is the influence of phonological awareness on children who have dyslexia² may cause late reading ability. Bradley & Bryant's study discovered that children with dyslexia have not only lower phonological performance but also low literacy skills compared to their teens. A variety of findings approved that dyslexic puberty shows low phonological awareness along with short-time memory.

There are other shreds of evidence that show later reading skills happen with children who have normal development. For example, Cataldo & Ellis (1988) investigated the relationship between reading, phonological awareness, and spelling in 3rd-grade school children. Study shows how early phonological awareness happens, so late children will have the reading ability.

REFERENCES:

- 1.Brown, R. A. (1973). *First language: The early stage*. Cambridge, Harvard University Press.
- 2.Gillon, g. (2008). An intervention program for children at risk for reading disorder. The University of Canterbury. New Zealand.
 - 3. Gibson, E. J., & Levin, H. (1975). The psychology of reading. Cambridge, Mass.
 - 4. Gibson, E. J., & Levin, H." The psychology of reading". Cambridge. MIT Press.
- 5.Ingram, D. *Procedures for Phonological Analysis of Children's Language*. Baltimore University Park Press.
- 6.Ingram, D. (1974). *Phonological rules in young children. Journal of Child Language*. Stanford University.
 - 7. Kiparsky, P. (1975). Comments on the role of phonology in language. MIT Press.
 - 8.Menn, L. (1971). Photo tactic rules in beginning speech. Lingua. The Netherlands.
- 9.Read, C. (1975). *Children's categorization of speech sounds in English*. Urbana, National Council of Teachers of English.
- 10.Read, C. (1975). Lessons to be learned from the preschool orthographer. New York: Academic Press
- 11.Lenneberg. (1975). *Foundations of language development.* New York: Academic Press.
- 12.Smith, F. (1975). *The relation between spoken and written language*. New York. Academic Press.
 - 13Appendix. http://www.education.canterbury.ac.nz

RESPIRATORY SUPPORT FOR TRAUMATIC BRAIN INJURY

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² Dyslexia- a variable often familial learning disability involving difficulties in acquiring and processing language that is typically manifested by a lack of proficiency in reading, spelling, and writing

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The most common causes of cerebral injury in crtically ill patients are severetraumatic brain injury (TBI). TBI frequency in various regions of the Russian Federation is 4-4,5 cases per 1000 population per year. General mortality in traumatioc brain injury is 5-10%, but in severe forms TBI with intracranial hematomas, foci of head contusion brain mortality increases to 41-85%.

The main factors of delayed cerebral injury include; arterial hypotension, hypoxemia, hyper and hypocapnia, anemia, hyperthermia and intracranial hypertension. All these conditions trigger a chain of pathological reactions leading to impaired delivery of oxygen and nutrient substrates to brain cells. One of the most effective methods to maintain the necessary cerebral perfusion and oxygenation is respiratory support.

In cases of TBI, respiratory dysfunction is the most common medical complication which occurs. Up to one-third of patients with severe traumatic brain injury develop Acute Respiratory Distress Syndrome. In this syndrome, there is inflammation of the alveolar-proteins entering the interstitial space and alveoli. Between 20 and 30 % of individuals who develop ARDS die as a result of the pulmonary infiltrate leading to respiratory failure.

Respiration control within the Central Nervous System is effective. Respiration is controlled by respiratory centres: the inspiratory and expiratory centres are located in the medulla oblongata; the pneumatic and apneustic centres are located in the pons. Together they are known as the Respiratory Control Centres(RCCs).

Neurones in the medulla trigger inspiration (sending signals to the phrenic and intercostal nerves). Frequency of respiration is controlled by the pneumatic center, and the intensity of breathing is controlled by the apneustic centre.

According to pathophysiology, there are several possible mechanisms which are thought to contribute to the respiratory complications seen in cases of brain injury.

One of them is Sympathetic Storm. There is an (within seconds) sympathetic discharge when an injury occurs which raises plasma adrenaline levels to approximately 1,200 times the normal c value. The adrenaline levels do then fall, but they remain at 3 times higher than normal for approximately 10 days. This results in elevated intravascular pressure, which damages the endothelium and produces pulmonary oedema (due to disruption on the alvelolar-capillary barrier). Pulmonary oedema becomes protein-rich and goes into interstitial and alveolar spaces.

There is also inflammatory theory. As a direct result of the brain damage, a systematic inflammatory reaction occurs, which in turn brings about an alternation in blood-brsaain barrier permeability and infiltration of neutrophils and activation of macrophages in the alveolar spaces causing secondary damage to the lung tissue.

It is clear that, respiratory assessment of traumatic brain injury patient require medical information. The respiratory physiotherapist should pay close arttention to the following information when assessing a patient with traumatic brain injury in the acute situation:arterial blood gases; chest X-rays; respiratory patterns; pulmonary function testing; peak cough flow.

This is time saying about past respiratory history. This will frequently be provided by family memmbers in cases of acute and severe traumatic brain in jury. The therapist should inquire about any previous respiratory conditions, as well as smoking history.

In fgeneral observation, the therapist will watch the patient and note the general respiratory pattern and posture; whether there is any cyanosis or accessory muscle use, as well as noting speech patterns if appropriate.

A respiratory pattern assessment includes: breathing rate; depth of breaths,-the symmetry of air intake/lung expansion; the regularity of breaths.

Other assessment techniques may include:

-Percussion - it is used to detect chest resonance. Percussion applied to the patient's chest prodiuces audible sounds which camn be interpreted by a skilled examiner to discern fluid, air or solid material wthin the chest cavity.

-Auscultation -it involves using a stethoscope to listen to lung sounds. Abnormal lung sounds includes wheezes, crackles, rhonchi and pleural rub.

The respiratory management is all about recovering patients from illnesses tpo healthy lifestyle. In ther acute stages of traumatic brain injury, the aims of management in the In tensive Care Unit are to maintain oxygen delivery in order to limit secondary neurological damage. Mechanical ventilation is commonly used with 3 aims: 1.To prevent/minimize hypoxia; 2. To prevent/minimize hypercapnia; 3. To protect the airway from the risk of aspiration. It is acknowledge that difficulties are frequently encountered when weaning these patients from mechanical ventilation.

A number of recent studies have investigated the use of protective ventilation in the early stages following traumatic brain injury.

Conclusion: During mechanicall ventilation, the aim of physiotherapy is to optimize respiratory function while maintaining the neuromusculoskreletal system. Physiotherapy interventions include: Positioning – to use gravity to aid sputum removal from the lungs—Manual and ventilator hyperinflation —Weaning from mechanical ventilation; Non-invasive ventilation; Percussion, vibration, suctioning – all aim to aid removal of sputum; Respiratory muscle strengthening; Breathing exercises and mobilization.

LITERATURE

- 1. Bein T, Grasso S, Moerer O, et al. The standard of care of patients with ARDS: ventilatory settings and rescue therapies for refractory hypoxemia. Intensive Care Med. 2016;42(5):699-711. doi: https://doi.org/10.1007/s00134-016-4325-4
- 2. Brain Trauma Foundation. Guidelines for the Management of Severe Traumatic Brain Injury (4th Edition). Neurosurgery. 2017;80(1):6-15. doi: https://doi.org/10.1227/NEU.000000000001432