Sleep and breathe

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A 55 years old man comes to visit to our ER suffering from dyspnoea without other symptoms.

Anamnesis:

- Moderate COPD (Gold stage III): heavy past-smoker and varnisher
- Chronic respiratory acidosis with full kidney compensation
- Recent thorax CT: no signs of emphysema, interstitial disease or other diseases
- Last Spirometric exam: mixed obstructive-restrictive pattern (FEV1/FVC=50%, FEV1=45% and DLCO 50% of predicted)
- No other significant information

While waiting for the visit, he looses coscience and falls down: the physician finds him in respiratory arrest few seconds later.

ALS (Advanced Life Support) maneuvers are started; the presentation rythm is a non-shockable one (PEA: pulseless electrical activity).

The patient is intubated, ventilated and after 2 RCP cycles a valid pulse becomes clear.

One month before, the man had contacted the territorial emergency service (118) for dyspnoea and had been found at home in cardiac arrest.

He had been resuscitated and transferred to our Hospital where he had been hospitalized into our ICU and, after prompt extubation (2 days), transferred to our HDU where he had undertaken several cycles of non-invasive ventilation for persistent hypercapnic respiratory failure.

That time, hemogas was consistent with respiratory acidosis but blood analysis, imaging exams, Holter-ECG, coronary angiography were *all unremarkable for plausible causes of cardiac arrest.*

He had been dismissed with the prescription of nocturnal CPAP because of sleep apneas and diurnal hypercapnia with the indication for a polysomnography in order to further specifies the kind of apnea-hypopnea episodes.

A diagnosis of ***cardiac arrest due to acute hypercapnic respiratory failure*** was done.

Which are "the five most dangerous" in the ED?

1. Acute Coronary Syndromes (STEMI, NSTEMI, sudden cardiac death) and Arrythmias
2. Aortic dissection and aortic syndromes
3. Tense Pneumothorax
4. Massive Pulmonary Embolism
5. Ischemic/Hemorragic stroke

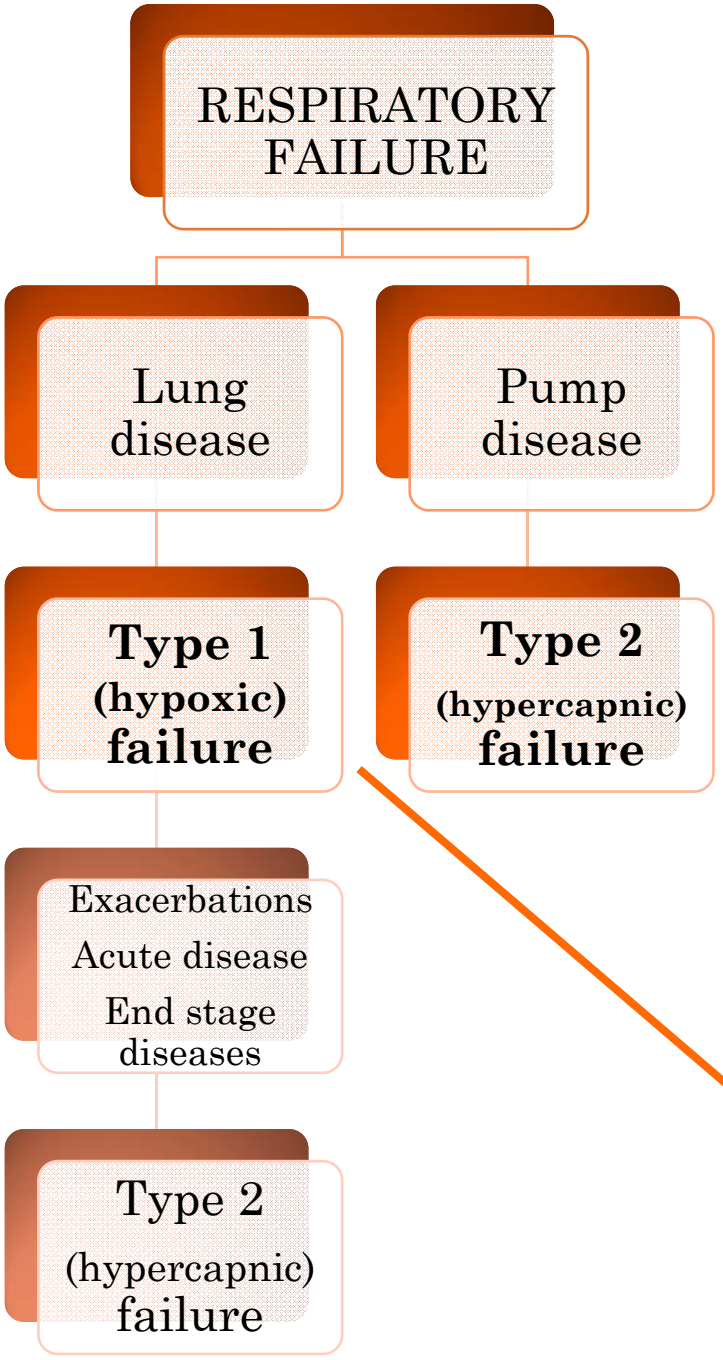
Dou you agree with the indications at the dismission?

