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Microactuated Neural Probes to Compensate for Brain Micromotion

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Abstract— **One of the dominant failure modes of chronic neural implants is micromotion of the surrounding brain tissue relative to the implant leading to neuronal drift and shear injury. In this study, we have (a). Assessed the micromotion in the somatosensory cortex and (b). Designed, developed and tested a microactuated neural probe that can compensate for brain micromotion. We used a differential variable reluctance (DVRT) transducer in adult rats (n=8) to monitor micromotion in the somatosensory cortex. Electrostatic microactuators were fabricated using the SUMMiT (Sandia's Ultraplanar Multi-level MEMS Technology) process, a 5-layer polysilicon micromachining technology of the Sandia National labs, NM. In anesthetized rats, surface micromotion was observed to be in the order of 2-25 μm due to pressure changes during respiration and 1-3 μm due to vascular pulsatility. In addition there were long-term drifts in the order of 80 μm due to changes in the anesthetic level. The microactuated neural probe was capable of moving in steps of 1 μm with an aggregate translational capability in the order of several millimeters. In conclusion, there is significant micromotion in the surface of the somatosensory cortex that could lead to failure of chronic neural implants. Microactuated neural probes are capable of compensating for this micromotion.**

Keywords—**chronic implants, microelectrodes, neural prostheses**

I. Introduction

Failure of chronically implanted neural implants due to relative micromotion between the implant and the surrounding brain tissue is a problem not easily addressed. Sources of motion are readily identified as physiological (including cardiac and respiratory pulsations), behavioral (occurring from spontaneous head and/or trunk displacements) and/or mechanical disturbances of the lead wire translated to the electrode beneath the closed craniotomy. Several methods have been reported for reducing physiological and behavioral motion artifacts [1-15], and many of these are summarily characterized by Britt and Rossi [16]. These methods, while demonstrating some success in reducing the effects of pulsations on recordings, have the distinct disadvantages of involving invasive procedures and movement restrictions, essentially moving recording scenarios further from the ultimate goal of being able to record from neurons in their native state. Few investigators have reported quantitative data on the extent of brain motion [16, 17] and even less work has been done on the modeling of electrode motion relative the brain [18]. The significance of brain motion cannot be disputed when confronted with the number of researchers who have addressed this barrier to stable chronic recordings and more recently in brain mapping and imaging studies, yet measurement of motion of the brain is a daunting task.

With these concerns in mind we have developed a microactuated Neural probe chip *for continuous and independent steering* of implantable microelectrode arrays. Our long-term goal is to be able to automate the tracking of extracellular responses of individual neurons or a population of neurons *in vivo* for long periods of time.

II. Methodology

A microminiature differential variable reluctance transducer (DVRT) was used to track micromotion. The core of the transducer weighs less than 25 mg (Microstrain Inc., Williston, VA). The demodulated voltage output of the DVRT was linearly correlated to displacement (4.7 mV/μm). Adult Wistar rats (n=5) were anesthetized with a cocktail (100 mg/ml ketamine, 20 mg/ml xylazine, 10 mg/ml acepromazine mixed with sterile water). The animals were intubated and EKG and end-tidal CO_2 were continuously monitored. The animals were placed in a stereotactic frame and a craniotomy was done centered around a point in the somatosensory cortex (3mm lateral and 2 mm posterior to the bregma according to the rat atlas [19]). Two different craniotomy sizes were studied in different animals. The DVRT sensors were serially placed at several positions in the somatosensory cortex to evaluate micromotion. Power spectral and spectral coherence analyses were performed on the demodulated voltage output from the DVRT, the EKG and the respiratory rate to isolate components of micromotion that correspond to vascular pulsatility and respiration respectively.

Electrostatic microactuators and microelectrodes were fabricated using the SUMMiT (Sandia's Ultraplanar Multi-level MEMS Technology) process. The polysilicon microelectrodes have been shown to record extra-cellular action potentials from single neurons in-vivo [20].

