

LECTURE 16: 29 MAR 2005, CPGs in *TRITONIA* AND *LYMNAEA*

READINGS:

- (1) Getting, PA. Neural control of swimming in Tritonia. In: *Neural origins of rhythmic movements*. Cambridge: Cambridge Univ. Press, pp. 89-128, 1983.
- (2) Katz, PS et al. Dynamic neuromodulation of synaptic strength intrinsic to a central pattern generator circuit. *Nature* 367: 729-731, 1994.
- (3) Syed, NI et al. In vitro reconstruction of the respiratory central pattern generator of the mollusk *Lymnaea*. *Science* 250: 282-5, 1990.

(1) review Tritonia CPG function - cell, synaptic properties and connectivity of CPG neurons

(2) neuromodulation of CPG circuits via 5-HT inputs from DSI neurons (Katz 1994)

(AA) DSI neurons modulate C2 synapses (both to DSI and DFN neurons)

(A) C2 cells first excite then inhibit DSI

(B) In the absence of swim cycle, stimulation of DSI enhances the inhibitory phase of the synaptic input from C2 (neuromodulation)

(C) this suggests that ongoing swim patterns cause the self-inhibition of DSI

(i) this may be important given the episodic nature of the pattern (i.e., it is not to be maintained, just a transient activity for the animal)

(D) stimulation of DSI also enhances the C2 excitation of DFNs

(i) this is dynamic, and declines rapidly after DSI activity ceases

(ii) ensures that DFN activity is effective and strong during swim cycles

(iii) but declines rapidly once DSI activity declines, which signals the end of a swim episode

(E) DSI neurons are serotonergic (neuromodulator 5-HT)

(i) presence of 5-HT in the bath occludes the effect of DSI stimulation (they are not additive)

(3) *Lymnaea stagnalis* - the pulmonate mollusk

(A) unlike opisthobranch mollusks (e.g., *Aplysia* or *Tritonia*), pulmonates respire via lungs and pneumostome openings

(B) input 3 (I.P3.I) - expiratory

(C) visceral dorsal 4 (V.D4) - inspiratory

(D) right pedal giant dopamine cell (R.Pe.D1) - excites I.P3.I via post-inhibitory rebound (PIR)

(E) I.P3.I activity further excites R.Pe.D1 and inhibits V.D4.

(F) upon release from inhibition, V.D4 fires a cycle of action potentials

(G) V.D4 and R.Pe.D1 have inhibitory reciprocal connections

(H) no pair of cultured neurons could generate respiratory-like rhythm in *Lymnaea*

(I) co-culture of all three neurons (I.P3.I, V.D4, and R.Pe.D1) allowed patterns to emerge when depolarizing current was injected in R.Pe.D1

(i) R.Pe.D1 stimulation hyperpolarized I.P3.I neurons, which recover with PIR,

(ii) which further excites R.Pe.D1

(iii) combined activity of R.Pe.D1 + I.P3.I neurons causes PIR excitation of V.D4

(J) Here again we see the importance of reciprocal inhibition with delayed excitation