Had you further investigated this patient despite all negative exams?

If yes, which investigation have you proposed?

24 h after the admission, the patient was successfully extubated and without neurologic sequelae. Again transferred to our HDU where nocturnal non-invasive ventilation was started owe to apneas and morning hypercapnia.

Do you know mechanisms and causes of hypercapnia?



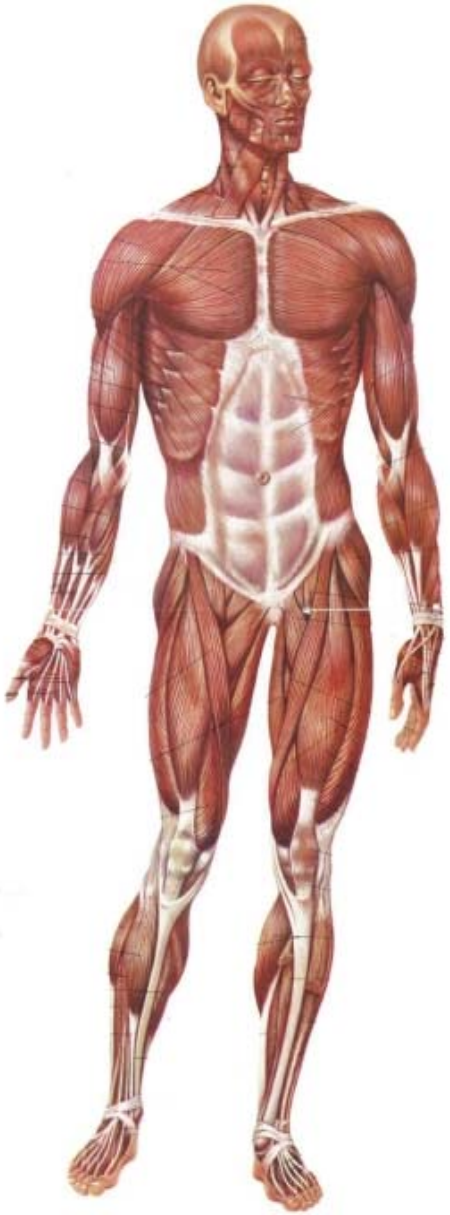
1 mechanism of hypercapnic respiratory failure:

