tivity. The activity in the middle constrictor increased with increasing depth. At a very deep level a decreased activity was observed; but even the last breath in deep anesthesia showed electromyographic activity. **Conclusions:** Because of a practically unaltered time sequence of activation it is assumed that unaltered central pathways were available for the individual muscles when anesthesia was deepened. Because no parallelism was observed in the responses of these motor functions to changes of depth of anesthesia, it can be tentatively concluded that each depends on a different, though related, central regulatory mechanism. [This work was supported in part by a grant from the Burroughs Wellcome & Co. Inc. The Fluothane used in this study was provided by Ayerst Laboratories.]

**Effect of Reserpine on Cardiac Function During Thiopental-Cyclopropane Anesthesia.**

The direct, depressant action of cyclopropane on the heart is not overtly demonstrated in the intact organism because, as suggested by Price and others, this agent causes the release of norepinephrine which has a positive inotropic effect on the myocardium. Reserpine depletes cardiac tissue of its norepinephrine content and Blinks and Waud have demonstrated that pretreatment with reserpine effectively blocks the positive inotropic effect normally caused by stimulation of the cardiac accelerator nerves. It might therefore be expected that reserpine pretreatment would unmask the direct depressant effects of cyclopropane on the heart. Clinical evidence in support of this possibility has emerged in case reports describing cardiovascular collapse following anesthesia in serpinized patients. A study of the effect of reserpine pretreatment on cardiac function during anesthesia induced with thiopental and maintained with cyclopropane is currently being carried out in dogs.

**Methods:** Cardiac function was evaluated by measuring the cardiac output (dye-dilution) and the rate of rise of left ventricular pressure during the isometric phase of systole. The latter measurement was obtained by differentiating the electrical analog of left ventricular pressure and is, according to Rushmer, an index of myocardial "contractility." These two parameters plus heart rate and mean arterial blood pressure (MABP) are measured in the unanesthetized animal and compared with similar measurements made following 45 minutes of thiopental-cyclopropane anesthesia. Experiments on each animal are done before reserpine and following each of three successive reserpine treatments which consist of: (1) reserpine 10 \( \mu g/kg/day \times 10 \), (2) reserpine 20 \( \mu g/kg/day \times 5 \), and (3) reserpine 100 \( \mu g/kg/day \times 1 \). Cyclopropane 25 per cent in O\(_2\) is administered by nonrebreathing technique such that equal end-expired cyclopropane concentrations are maintained in the pre- and post-reserpine experiments. Respirations are controlled and the P\(_{CO_2}\) is held within the normal range. **Results:** To date, five dogs have been carried satisfactorily through the study. Reserpine in all doses caused a decrease in heart rate, MABP and left ventricular "contractility" in the unanesthetized animals. Cardiovascular collapse following anesthesia did not occur in any experiment, even after the high dose of reserpine. Changes caused by anesthesia are expressed as the average of the percentage changes from the unanesthetized values for each experiment and are as follows: Heart rate and MABP increased 30 per cent and 5 per cent respectively, while "contractility" decreased about 30 per cent, both before and after reserpine; *i.e.*, reserpine exerted little or no effect upon the per cent change caused by anesthesia in these parameters. Following anesthesia before reserpine, cardiac output fell an average of 13 per cent below the awake value. After reserpine, cardiac output was depressed more severely by anesthesia. In those animals who had received the high dose of reserpine, the average decrease below the awake value was 37 per cent. Whether this decrease is significantly greater than the decrease before reserpine is not known at present.

**Conclusions:** The findings indicate that reserpine depression of the normal dog does not severely inhibit his ability to compensate for the cardiovascular depressant effects of thiopental-cyclopropane anesthesia.