemic hypertension (HTN)

- **Intact spinal cord**: mediated by descending inhibitory input from supraspinal centers

- **Injured spinal cord**: loss of inhibitory control
Pathophysiology

- Baroreceptors in blood vessels detect hypertensive crisis — signal brain
- Hypertension
- Widespread vasoconstriction
- Massive sympathetic response
- Level of spinal cord injury = T6 or above
- Descending inhibitory signals blocked at spinal cord injury
- Heart rate slowed
- Afferent stimulus
- Full bladder or stimulus from bowel
Pathophysiology

- Elevated blood pressure (BP) sensed by carotid sinus and aortic body
- Compensatory mechanisms initiated (in an attempt to correct systemic hypertension)
- Sinoatrial node of heart activated by CN X (bradycardia) = ineffective
- Increase in parasympathetic activity above LOI ↑ vasodilation
Pathophysiology
CHARACTERISTICS
Characteristics

• Occurs when $\text{LOI} \geq T6$

• Most common causes
  • Bladder/Bowel/others

• Usually brief

• Can last days/weeks/indefinitely
Characteristics

• Can occur in acute and chronic SCI

• SBP as high as 300 mm Hg

• As many as 40 times per day (11 times/day on avg)
Characteristics

Signs and symptoms (Can be asymptomatic)

- Severe headache
- Anxiety
- Profuse sweating above LOI
- Flushing and piloerection above LOI
- Dry and pale skin below LOI
- Blurred vision
- Nasal congestion
- Bradycardia, arrhythmias, atrial fibrillation
Characteristics

Diagnosis

• Peaks/valleys
• Can be silent

Usually present **2-3 months after injury**

• Can happen sooner
Characteristics

Misconceptions

• Hypotension
• Cannot cause death
• It is not important to check BP
• SBP 130s-140s are “ok”
Characteristics

Those @ higher risk

The higher the injury level

The more severe the injury
  • Complete tetraplegia: ~ 91%
  • Incomplete tetraplegia: ~ 27%

Chronic more than acute SCI
  • As early as 4 days post injury
TREATMENT
Treatment

Nonpharmacologic management

- Protocol
- Proceed with meds if SBP > 150 mm Hg in an adult

Pharmacologic management
NONPHARMACOLOGIC
Protocol

In the care of the px with Autonomic Dysreflexia (AD) the nurse will:

1. Identify AD as medical emergency in px with SCI at T6 level or above who are no longer in spinal shock.
2. Assess the pt for one or more signs/symptoms of AD. These most commonly include:
   - Headache which may become severe and pounding
   - A sudden increase in BP of 20 mm HG above baseline. Note: normal BP for persons with quadriplegia may be as low as 90-110. It may also be accompanied by bradycardia.
   - Diaphoresis or flushing above the level of injury
   - Piloerection (goosebumps) above the level of injury
   - Bradycardia
   - Chills without fever
   - Changes in vision – blurred or tunnel
   - Anxiety/apprehension
Protocol

3. If BP is elevated, sit patient upright, position legs in a dependent position, remove binder, TEDs, loosen clothes, etc. to decrease hypertension. Then proceed to find the cause.
   • Monitor and document BP and pulse every 2-3 minutes until parameter have returned to baseline. Continue to observe and document every 15 minutes for a minimum of 1 hr. and then every 30 minutes for 2-3 hours.

4. Notify physician of episode

5. Assess for noxious stimuli cause of AD (begin with urinary system).
   • **Bladder Distention (90% of AD related to urinary system).**
     • Catheterize the patient using lidocaine gel as a lubricant and inserting lidocaine gel into the urethra prior to catheter, if an indwelling urinary catheter is not in place.
Protocol

• If patient has an indwelling catheter, check for urinary flow, overfilled bag, or kinks in tubing. If patient has an indwelling catheter and urine is not flowing, gently irrigate with sterile normal saline (begin with 10 mL, do not exceed 30 mL total)

• A bladder scan may be done to help support the etiology if unsure.

• **If indwelling catheter remains clogged after the irrigation, remove the catheter and replace.** (Use lidocaine gel when inserting new catheter)

• **If symptoms persist, assess bowel status**
Protocol

Bowel Status (8% of AD is related to bowels):

• Palpate patient’s abdomen for distension

• Insert **lidocaine gel** into the rectum. Wait 2-3 minutes then gently insert lubricated gloved finger. Evaluate for impaction. If impaction is found, discuss with physician and gently remove impaction if ordered.

