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Non Invasive Assessment Of The Effect Of Implantable Phrenic Nerve Stimulation in Two Paediatric Patients using Structured Light Plethysmography.

Type: Case Report

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Abstract Body

Introduction:
Patient A is a 17 year old girl who sustained a C1 complete spinal cord injury at the age of 9 years. Patient B is a 10 year old boy who sustained a complete C1 spinal injury at the age of 6 years. Both children show no respiratory drive and have been invasively ventilated via tracheostomy since the time of injury. Neither has any other significant pathology. Following assessment both patients underwent insertion of bilateral Atrostim Implantable Phrenic Nerve Stimulators (IPNS) in 2013.

Methodology:
The children were studied using Structured Light Plethysmography (SLP) (Thora3Di™ PneumaCare Ltd, UK). SLP enables non-invasive, non-contact assessment of the compartmental change of Rib Cage (RC) v Abdominal (AB) and Right (RH) v left (LH) hemi-thorax. The movement of a projected grid of light is analysed to derive a Konno-Mead loop (KM) from which KM Principal Angle (Phi), KM Spread, Overall phase (OPhi), Windowed phase (W phase), Phase breath (B phase) and a measure of variation of per-breath phase (B phase Ent) can be calculated. Each child was monitored in a sitting and supine position both on invasive ventilation and on IPNS. Patient B was reassessed at 75% and 100% IPNS.

Results:
RC v AB: In both patients, in both positions, Phi became more negative with IPNS, indicating a shift from RC to an AC / diaphragmatic volume recruitment. There was an increase in KM spread in the supine position, but either no change or a negative change in the sitting position. KM Spread and Phase measurements became positive indicating a more complex multi phased mode of volume recruitment. Measurements changed when Patient B was paced at 75% and 100% IPNS; Phi becoming more negative and the KM spread, decreased. B Phase Ent decreased in both positions indicating more respiratory stability.

RH v LH: Patient A showed an increase in right sided ventilation in the sitting position following IPNS. There was a change in Phi between sitting and lying positions for both patients, however these changes were not the same for each patient. There was no difference in KM spread before or after IPNS in either patient.

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Patients A & B, Chest V Abdominal Wall Components on Ventilation and IPNS

	KM Angle	KM Spread	Overall Phase	W Phase	B Phase	B Phase ENT
Pat A Sitting, Ventilator	23.6	0.08	25.6	2.9	3.9	0.68
Pat A Sitting IPNS	-14.6	0.27	35.9	7.5	14.3	0.57
Change	-38.6	0.19	10.3	4.6	10.4	-0.11
Pat B Sitting Ventilator	31.4	0.25	53.4	11.6	14.4	0.82
Pat B Sitting IPNS	-28.7	0.51	58.9	23.1	49.4	0.67
Change	60.1	0.26	5.5	11.5	35	-0.15
Pat A Supine Ventiator	-26.1	0.05	8.8	0.8	1	0.93
Pat A Supine IPNS	30.9	0.05	17	1.4	3.7	0.54
Change	57	0	8.2	0.6	2.7	-0.39
Pat B Supine Ventilator	0.8	0.21	27.9	4	7.4	0.78
Pat B Supine IPNS @75%	-48.6	0.18	65.2	94.1	74.1	0.62
Change	-33.8	-0.11	31.9	6.3	15.6	-0.12
Pat B Supine IPNS@100%	-34.6	0.1	59.8	10.3	23	0.67
Change	-47.8	-0.03	37.3	90.1	66.7	-0.17

Conclusion:

IPNS offers significant quality of life improvements for the patient, but effectiveness is often subjective. SLP offers a non–invasive method to objectively quantify and record the regional, idiosyncratic and positional effectiveness of IPNS, when compared to conventional ventilation and varying % IPNS support.