

Multiscale Model of Neural Control of Breathing



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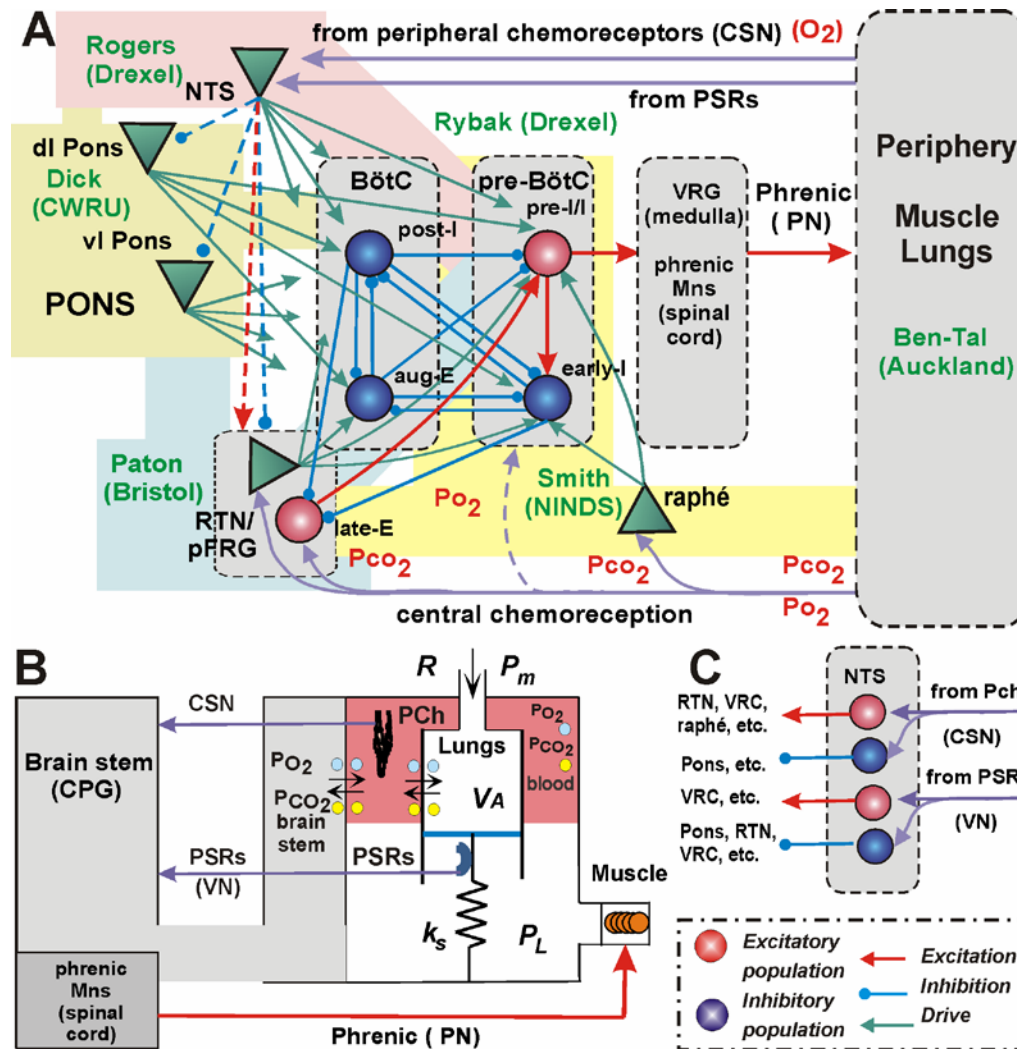