• If symptoms persist, assess for other noxious stimuli
Protocol

Other Noxious Stimuli:

- Pressure ulcer or treatment for them
- Other skin irritants (wrinkled sheets, ingrown toenail, sitting on bump such as wallet, or other item in the rear pocket)
- VTE
- Menstrual cramps, labor, other reproductive issues
- Constrictive clothing
- Pain
- Surgical and diagnostic procedures
- Fractures
PHARMACOLOGIC MANAGEMENT
Pharmacologic management

Severity varies

- If no resolution and SBP remains at or higher than 150 mm Hg in an adult, proceed with medication

Use antihypertensive agents

- Rapid onset
- Short acting
Pharmacologic management

• There is *no gold standard medication*

• Selection is provider-dependent

• Most recommendations based on non-SCI patients with hypertensive crises
Pharmacologic management

Examples

• Nifedipine
• Nitrates
• Captopril
• Terazosin/Prazosin
• Beta blockers
• Hydralazine
• Prostaglandin E2
• Sildenafil
Pharmacologic management

Nifedipine

• Ca channel blocker
• Decreased peripheral vascular resistance and modest fall in sbp and dbp (5-10 mmHg sbp)
• No issues with its use for AD
• However, issues with non-SCI population
  • Heart attack, stroke, death
• Therefore, moving away from this agent
Pharmacologic management

Nitrates

- Vasodilator effect on peripheral arteries and veins
- Do not use if recent use of sildenafil (usually within 24 hrs)
- If so, recommend using nonnitrate agent
- Expert opinion
Pharmacologic management

Prazosin/Terazosin

- Alpha 1 adrenoreceptor blocker
- Can be used in cases were cause of AD not identified
- Used in studies for up to 3 months or 2 weeks respectively
- Prazosin should not affect resting blood pressure
Protocol

7. Administer 1 inch of Nitroglycerin 2% transdermal ointment if cause cannot be found and the elevated pressure is at or above 150 mm Hg systolic. Remember that BP will fall quickly after noxious stimuli is identified and removed, so patient might experience rebound hypotension when pharmaceutical are used. **Nitroglycerin 2%** ointment is the drug of choice as it can be removed when noxious stimuli is removed.
   - If allergies to Nitroglycerin 2% ointment or if patient has used nitrate based medications and/or PDE-5 inhibitor (e.g. Sildenafil (Viagra)) in the last 48 hours, first contact physician for consideration of and order for a different agent such as **Hydralazine** 10mg PO x1 dose.

8. Once AD is removed, complete a neurologic exam and notify physician of AD protocol implementation and exam findings.

9. **Educate patient and family** regarding AD and document.
PREVENTION
Prevention under particular circumstances

• Bladder procedures
• Anorectal procedures
• Pregnancy and labor
• General surgery
• FES exercise
AD prevention during **bladder procedures**

- Bladder irritation and/or distention is #1 cause for AD

- Bladder program

- Urologic surveillance
  - UDS, cystoscopy, botulinum toxin, anticholinergics, bladder/sphincter surgery, etc
  - Procedures themselves can trigger AD
AD prevention during anorectal procedures

Bowels are 2\textsuperscript{nd} most common cause of AD

- Constipation
- Hemorrhoids
- Anal fissures
- Digital stimulation
- Rectosigmoid distention
- Anal manipulation
- Anoscopy
- Flexible endoscopy
AD prevention during anorectal procedures

One study showed decreased AD with intersphincteric anal block with lidocaine

One study showed topical lidocaine not preventing AD during anorectal procedures
AD during Pregnancy and Labor

- Women = 1/3 of SCI population

- SCI does not affect ability to become pregnant
AD during Pregnancy and Labor

- High risk of developing uncontrolled AD during Labor & Delivery (L&D)
- Early recognition is essential
- Usually timed with uterine contractions
- Usually > T10, contractions only present as abdominal discomfort
- Anesthesia is recommended during L&D regardless of whether sensation is present
AD during Pregnancy and Labor

• Epidural anesthesia is best option for control of AD
  • Only 4 studies (n=54)
  • Observational evidence