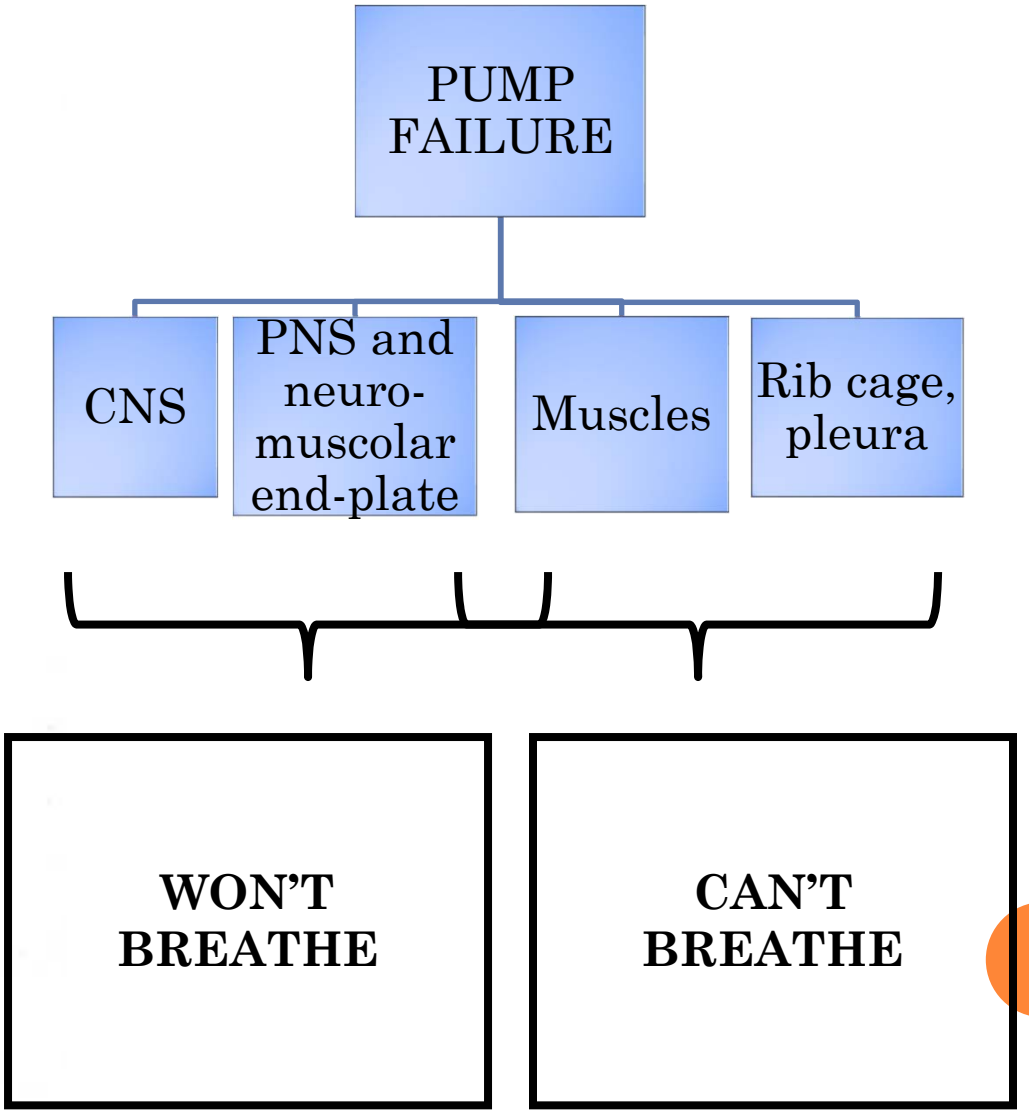
1. **<u>Hypoventilation</u>**

5 mechanisms of hypoxic respiratory failure:

1. Shunt
2. Ventilation/perfusione mismatch
3. Diffusion impairment
4. Reduced inspiration fraction of O_2
5. *Hypercapnia*

HYPERCAPNIA = HYPOVENTILATION = "PUMP FAILURE"