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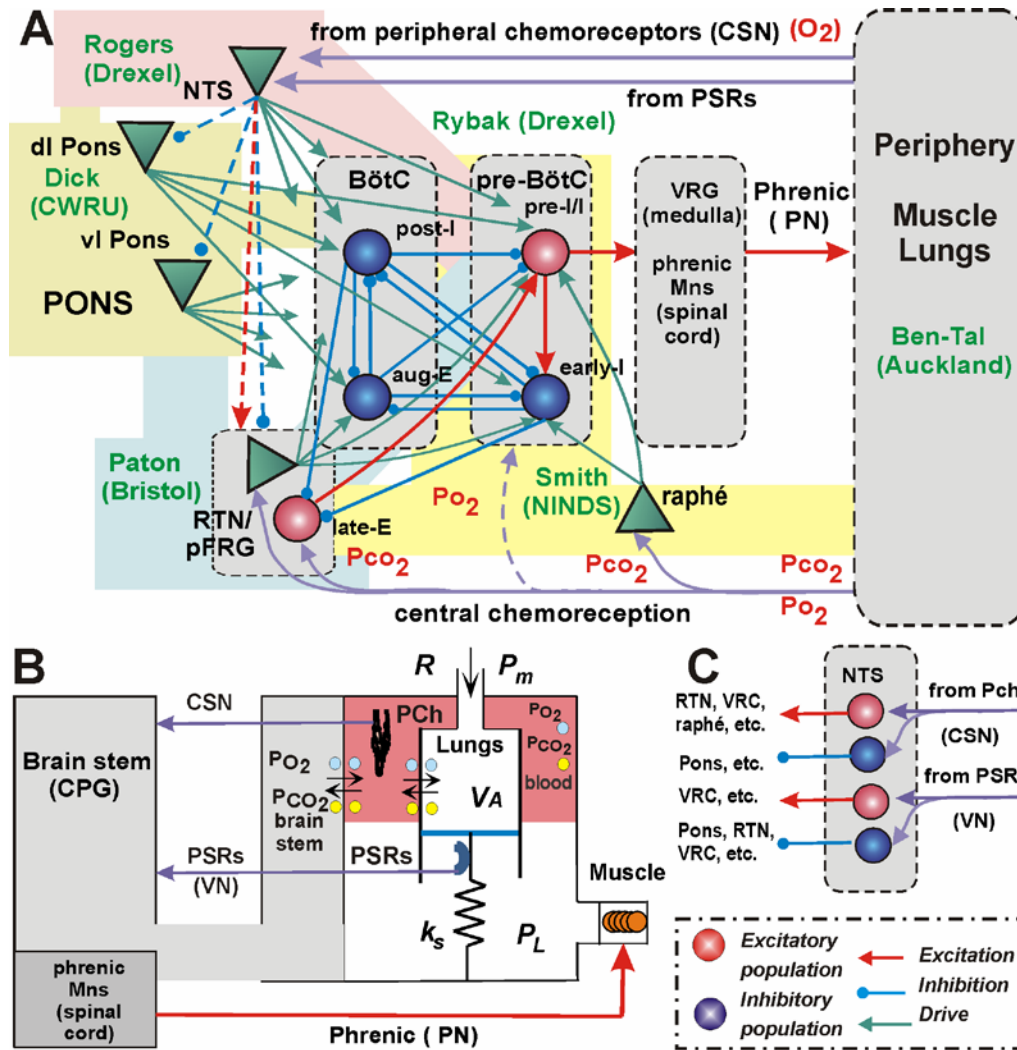
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Question:

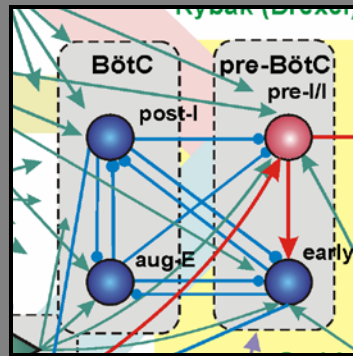
**What can we learn from simple models
to study complex systems?**

