# Poster presentation

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# System identification of the crab cardiac neuromuscular transform by a new method

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The neuromuscular transform (NMT) is the transform of the motor neuron spike train to the waveform of muscle contraction [1]. The NMT is often challenging to understand and model predictively. Each spike elicits an elementary contraction response, but the spike train can be of arbitrary temporal complexity and each elementary response is modified by the previous history of the activity. Furthermore, in slowly contracting muscles the elementary responses summate and fuse and cannot be seen in isolation. Ideally, we would like to characterize the NMT in terms of a small number of functions - the elementary response kernel and additional functions that describe the dependence on previous history - that will predict the response to any arbitrary spike train. Here, using a "decoding" method that we have developed [2], we have characterized in this manner the NMT of the neurogenic heart of the blue crab, Callinectes sapidus. As data, we recorded the heart muscle contractions elicited by random Poisson motor neuron spike trains (Fig. 1A). From these data, the method extracted three functions whose combination, according to a simple model (Fig. 1B), defined the contraction response: K, the elementary contraction kernel; H, a history kernel; and F, a static nonlinearity (Fig. 1C). H was negative immediately after each spike and then, peaking after several seconds, positive. To test the hypothesis that this shape reflected plasticity at the cardiac neuromuscular junctions that mediate the NMT, we recorded, in response to similar random spike trains, the underlying EJPs (not shown). When decoded,

these yielded essentially the same H, confirming its synaptic, and probably presynaptic, origin. The negative phase can be interpreted as depression, and the positive phase as facilitation, of potential transmitter release after each spike. The shape of *H* has interesting implications for the case of the endogenous spike pattern where the spikes are fired in bursts several seconds apart. Each spike inhibits the response to the subsequent spikes in the same burst, and the entire burst would produce little contraction were it not preceded, by an interval well matched to the positive phase of H, by the previous burst. Thus at the natural frequency of the cardiac rhythm each burst not only produces its own contraction, but enables that of the next burst. We are now examining how the functions of the NMT are altered by endogenous neuromodulators of the crab cardiac system [3].



#### **Figure I**

Decoding of the crab cardiac NMT. A: representative recording of contraction amplitude (muscle tension) of the heart muscle (blue curve) in response to a train of 295 random motor neuron spikes (blue dots along the baseline) generated by a Poisson process with a nominal rate of 2 Hz, and the contraction estimated by the decoding method (red curve). B: the decoding model. t = time;  $t_i$ ,  $t_j = \text{time}$  of spike *i*, *j*;  $R = \text{overall contraction response waveform; <math>K = \text{elementary contraction kernel}$ ; A = factor scaling the amplitude of K at each spike; H = elementary history kernel; F = static nonlinear function. C: K, H, and F decoded from the dataset in A.

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