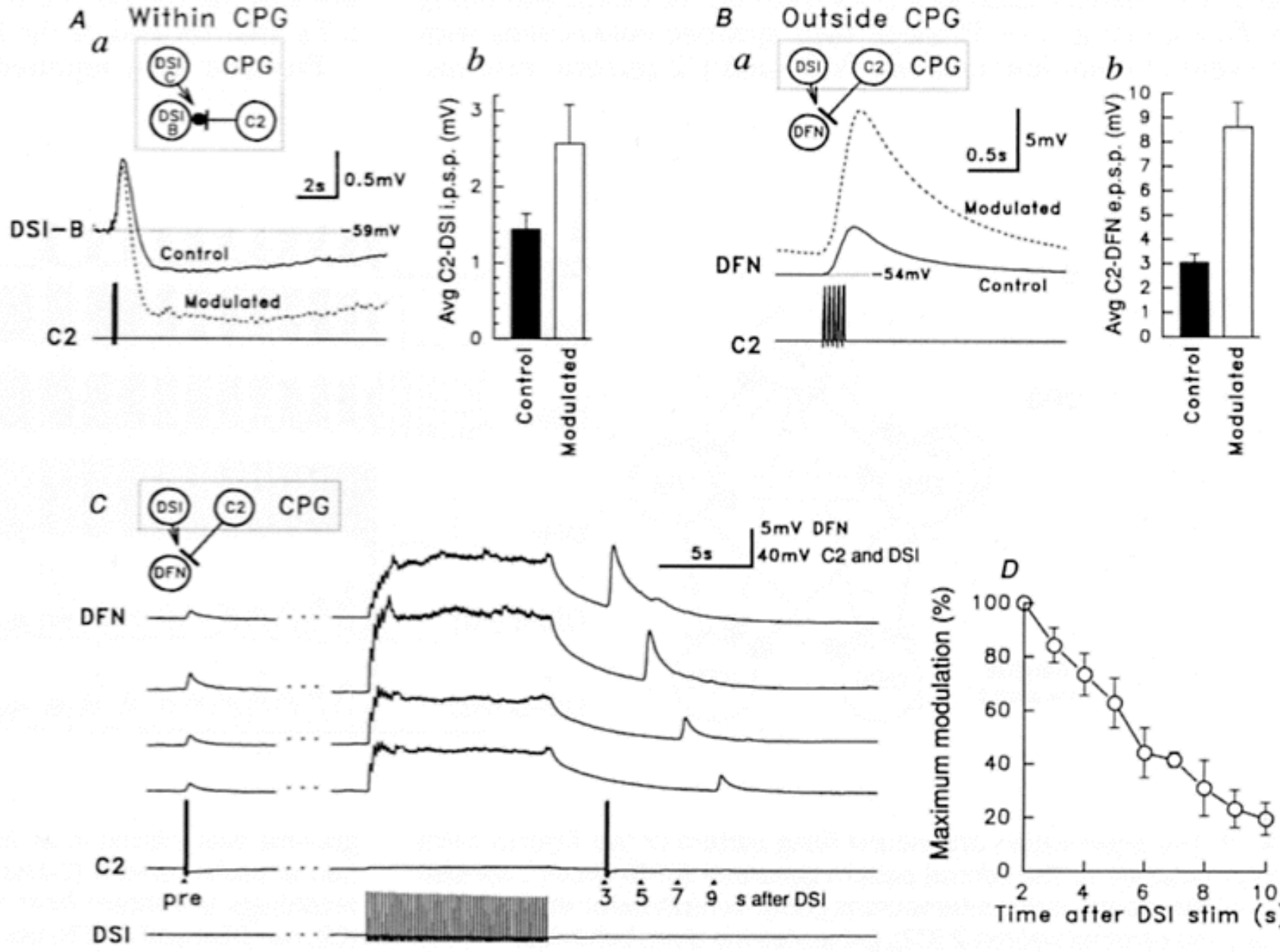
(K) replacing the R.Pe.D1 with other neurons of known transmitter types, which failed to restore respiratory rhythm