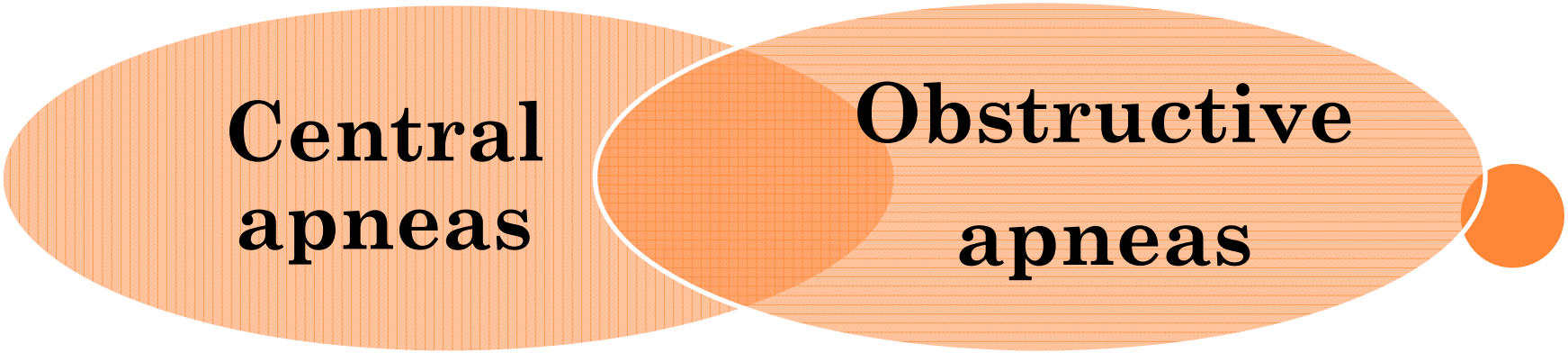


Despite doing nocturnal CPAP, ***strict clinical observational highlighted a high number of sleep-apneas not followed by any efforts of inspiration.***

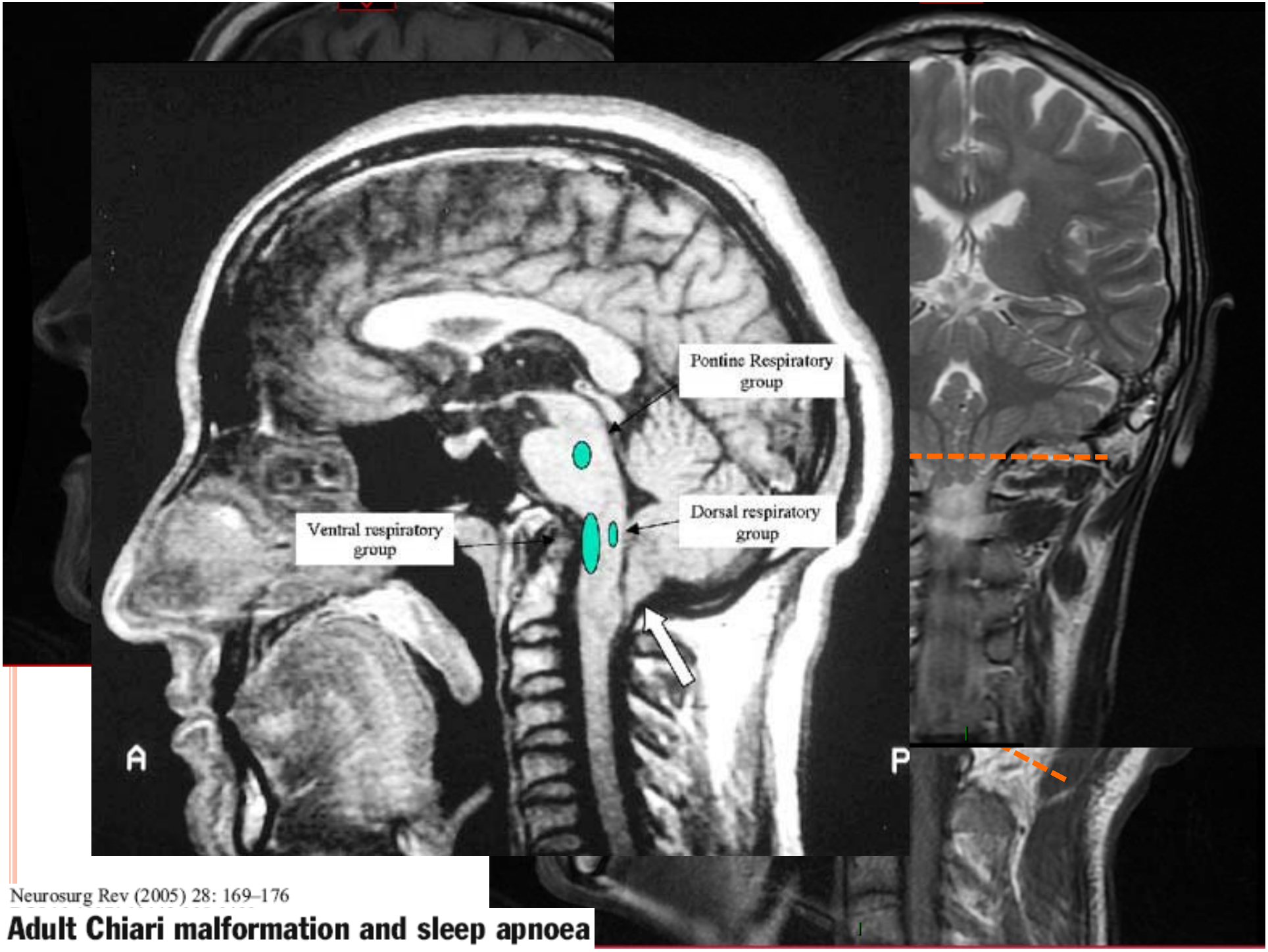
Therefore a "won't breathe" apnea syndrome was hypothesized and *a neuromuscolar disease was supposed.*

