

In contrast to the major stimuli for central
Peripheral chemoreceptors regard hypoxia as a stimulus that is most detrimental
If the response to hypoxia is depressed or removed
Then a bad situation will have ensued

At 1-2mm in size who would have guessed
That they are better perfused than all the rest
But structure to function, it all makes sense
Put them near the aorta and carotids and the situation won't be tense

But which is more influential? Or are they the same?
Studies have shown that in regards to carotids, the aortic response is lame
Although different in their blood supply and innervation
They both terminate in the NTS near the breathing station

But how do they sense this change? It can't be magic
To explain this response it must be histologic
Glomus and Sustentacular are the two types of cells that have been located
And from recent studies they have discovered how they are innervated

Glomus cells are sensitive to pH, PCO₂ and PO₂ changes
They transduce their changes through DA, ACh, NE, and neuropeptides
However, type II cells what do they do?
They merely lie around, and comfort the others to name a few

But how are changes in O₂, pH and CO₂ transduced
5 hypotheses have been suggested for this to be explained
In the Cholinergic and Dopaminergic hypotheses
The response to hypoxia was from multiple peptides

Whereas the metabolic, acidic and membrane protein channel theories
Focus on the changes in intracellular processes to hypoxia to eliminate all worries
But from this all what do we really know?
A change here, and change there, what did we really show

But from this all we can show some changes
A drop of PO₂ below 60mmHg causes CSN firing to reach maximal ranges
But the response is not limited to this
A decrease in blood flow, and drug poisoning can lead to a state of bliss

But from this all what do I know?
Was my decreased response due to peripheral or central, how can I show
But for now all will have to agree
That it would be safer for me to relax and watch some good ol'fashioned T.V.

To Be Or Not To Be Peripheral, That Is The Question!

It was a fine day when I set out to play
The task was at hand, there was no time to delay
I began to run and to my dismay
My breathing did not sway

Central or peripheral, who was to blame?
It was evident that the consequences were no game
Hypercapnic or hypoxic, how could I tell?
Pondering my thoughts I almost fell.

If central, how could it be so?
At 3mm deep in the medulla, I would never know
From Phyl 422 I knew that they enjoyed Pco₂
And who should ever know, but they were 80% of the CO₂ response too.

Through the CSF they sense any change
Preferably to levels above the normal range
In bilateral pairs: rostral, intermediate and caudal
They can cause a change in breathing you just can't fondle.

A solution with low pH or high Pco₂ applied to the brainstem
Causes a change in minute ventilation, similar to eye-movement during REM
Anaesthetics and cold were different nonetheless
When applied to the medulla, I felt so dizzy I wanted to wear my mother's dress.

The response to hypercapnia was not uniform
To see the differences all we needed to do was compare to the norm
When under anaesthetic or asleep, the response is low
But hypoxia and salicylates really make your breathing go!!

Eugene Nattie, although sound in his own mind
Was subject to speculation for what he did find
For now we will all have to agree
That all speculation we suggest is hard to see

But the discussion does not end here
Central may enjoy the presence of CO₂ when it is near
But there is more to life than just one gas
Therefore, without our peripheral sensation we would never pass

Although central plays the main role in CO₂ sensation
It is the actions of peripheral chemo's that save us from a deadly situation
They cause an immediate change in the rate and depth of breathing
Without this response we would lose all feeling