• Recommended both in:
  • Vaginal delivery
  • Cesarean section
AD during general surgery

AD can be triggered by somatic and visceral noxious or nonnoxious stimuli below LOI

Measures

- Peripheral anesthetic block
- Epidural anesthesia
- General anesthesia
AD during general surgery

• Even with partial or complete lack of sensation, AD can be triggered.
• Anesthesia and General Surgery need to be aware.
• Up to 90% of pts undergoing surgery with topical or no anesthesia, will experience AD.
• Spinal or general anesthesia should be enough to prevent AD.
COMPLICATIONS
Complications

- Intracranial hemorrhage
- Retinal detachment
- Seizures
- Myocardial infarct
- Cardiac arrhythmias
- Death
Death from AD

Few deaths reported
*(does not mean they do not happen)*

Literature review shows that SBP as low as 180 mm Hg has caused death
Life-threatening outcomes associated with autonomic dysreflexia: A clinical review.

N=32 cases of death or life-threatening complications of AD

- Twenty-three (72%) cases were CNS-related
- Seven (22%) cases were CV-related
- Two (6%) cases were pulmonary-related

Seven (22%) deaths were noted as a direct result of complications following an AD attack.
PREVENTION
Prevention

Education

- Patient
- Family/Caregiver
- Healthcare provider
- Medication

Top causes

- Bladder
- Bowel
What we do in inpatient rehabilitation

**Education**

- Signs and symptoms
- Prevention
- Treatment
  - Meds
What we do in clinic

Education

• Physician
• SCI Nurse Coordinator

• New vs. old
• Verbal/Written/Online information
• Blood pressure “homework”
• Meds prn
Challenges (many)

• ER
• EMS
• Rehabilitation personnel
• Non-rehabilitation personnel
RESEARCH
Research

AD knowledge

- Rehab personnel
- Non-rehab personnel
- ER
- EMS

The ABCs of AD
AD knowledge

• **Krassioukov, et al:** *Autonomic dysreflexia: translating knowledge into practice for Canadian emergency room personnel.*

• n=133 (paramedics, ER MDs/RNs) in 3 canadian provinces
AD knowledge

• >80% rated AD knowledge as “poor” or “fair”
• 25% failed to define AD correctly
• Only 50% knew 3 or more associated S&S
The ABCs of AD

• Tomasone, et al 2014
• Free online educational module (wp-dev.jibc.ca/abcofad/)
• Target: prehospital emergency care workers (paramedics, ER RNs and MDs)
  • Implementation of newly acquired knowledge did not improve
The ABCs of AD

• Similar projects to determine the level of AD knowledge in the US could be pursued
• First step is increasing knowledge
• Next step is changing providers’ practice
STANDARDS FOR AUTONOMIC ASSESSMENT
Standards for Autonomic Assessment

International Standards for the Neurological Classification of Spinal cord Injury (ISNCSCI)

- Motor Function
- Sensory Function
Standards for Autonomic Assessment

ISNSCI successful

However, autonomic assessment is lacking
Autonomic Dysfunction

Limited information on relationship between:
- Level and completeness of the SCI lesion
- Degree of autonomic dysfunction

International standards to document remaining autonomic function after spinal cord injury
Autonomic Dysfunction

After SCI, changes in the Autonomic Nervous System (ANS) are difficult to document with bedside exam:

- General autonomic function
- Bladder
- Bowel
- Sexual function
Goals

Framework for the assessment of specific autonomic functions following SCI

For it to be part of clinical evaluation of individuals with SCI
# Autonomic Standards Assessment Form

**Patient Name:**

## General Autonomic Function

<table>
<thead>
<tr>
<th>System/Organ</th>
<th>Findings</th>
<th>Abnormal conditions</th>
<th>Check mark</th>
</tr>
</thead>
<tbody>
<tr>
<td>Autonomic control of the heart</td>
<td>Normal</td>
<td>Bradycardia</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Abnormal</td>
<td>Tachycardia</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Other dysrhythmias</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Unknown</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Unable to assess</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Autonomic control of blood pressure</td>
<td>Normal</td>
<td>Resting systolic blood pressure below 90 mmHg</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Abnormal</td>
<td>Orthostatic hypotension</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Autonomic dysreflexia</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Unknown</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Unable to assess</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Autonomic control of sweating</td>
<td>Normal</td>
<td>Hyperhidrosis above lesion</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Abnormal</td>
<td>Hyperhidrosis below lesion</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Hypohidrosis below lesion</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Unknown</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Unable to assess</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Temperature regulation</td>
<td>Normal</td>
<td>Hyperthermia</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Abnormal</td>
<td>Hypothermia</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Unknown</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Unable to assess</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Autonomic and Somatic Control of Broncho-pulmonary System</td>
<td>Abnormal</td>
<td>Unable to voluntarily breathe requiring full ventilatory support</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Impaired voluntary breathing requiring partial vent support</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Voluntary respiration impaired does not require vent support</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Unknown</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