(i) 5-HT cells from left pedal ganglion

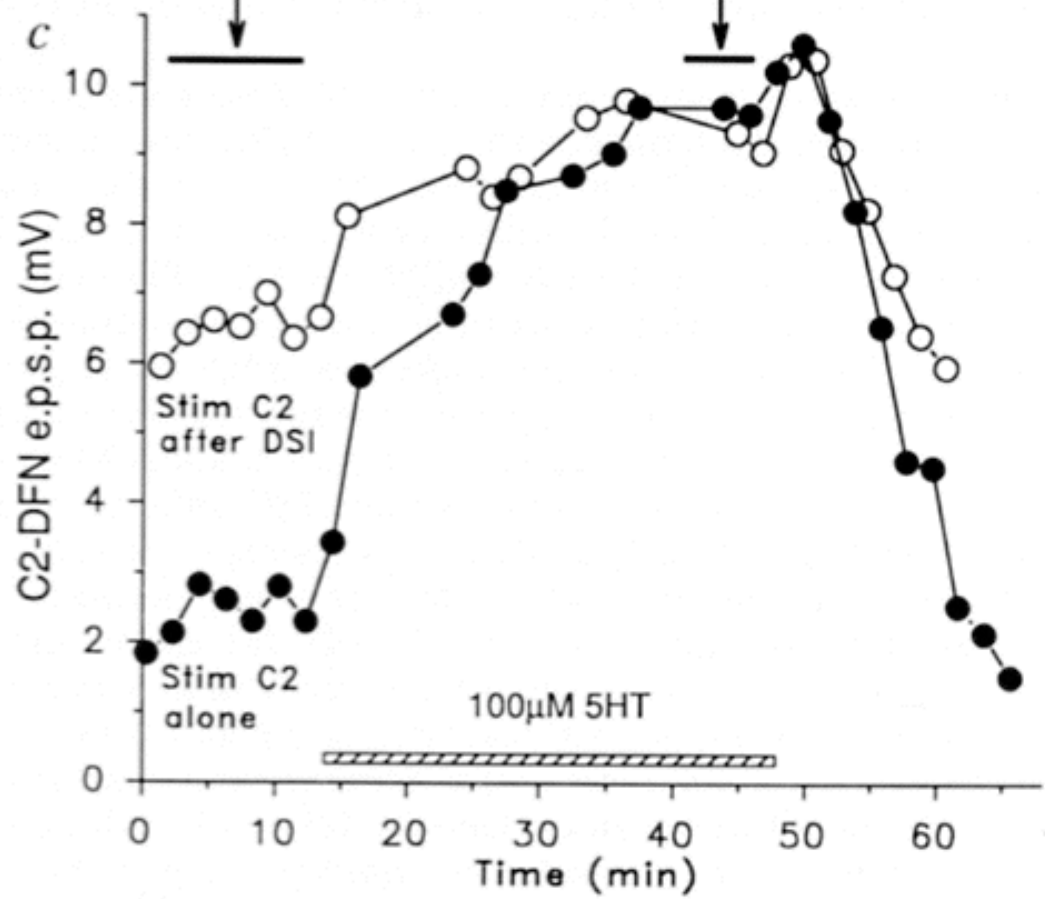
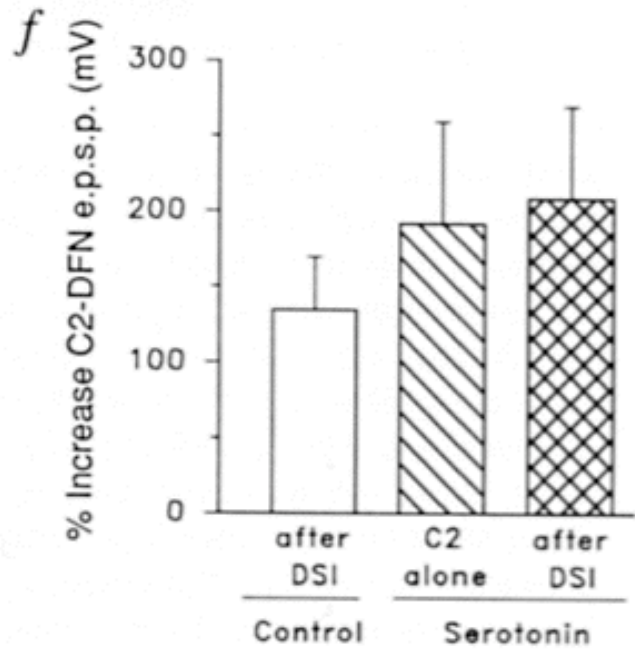
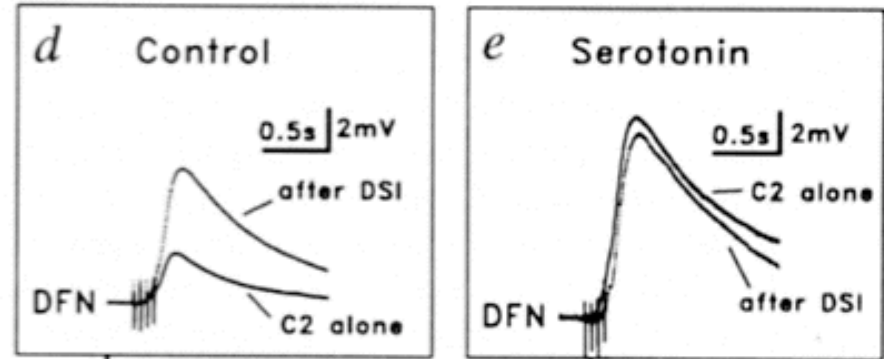
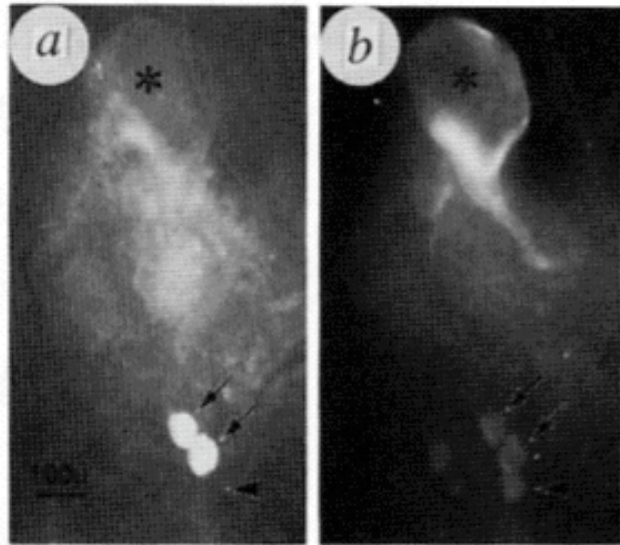
(ii) ACTH-like peptide of visceral ganglion