III. Results

A. Micromotion in the somatosensory cortex

Raw demodulated data from the DVRT is shown in Figs. 1 and 2. Micromotion due to pressure changes in the brain corresponding to the respiratory rhythm was in the order of 2-25 μm. In Fig. 1, a typical micromotion of

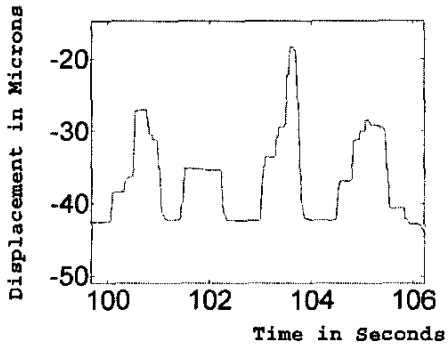

Fig. 1. Micromotion in the somatosensory cortex caused by respiratory rhythms measured using a DVRT transducer,

approximately 25 μm is evident in the somatosensory cortex. Further, long term aperiodic drifts of the brain surface lasting several minutes were also observed as shown in Fig. 2. These drifts were approximately 80 μm. Micromotion due to pressure changes in the vasculature was found to be in the order of 1-3 μm. A high degree of spectral coherence was observed between the displacement waveform and the EKG signal at the fundamental frequency of the EKG signal (approximately 2-4 Hz) corresponding to the heart rate. Similarly, there was a high degree of spectral coherence between the displacement waveform and the end-tidal CO_2 waveform at the fundamental frequency of approximately 1-2 Hz corresponding to the respiratory rate.

B. Electrostatic Microactuator

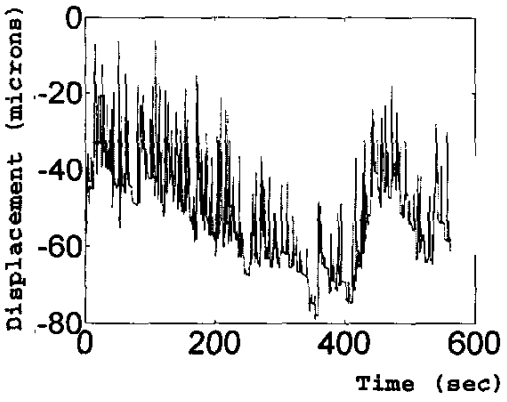

Fig. 2. Long-term (over several minutes) drifts in the order of 60-80 μm observed in the somatosensory cortex.

A picture of the polysilicon microprobe extended from the Neural probe chip is shown in Fig. 3. A photograph of the Neural probe chip is shown in Fig. 4. With a gear ratio of 144:1 between the microactuators and the microelectrodes, the polysilicon microelectrodes can be moved in steps of 1 μm or less with a total translational movement of several millimeters. The rate of actuation depends on the frequency of the excitation waveforms, which is in the order of hundreds of kilohertz enabling extremely fast tracking capabilities.

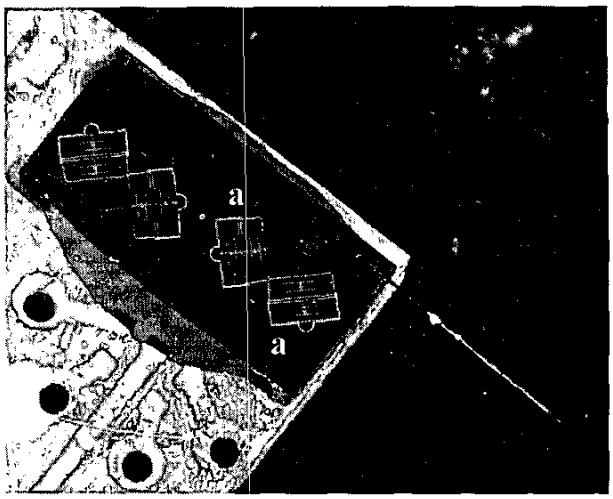

Fig. 3. A picture of the polysilicon microelectrode extended out of the edge of the Neural probe chip. The microactuators are labeled 'a'.

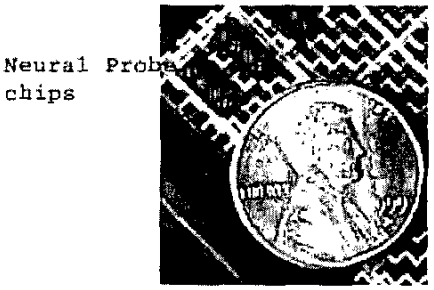

Fig. 4. Two Neural probe chips shown alongside a penny for size comparison.

IV. Discussion and Conclusion

There is significant periodic micromotion in the somatosensory cortex due to respiration and vascular pulsatility. In addition, there are aperiodic drifts in the micromotion on the order of 80 μm. Micromotion in the order of tens of microns would be of serious concern in applications where we are trying to record extra-cellular action potentials from single neurons chronically. However, micromotion even in the order of several microns would be of concern during intra-cellular recording applications in-vivo. The magnitude of micromotion observed in this study confirms the role of micromotion as a dominant failure mode in chronic neural implants. The data reported here were obtained from anesthetized animals. Future

experiments will attempt to quantify the micromotion due to behavior.