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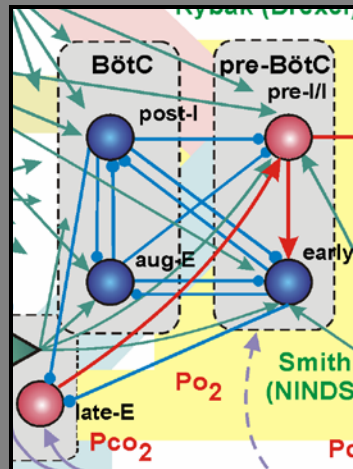
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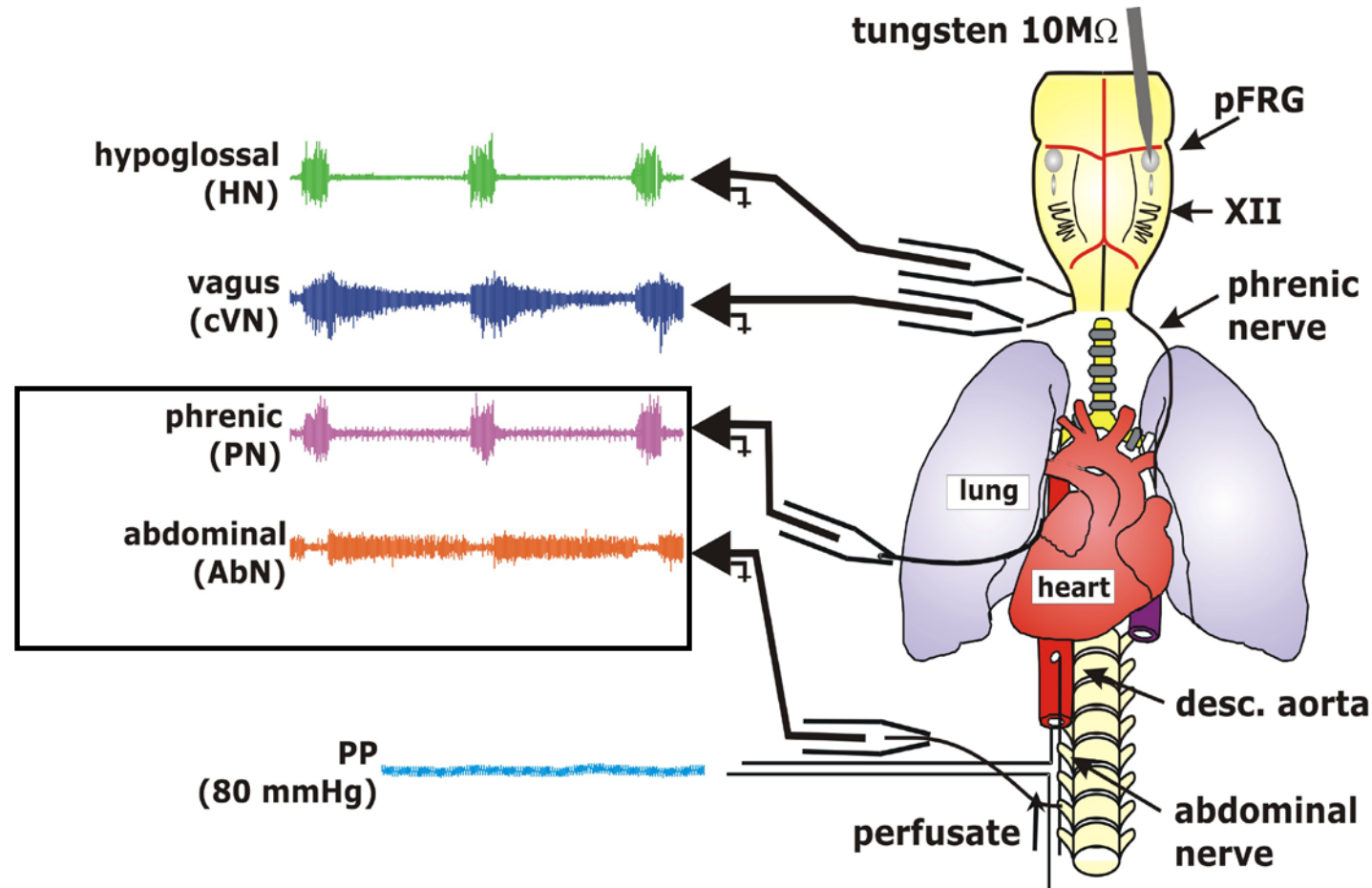
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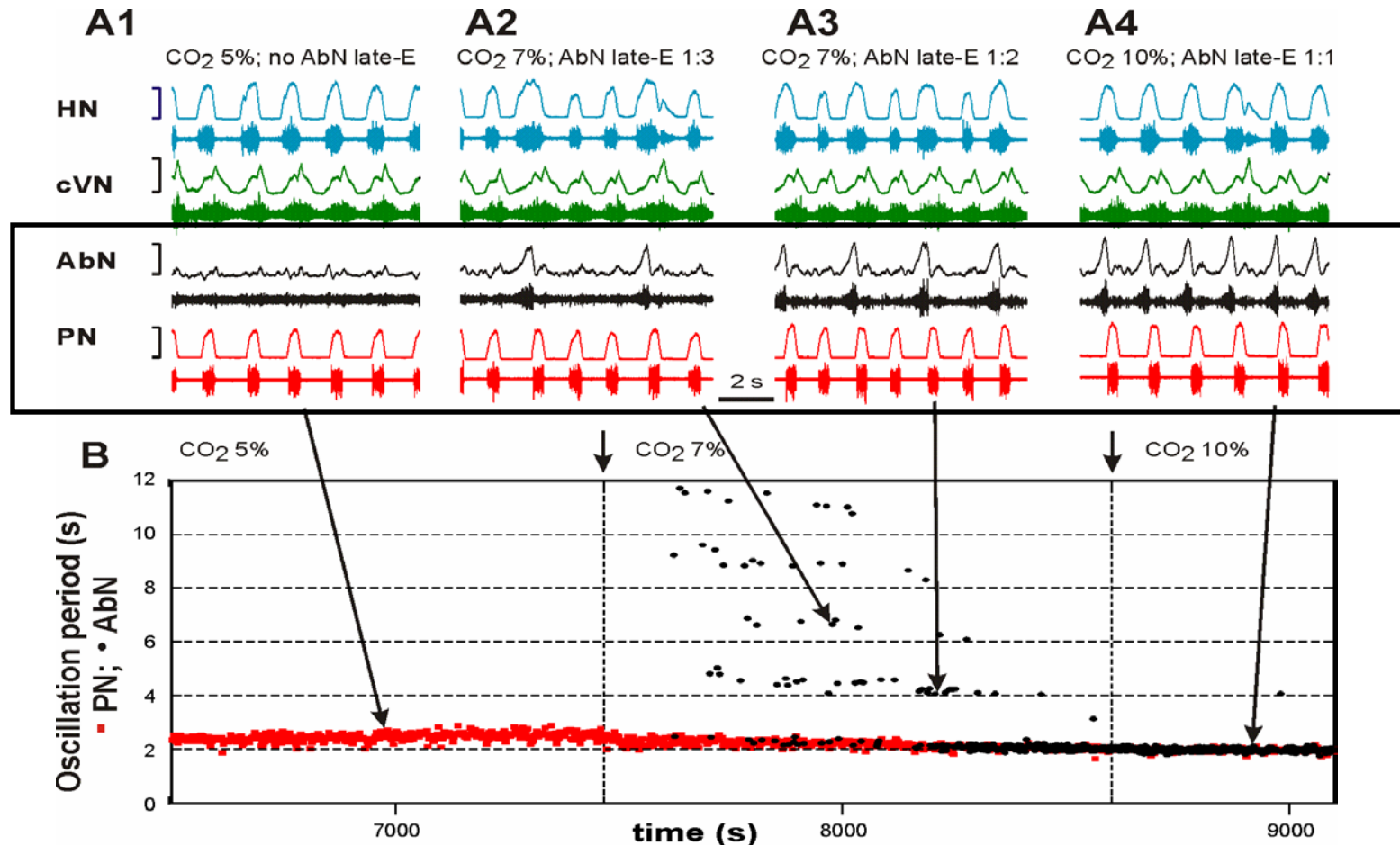
Generation and quantal acceleration of the abdominal late-expiratory activity development of hypercapnia (experimental data)