- Central sleep apnea (CSA) is defined by the cessation of air flow without respiratory effort. It is characterized by a lack of drive to breathe during sleep, resulting in insufficient or absent ventilation and compromised gas exchange.

- This condition is in contrast to obstructive sleep apnea (OSA), in which ongoing respiratory effort is present during respiratory events



Acute Hypoventilation		Chronic hypoventilation	
Respiratory drive impairment	Drugs	**CNS diseases**	Central Apnoeas (ex. Ondine maledition)
	CNS diseases (infections, **stroke**, trauma, **neoplasia congenital**)		
Peripheral nervous system	Spinal Shock	**Rib cage and pleura diseases**	Severe cyphosis
	Transverse myelitis		Thoracoplastic
	Tetanus		OHS
	Amyotrophic lateral sclerosis		Pleural drainage
	Poliomyelitis		Neuromuscolar diseases
	Guillain-Barrè disease	**Muscle diseases**	Sclerodermia
	Myasthenia		Polimyositis
	Organophospates intoxication, botulism		SLE
Muscle diseases	Muscle dystrophies		
Rib cage and pleura disease	Rib, diaphragm trauma	**Others**	Neuroendocrine (ex. Myxedema)
	Acute hyperinflaction		Electroytes disturbances
			Malnutrition
Lung, airway diseases	Muscle fatigue	**Lung, airway diseases**	COPD exacerbation



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Adult Chiari malformation and sleep apnoea

ARNOLD-CHIARI MALFORMATIONS (ACM)

Chiari malformation (CM) is primarily characterized by herniation of the cerebellar tonsils into the foramen magnum

Clinically, two main types of CM represent the vast majority of the cases: type I (adult type) (Fig. 1) and type II (infant type)

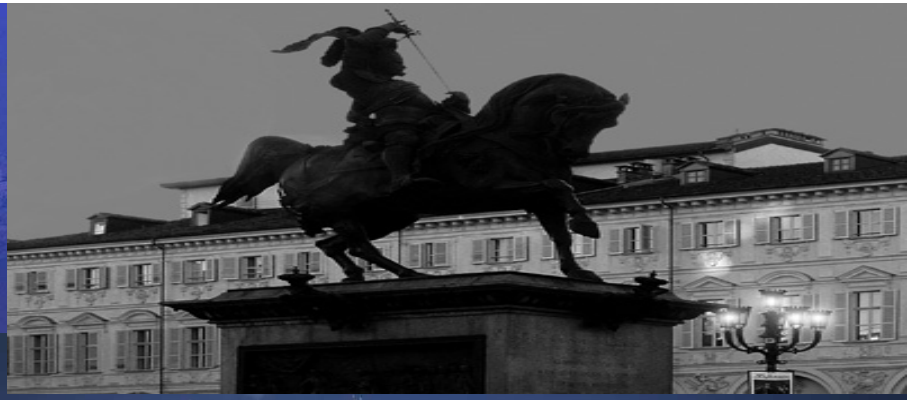
Respiratory dysfunction as the first manifestation of CM clinical features, including nocturnal hypoventilation, was described in 1965. The most frequently described respiratory dysfunction is sleep apnoea