(L) phasic (but not tonic) application of DA elicited alternating respiratory-like oscillations

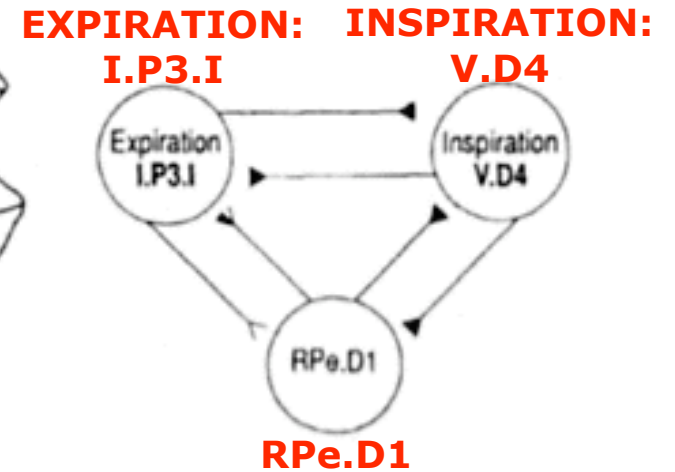
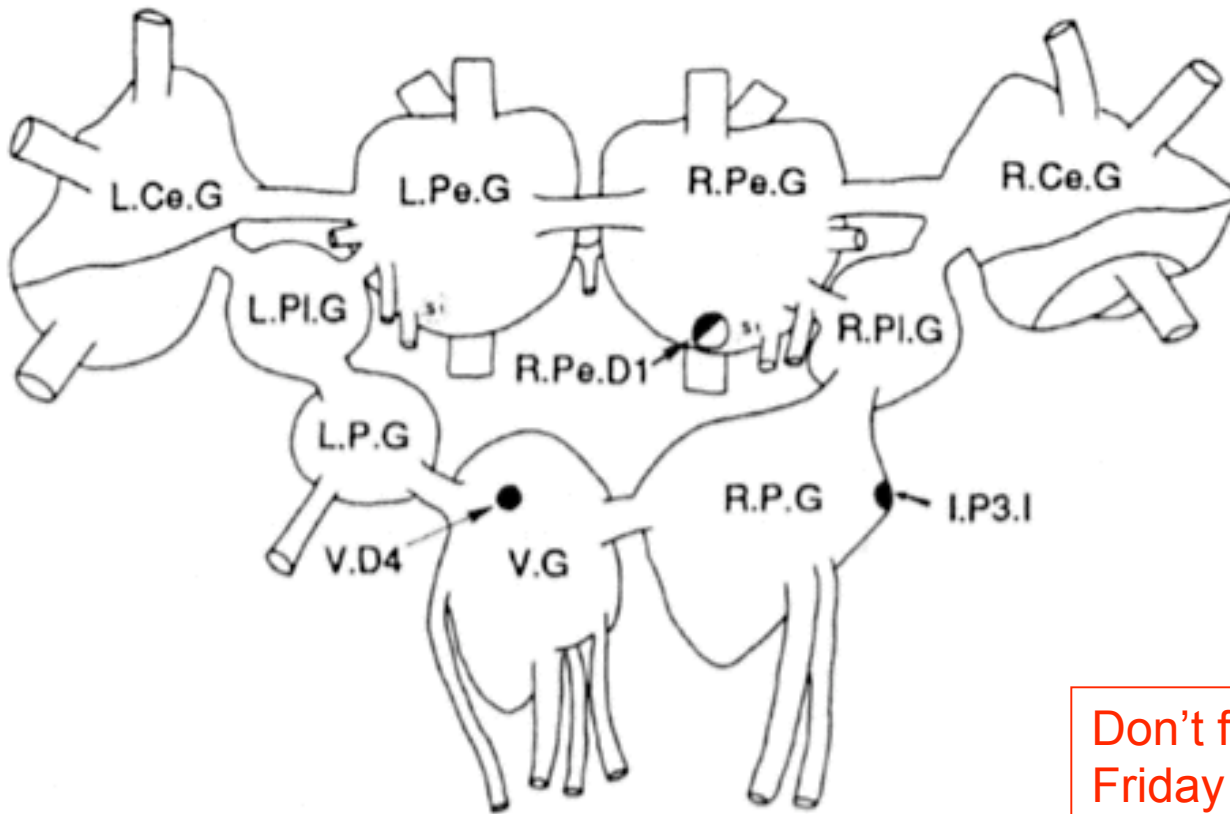
DSI INTERNEURONS MODULATE SYNAPTIC STRENGTH