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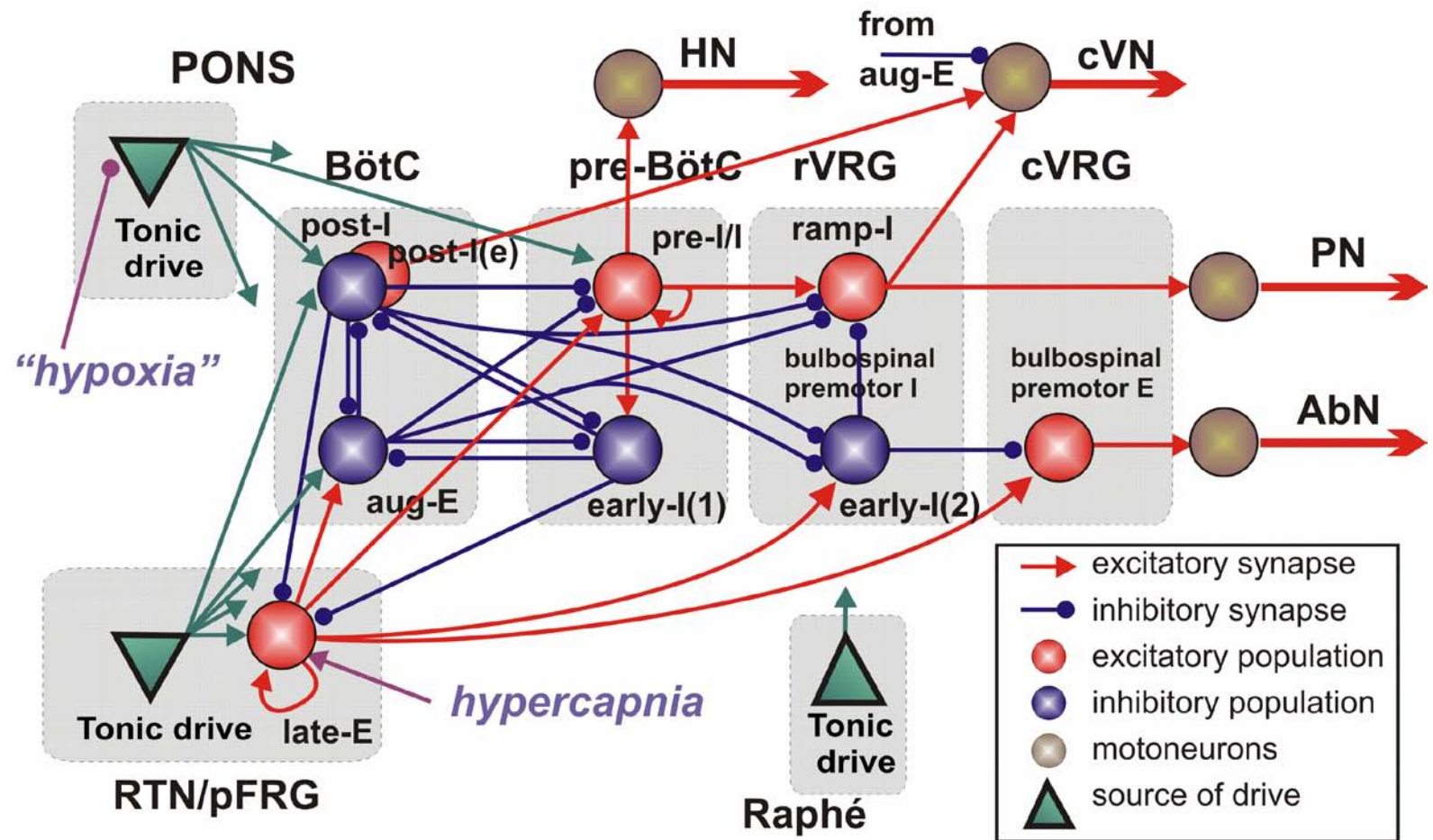
Generation and quantal acceleration of the abdominal late-expiratory activity with the development of hypercapnia (experimental data)