## Lower Urinary Tract, Bowel and Sexual Function

<table>
<thead>
<tr>
<th>System/Organ</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Lower Urinary Tract</strong></td>
<td></td>
</tr>
<tr>
<td>Awareness of the need to empty the bladder</td>
<td></td>
</tr>
<tr>
<td>Ability to prevent leakage (continence)</td>
<td></td>
</tr>
<tr>
<td>Bladder emptying method ________ (specify)</td>
<td></td>
</tr>
<tr>
<td><strong>Bowel</strong></td>
<td></td>
</tr>
<tr>
<td>Sensation of need for a bowel movement</td>
<td></td>
</tr>
<tr>
<td>Ability to Prevent Stool Leakage (Continence)</td>
<td></td>
</tr>
<tr>
<td>Voluntary sphincter contraction</td>
<td></td>
</tr>
<tr>
<td><strong>Sexual Function</strong></td>
<td>Psychogenic</td>
</tr>
<tr>
<td>Genital arousal (erection or lubrication)</td>
<td>Reflex</td>
</tr>
<tr>
<td>Orgasm</td>
<td></td>
</tr>
<tr>
<td>Ejaculation (male only)</td>
<td></td>
</tr>
<tr>
<td>Sensation of Menses (female only)</td>
<td></td>
</tr>
</tbody>
</table>

2 = Normal function, 1=Reduced or Altered Neurological Function
0=Complete loss of control NT=Unable to assess due to preexisting or concomitant problems

## Urodynamic Evaluation

<table>
<thead>
<tr>
<th>System/Organ</th>
<th>Findings</th>
<th>Check mark</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sensation during filling</td>
<td>Normal</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Increased</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Reduced</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Absent</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Non-specific</td>
<td></td>
</tr>
<tr>
<td>Detrusor Activity</td>
<td>Normal</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Overactive</td>
<td></td>
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<tr>
<td></td>
<td>Underactive</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Acontractile</td>
<td></td>
</tr>
<tr>
<td>Sphincter</td>
<td>Normal urethral closure mechanism</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Normal urethral function during voiding</td>
<td></td>
</tr>
<tr>
<td></td>
<td>incompetent</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Detrusor sphincter dyssynergia</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Non-relaxing sphincter</td>
<td></td>
</tr>
</tbody>
</table>
ASAF

• New system to document the impact of SCI on autonomic function

• It communicates the neuro effects of injury on autonomic function in all pts with SCI in conjunction with the ISNCSCI

• It provides a framework with which to communicate the effects of specific SCI on CV, broncho-pulmonary, sudomotor, bladder, bowel and sexual function
CONCLUSIONS
Conclusions

• AD has only been studied for < 100 years
• SCI patients with a T6 or higher LOI are at risk for AD
• AD can potentially be life-threatening
• AD has a significant impact on patient’s function and/or quality of life
• General patient population and healthcare providers will benefit from more AD knowledge
• Education is essential to assist with the prevention and treatment of AD
REFERENCES
References


References

Ginis KM, et. al. *Results of a knowledge translation initiative to enhance knowledge and use of autonomic dysreflexia clinical practice guidelines among Canadian paramedics.* ASIA/ISCOS conference 2015. K. Martin Ginis, Canada


References

http://www.asia-spinalinjury.org/

Wan D, et. al. Life-threatening outcomes associated with autonomic dysreflexia; A clinical review. The journal of spinal cord medicine 2014 vol 37 no 1


Silver, JR. The history of Guttmann’s and Whitteridge’s discovery of autonomic dysreflexia. Spinal Cord 2000, 38, 581-596
QUESTIONS?