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REFERENCES

[1] E. R. Kandel, W. A. Spencer, and F. J. Brinley, "Electrophysiology of Hippocampal Neurons .1. Sequential Invasion and Synaptic Organization," *Journal of Neurophysiology*, vol. 24, pp. 225-&, 1961.

[2] M. M. Todd, S. M. Toutant, and H. M. Shapiro, "The Effects of High-Frequency Positive-Pressure Ventilation on Intra-Cranical Pressure and Brain Surface Movement in Cats," *Anesthesiology*, vol. 54, pp. 496-504, 1981.

[3] R. Britt and A. Starr, "Synaptic Events and Discharge Patterns of Cochlear Nucleus Cells .2. Frequency-Modulated Tones," *Journal of Neurophysiology*, vol. 39, pp. 179-194, 1976.

[4] R. H. Britt, "Intracellular Study of Synaptic Events Related to Phase-Locking Responses of Cat Cochlear Nucleus Cells to Low-Frequency Tones," *Brain Research*, vol. 112, pp. 313-327, 1976.

[5] P. W. Davies, "Chamber for Microelectrode Studies in the Cerebral Cortex," *Science*, vol. 124, pp. 179-180, 1956.

[6] V. B. Mountcastle, P. W. Davies, and A. L. Berman, "Response Properties of Neurons of Cats Somatic Sensory Cortex to Peripheral Stimuli," *Journal of Neurophysiology*, vol. 20, pp. 374-407, 1957.

[7] D. H. Hubel, "Single Unit Activity in Striate Cortex of Unrestrained Cats," *Journal of Physiology-London*, vol. 147, pp. 226-&, 1959.

[8] G. W. Sypert and A. A. Ward, "Hyperexcitable Neuron - Microelectrode Studies of Chronic Epileptic Focus in Intact Awake Monkey," *Experimental Neurology*, vol. 19, pp. 104-&, 1967.

[9] A. A. Ward and L. B. Thomas, "The Electrical Activity of Single Units in the Cerebral Cortex of Man," *Electroencephalography and Clinical Neurophysiology*, vol. 7, pp. 135-136, 1955.

[10] B. D. Burns and J. G. Robson, "Weightless Micro-Electrodes for Recording Extracellular Unit Action Potentials from the Central Nervous System," *Nature*, vol. 186, pp. 246-247, 1960.

[11] F. Puletti and Blomquis.Aj, "A Technique for Recording Single Neuron Potentials in Human Spinal Cord - Technical Note," *Journal of Neurosurgery*, vol. 26, pp. 96-&, 1967.

[12] V. E. Amassian and D. Giblin, "Periodic Components in Steady-State Activity of Cuneate Neurons and Their Possible Role in Sensory Coding," *Journal of Physiology-London*, vol. 243, pp. 353-385, 1974.

[13] J. Massion, P. Angaut, and Albefess.D, "Etude Des Facteurs Impliques Dans Laccroissement Des Activites Corticales Observe Apres Cerebellectomie," *Electroencephalography and Clinical Neurophysiology*, vol. 18, pp. 455-&, 1965.

[14] K. Krnjevic and Lisiewic.A, "Injections of Calcium-Ions into Spinal Motoneurones," *Journal of Physiology-London*, vol. 225, pp. 363-&, 1972.

[15] J. C. Eccles, P. Scheid, and H. Taborikova, "Responses of Red Nucleus Neurons to Antidromic and Synaptic Activation," *Journal of Neurophysiology*, vol. 38, pp. 947-964, 1975.

[16] R. H. Britt and G. T. Rossi, "Quantitative analysis of methods for reducing physiological brain pulsations," *J Neurosci Methods*, vol. 6, pp. 219-29., 1982.

[17] M. S. Fee, "Active stabilization of electrodes for intracellular recording in awake behaving animals," *Neuron*, vol. 27, pp. 461-8., 2000.

[18] S. R. Goldstein and M. Salcman, "Mechanical factors in the design of chronic recording intracortical microelectrodes," *IEEE Trans Biomed Eng*, vol. 20, pp. 260-9., 1973.

[19] G. Paxinos and C. Watson, *The Rat Brain in Stereotaxic Coordinates*, 2nd ed. San Diego, CA: Academic Press Inc., 1986.

[20] J. Muthuswamy, D. Salas, and M. Okandan, "A chronic micropositioning system for Neurophysiology," presented at Joint Meeting of the IEEE Engineering in Medicine and Biology Society And the Biomedical Engineering Society, Houston, TX, 2002.