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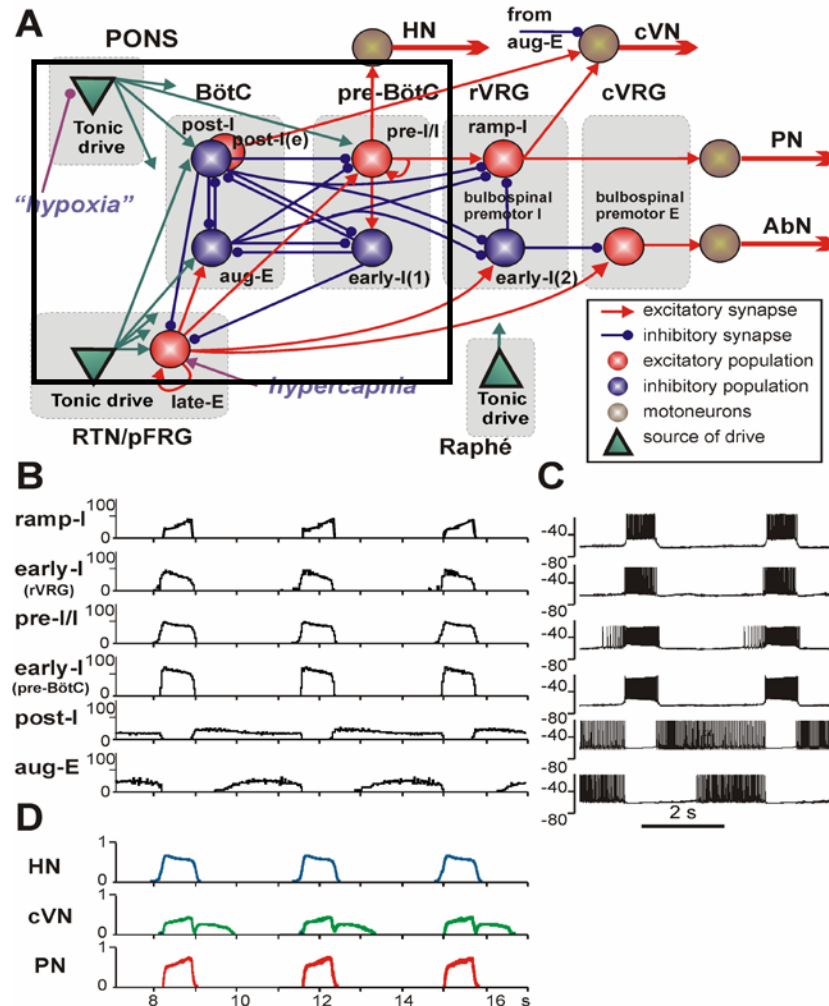
Complex computational model of the brainstem respiratory network



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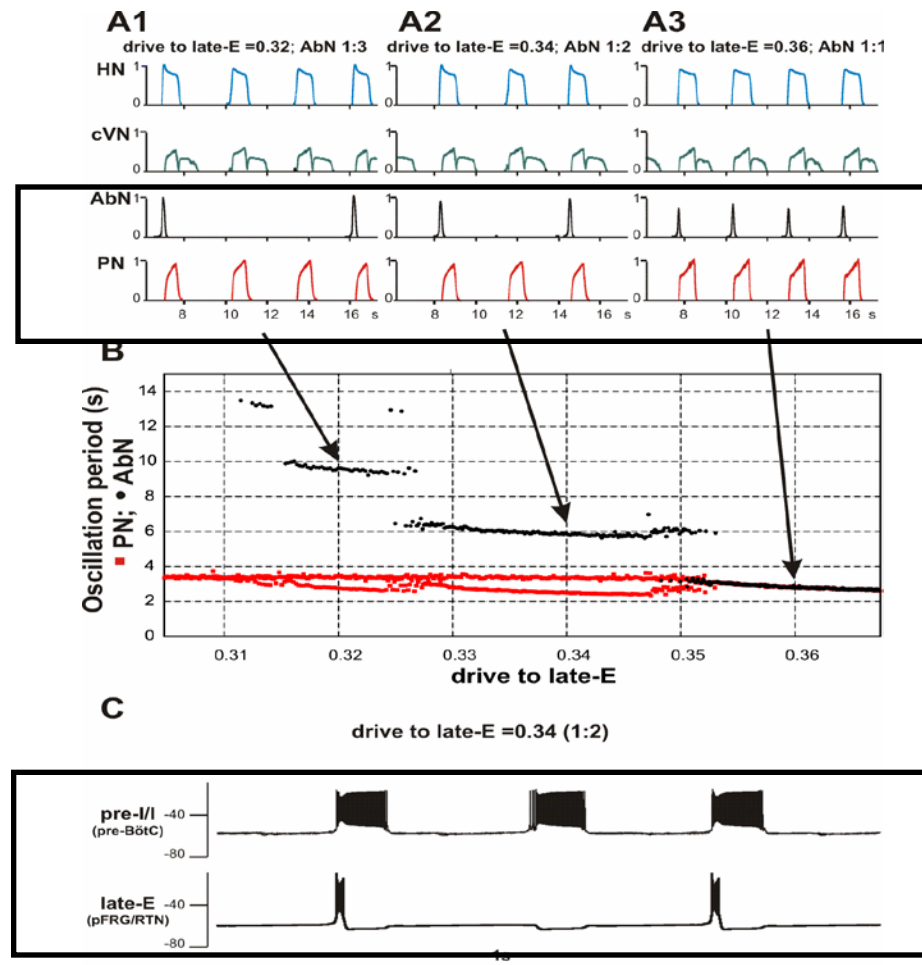
Complex computational model of the brainstem respiratory network