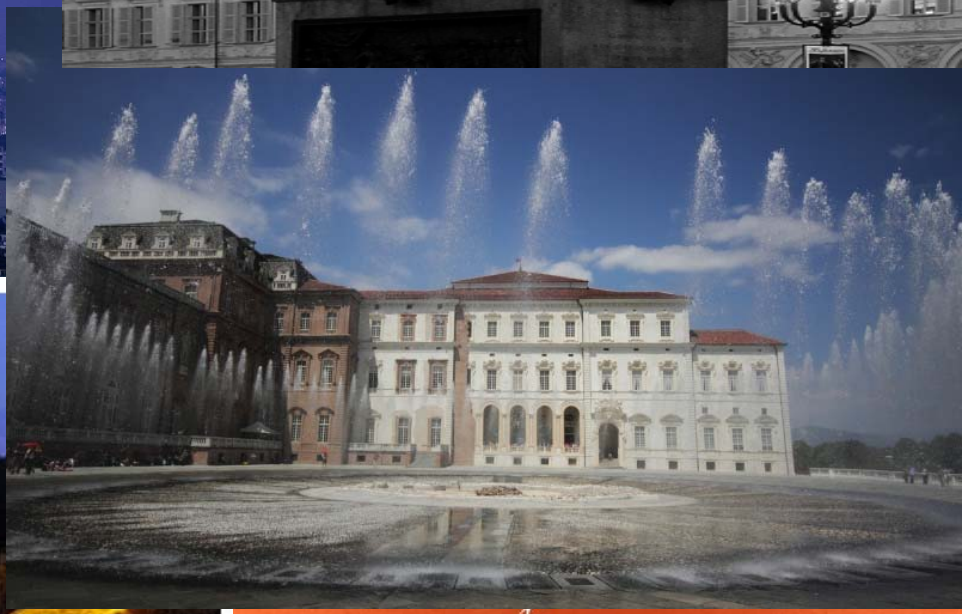
Respiratory arrest is an extremely rare presentation of type 1 ACM and few reports have been published

There are several case reports of improvement of respiratory events after surgical decompression of the posterior fossa, no strong evidences available

TAKE HOME MESSAGES

1. Hypercapnia is due to hypoventilation caused by the failure of one or more component of the respiratory "pump"
2. Although COPD is the most frequent cause of hypercapnic respiratory failure, several others should be remembered: OHS, trauma, drugs, sleep disturbances and neuromuscular diseases.
3. Sleep-apneas and "pump failure" could be ascribed to either a "won't breathe" (impaired central drive or disrupted trasmission of the signal) or a "can't breathe" mechanism. The former are central apneas while the latter are obstructive apneas.
4. There is quite overlap between these two. Polysomnography is the exam of choice when suspecting a nocturnal hypoventilation.
5. Arnold-Chiari type 1 is a common malformation of the adult that could be silent till the 5-6 decade of age.
6. ACM could appear as a respiratory disturbance or rarely as a respiratory arrest. MR is the gold standard for the diagnosis.
7. Despite scant evidences, respiratory arrest/failure is considered a compelling indication for the intervention of decompression of the posterior fossa.





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Passion lives here

Mechanisms of respiratory disturbance in Chiari I include

- compression or ischemia of the respiratory center in the medulla oblongata, resulting in central apneas
- traction upon the lower cranial nerves (IX e X) resulting in
  - Blockade of the impulses coming from both the aortic and carotid bodies (sensitivity to hypoxia)
  - Paralysis and hypotrophia of the muscles of upper airways (obstructive apnoeas)