DSI MODULATION IS SEROTONERGIC

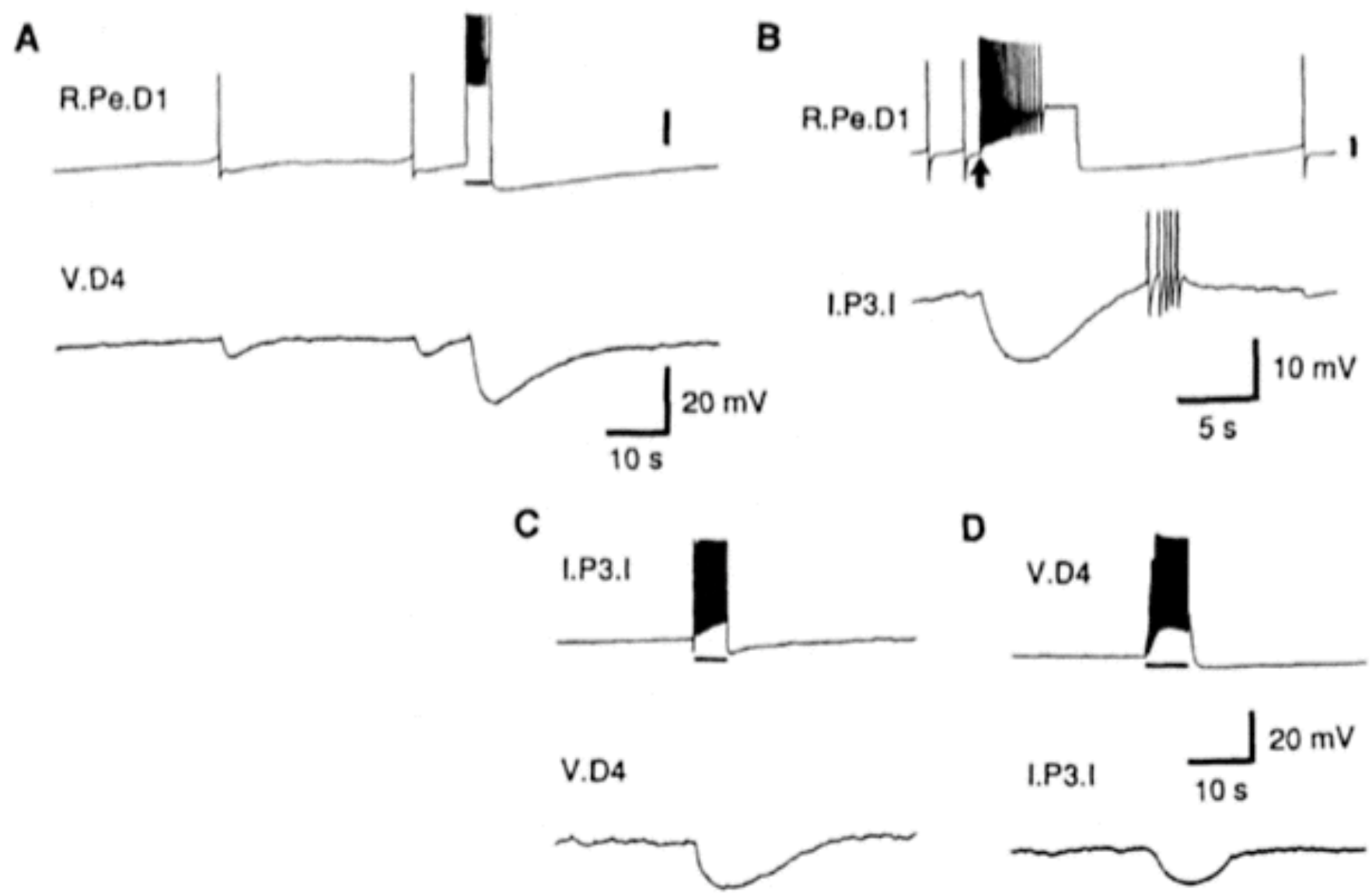


**CENTRAL GANGLIA OF
*LYMNEA STAGNALIS***

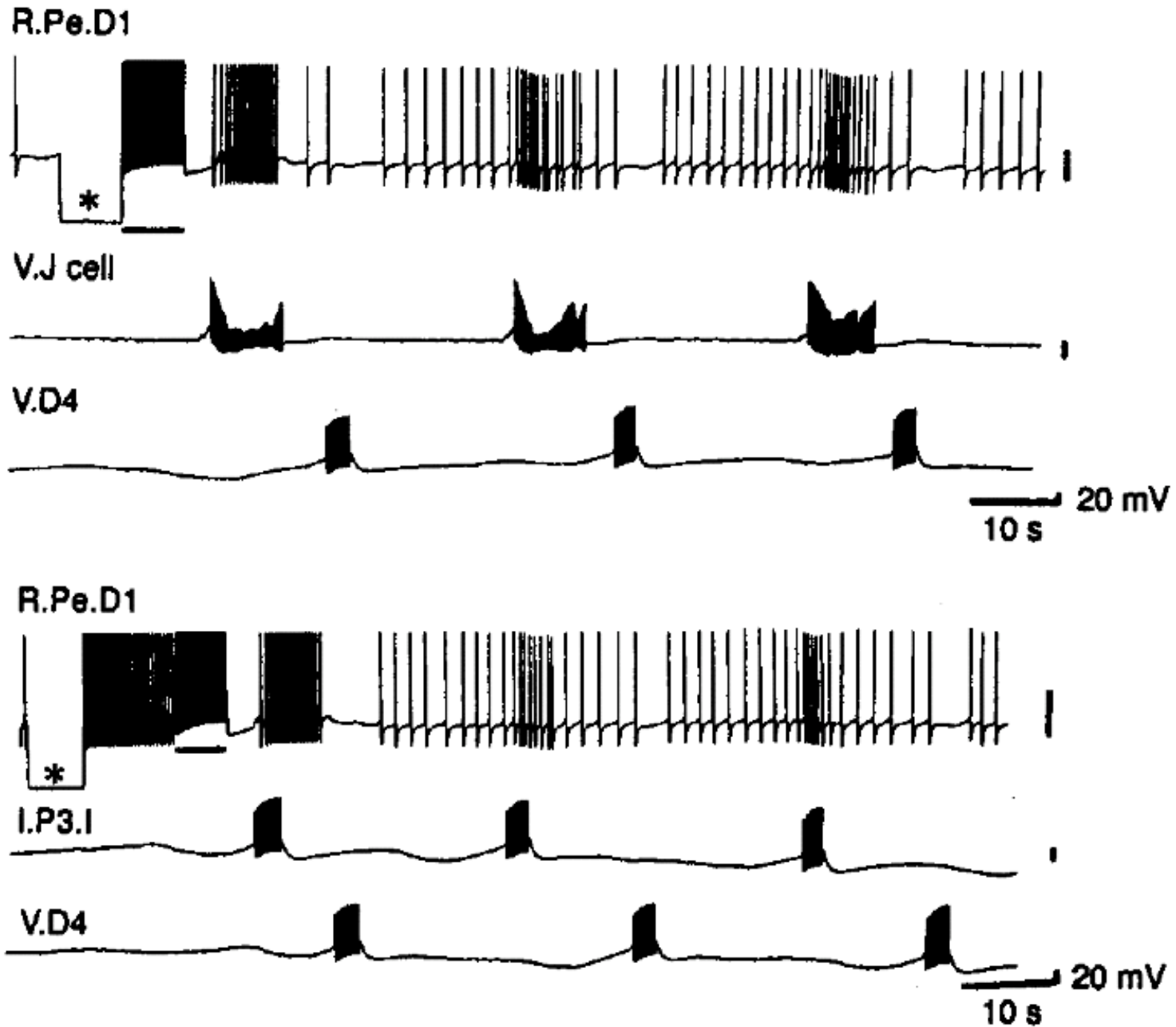


Don't forget Paul Katz Lecture
Friday April 15, 2005:
4 PM in Andrews Hall

IN VITRO RECORDINGS OF INTERACTING RESPIRATORY NEURONS



COMPARISON OF RECORDINGS *IN SITU* VERSUS *IN VITRO*



PULSATILE DOPAMINE ELICITS ALTERNATING BURSTS IN RESPIRATORY NEURONS *IN VITRO*