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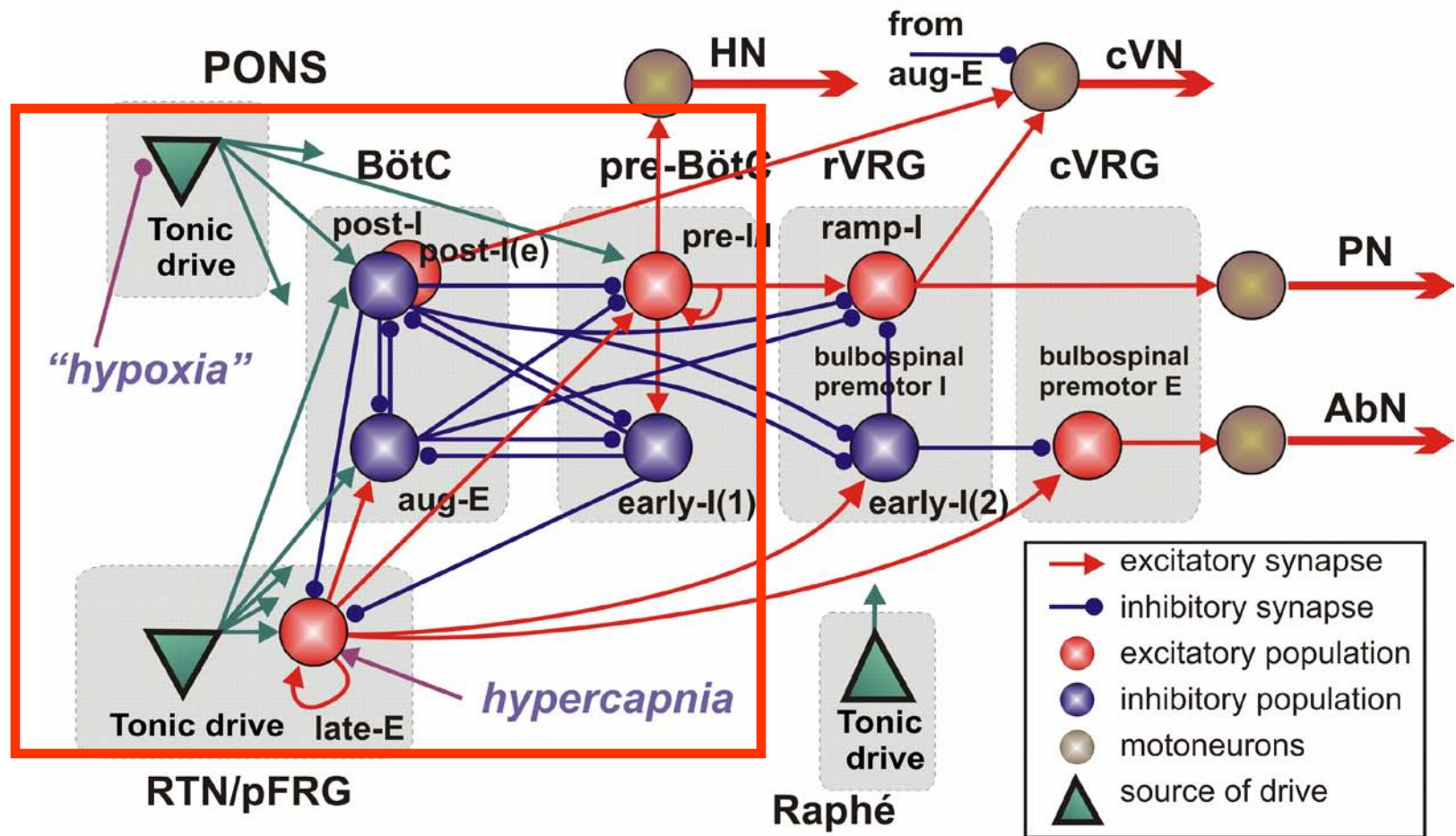
Generation and quantal acceleration of the abdominal late-expiratory activity with development of hypercapnia (model)



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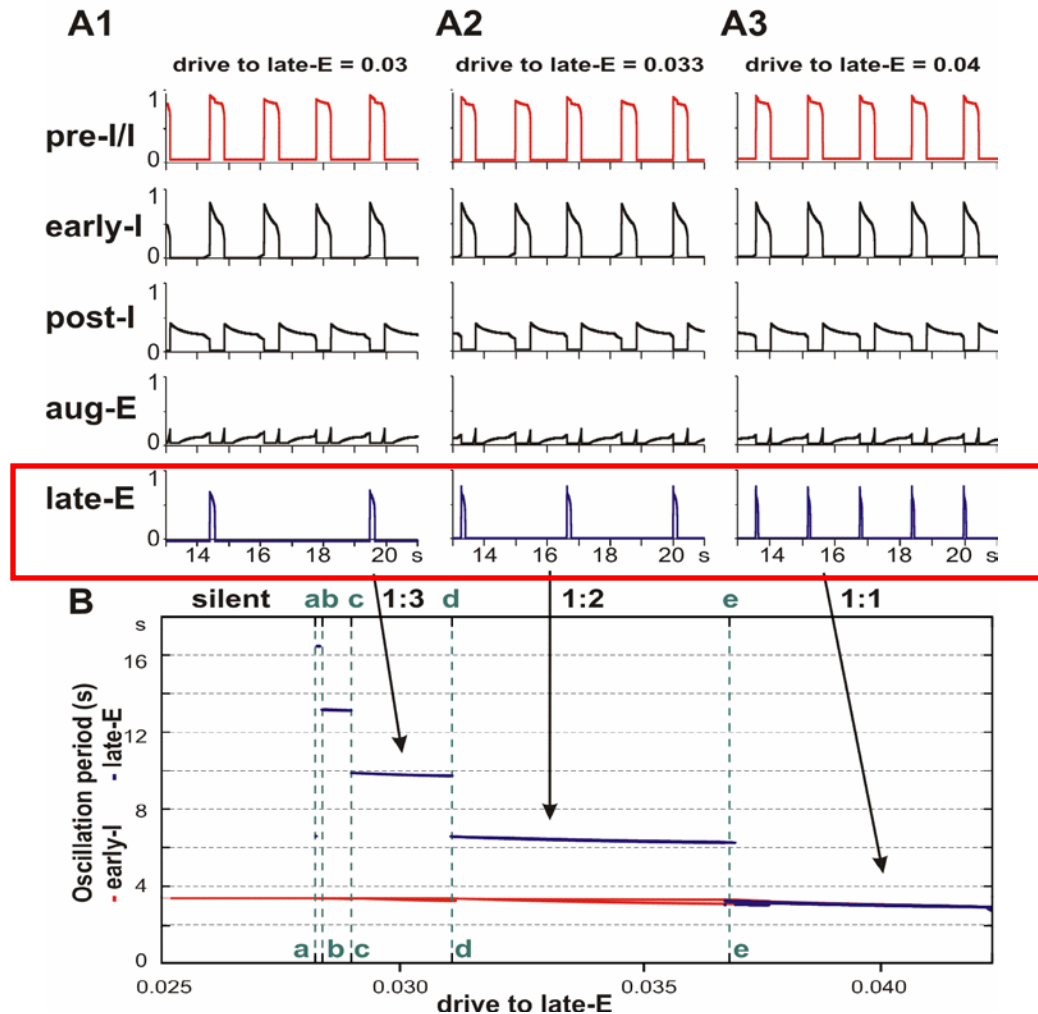
Complex computational model of the brainstem respiratory network



