

## Dysautonomia: Views and reviews

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### 1. INTRODUCTION

Dysautonomia may accompany DAI. Although DAI occurs only in TBI, Dysautonomia may occur in both traumatic conditions as well as less commonly in non traumatic conditions. Multiple clinicians have coined different name /syndrome for it without knowing that they are basically dealing with same entity (Table 1).

### 2. PATHOPHYSIOLOGY

Current evidence suggests two theories; (1) Association between hypothalamic injury and autonomic dysregulation; (2) Result of a functional disconnection of hypothalamus and diencephalon from rest of central nervous system probably because of axonal injury of afferent and efferent pathways. This explains the reported association between DAI and dysautonomia.

Diagnosis is based mainly on Seven main findings (criteria) are presented in the table 2. It has been suggested to consider the diagnosis of dysautonomia if atleast five of the findings are present. Pupillary dilatation is also common in dysautonomia but it is not a criteria. In case of infection may result in fever, tachycardia, tachypnea, and diaphoresis. Fever also worsens mental status and may result in posturing. Dysautonomia may only be diagnosed when infection is ruled out with investigations or fully treated. Dysautonomia typically presents with delayed onset minor symptoms at start if not treated becomes florid soon. Signs of dysautonomia are mistaken as agitation and infection.

### 3. PHASES OF DYSAUTONOMIA

Phase I is the time in which patient is intubated sedated with or without chemical paralysis. Dysautonomia is usually unrecognized in this phase; average duration is one week.

Phase II occurs after stoppage of sedation and symptoms of dysautonomia are apparent. Average duration 74 days but may be quite variable.

**Table 1**

Synonyms of Dysautonomia syndrome (Singleton et al. 2008)

Sympathetic storming(SS)	Paroxysmal autonomic Instability with dystonia (PAID)	Sympatho adrenal response
Paroxysmal .S.S	Autonomic dysfunction syndrome(ADS)	Central dysregulation
Neurostorming	Acute midbrain syndrome	Tonic cerebellar fits
Brain stem attack	Midbrain dysregulatory syndrome	Fiver of central origin
Acute hypothalamic instability	Hypothalamic-midbrain dysregulation syndrome	Tonic decerebrate response
Diencephalic seizure	Hyperpyrexia associated with muscle contraction	Dysautonomia

**Table 2**

Criteria / findings of dysautonomia syndrome (Singleton et al. 2008)

Tachycardia	Diaphoresis
Tachypnea	Rigidity
Hypertension	Posturing
Fiver	-

Phase III occur when diaphoresis stops and paroxysmal episodes of autonomic dysfunction subsides. It represents the 'burnout' dysautonomia leaving the patient with variable dystonia and/or spasticity.

#### 4. TREATMENT

Goal of dysautonomia treatment are to prevent secondary injury caused by the paroxysmal autonomic episode. There are three mechanisms by which dysautonomia can worsen secondary brain injury, hyperthermia, rigidity and paroxysm of dysautonomia with associated elevation of circulating catecholamine's level. No study has shown that treatment of dysautonomia improves functional outcome.

Numerous pharmacological interventions have been employed in attempt to treat dysautonomia. Narcotics and benzodiazepines are used to reduce paroxysm. Clonidine (an alpha 2 adrenergic agonist) causes decreased central sympathetic outflow, resulting in decreased blood pressure and reduced levels of circulating catecholamine. Dantrolene used for malignant hyperthermia via inhibition of intra cellular release of calcium, causing decreased muscle contraction. The hypertension and tachycardia of dysautonomia can be controlled effectively via the use of beta Blockers like labetalol. Bromocriptine (dopamine D2 agonist) has shown promise and potential to control dysautonomia by controlling fiver and diaphoresis. Intra thecal Baclofen appears to be quite effective autonomic dysfunction in limited number of patients. Finally non selective beta blocker Propranolol also appears quite useful in controlling multiple components of autonomic dysfunction. It reduces blood pressure, heart rate, temperature, and muscle tone, circulating catecholamine, cardiac work and resting energy expenditure (Singleton et al. 2008).

#### 5. CONCLUSION

In setting of sever TBI, both DAI and dysautonomia can exert a profound negative influence on the ultimate outcome of affected individuals.

#### REFERENCES

- Singleton RH, Okonkwo DO, Adelson DP. Diffuse axonal injury and dysautonomia. In: Bhardwaj A, Ellegala DB, Kirsch JR, eds. Acute brain and spinal cord injury: evolving paradigms and management. New York, NY: Informa Healthcare, 2008, 49-65