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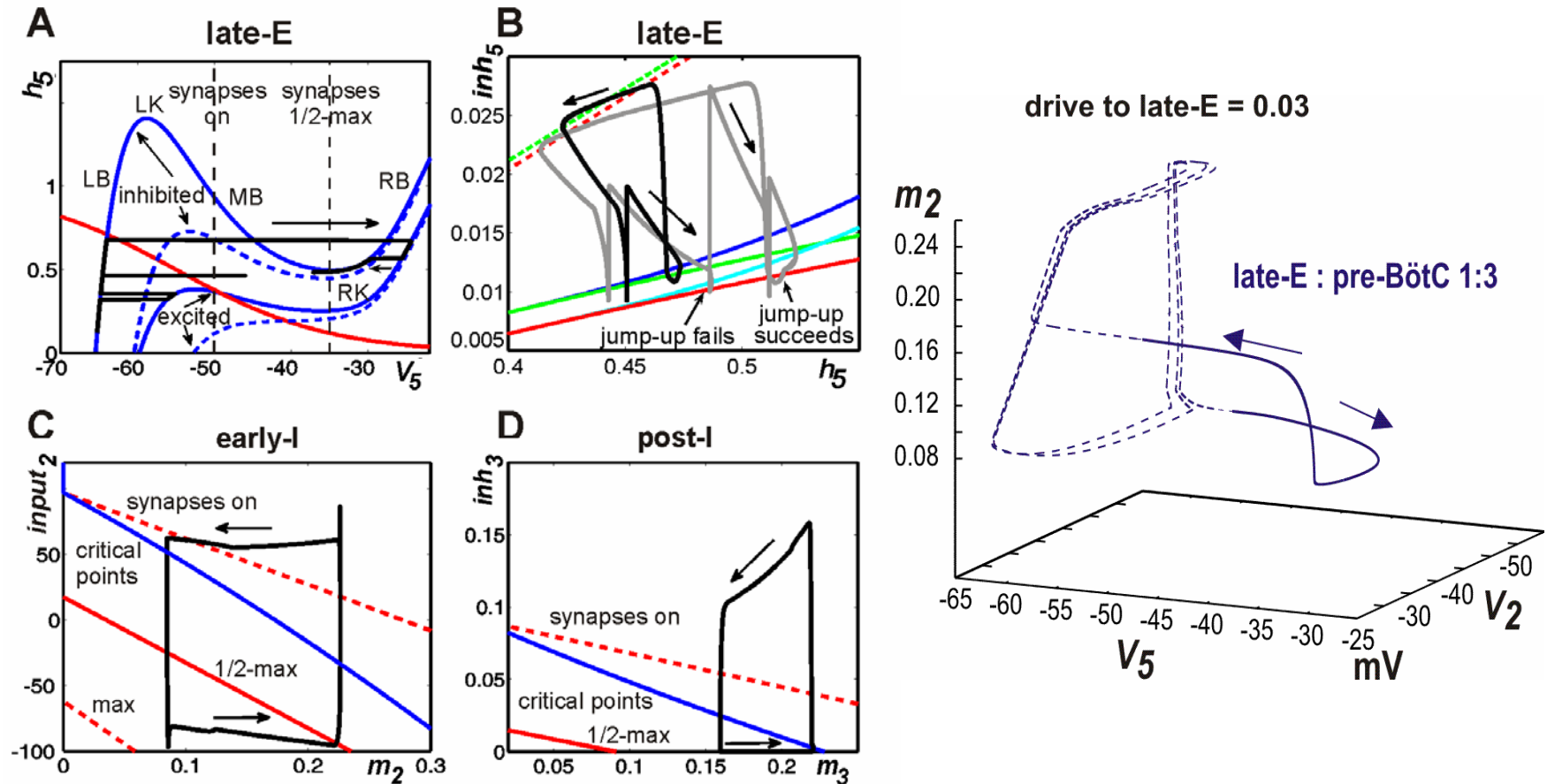
Simplified model



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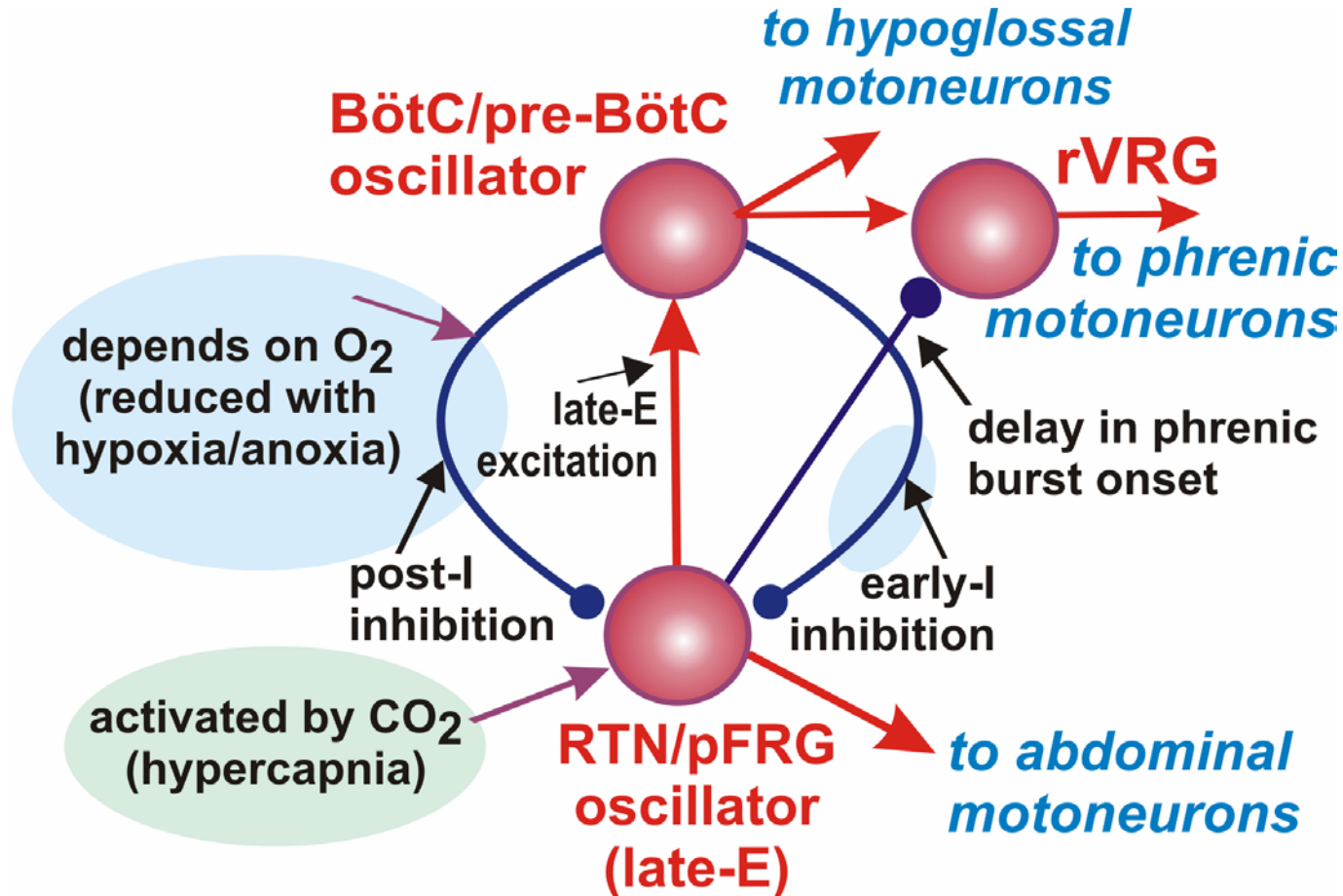
Multiscale Model of Neural Control of Breathing

Simplified model



Multiscale Model of Neural Control of Breathing

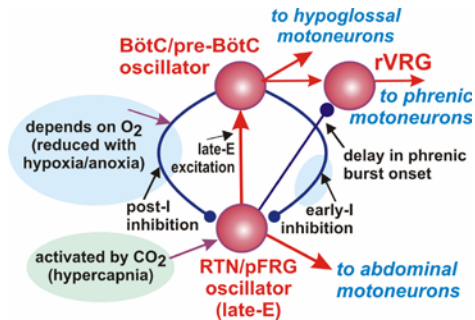
Proposed interactions between BötC/pre-BötC and pFRG/RTN



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Proposed interactions between BötC/pre-BötC and pFRG/RTN



Analysis of both the simplified and full models provided a plausible mechanistic explanation of the appearance and quantal acceleration of the abdominal late-expiratory activity with development of hypercapnia.

It is suggested that under normal metabolic conditions the RTN/pFRG oscillator is inhibited by both the post-I population of BötC during inspiration and early-inspiratory (early-I) population of pre-BötC during inspiration.

Therefore the late-E oscillations can be released by either a hypercapnia-evoked activation of chemosensitive RTN/pFRG neurons overcoming this inhibition or a hypoxia-dependent suppression of RTN/pFRG inhibition by BötC-pre-BötC circuits.