ograms is possible in large-scale studies.

It must be emphasized that these ratios depend upon approximations of complex functions in the EEG. Theoretically at least, some of the estimates could be obtained as well from power spectrum analysis, and in many cases the power spectrum may be a useful adjunct in determining applicability or interpretation. There are probably some circumstances where the ratios can be applied only with considerable caution; for example when dominance is not clearly monorhythmic. For these reasons, further work is needed to provide information on the limits of their applicability, provide stronger validation, and perhaps to obtain the terms with greater mathematical elegance. Meanwhile, I have shown (*5*) that these ratios, unlike single absolute parameters such as frequency or amplitude, do distinguish degrees of arousal in human subjects.

In consideration of the importance placed upon desynchronization in activation theory (*6*), it is apparent that the method proposed in this paper should be of use. In discussing frequency analysis, Knott (*7*) said, "an instrument designed to complement *visual* analysis should not lead just to further visual analysis." Since the remark also applies to correlation analysis, the ratios are offered for their possible use as data reduction techniques.

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8. Supported by grant MH-02553 from the USPHS. I thank James B. Jennings who ran the correlograms and critically read the manuscript.

8 June 1964

Reversible Cold Block of the Specialized Cardiac Tissues of the Unanesthetized Dog

Abstract. *The sinoatrial node and the bundle of His in intact, unanesthetized dogs were subjected to local cooling by means of surgically implanted devices. Impulse formation in the sinoatrial node could thus be suppressed at will and the heart rate regulated within a physiological range by an electrode driving the atrium. Reversible blockage of atrioventricular conduction was easily induced on cooling the region of the bundle of His.*

Local cooling of the specialized conduction system of the heart results in reversible suppression of impulse formation and impulse transmission (*1*). Hypothermia decreases the spontaneous activity of pacemakers (*2*), decreases conduction velocity in the atria and ventricles of mammalian hearts, and slows impulse propagation at the junction of the atrium with the atrioventricular node (*3*). A similar effect of local cooling has been noted with reference to impulse transmission in the central nervous system (*4*). The purpose of the study reported herein was to control heart rate and atrioventricular conduction in an intact unanesthetized animal. Local cooling of the sinoatrial node and of the bundle of His in the intact dog resulted in inhibition of impulse formation at the sinoatrial node and in varying degrees of blockage of atrioventricular conduction.

Two adult mongrel dogs were anesthetized with 30 mg of pentobarbital per kilogram of body weight, were intubated and kept under conditions of controlled ventilation. The chest was entered through the right fourth intercostal space and the heart exposed. In one animal a 30-cm length of fine silver tubing (inner and outer diameters, 0.165 cm and 0.178 cm, respectively) was wound into a tight flat coil about 2 cm in diameter (Fig. 1) and sutured to the right atrium over the region of the sinoatrial node. Polyethylene tubing (No. 200) was tightly fitted to each of the free ends of the coil. A pacemaker electrode was sutured to the right atrial appendage. The pericardium was closed loosely. The two lengths of polyethylene tubing, together with the electrode cable, were brought out of the chest at a point between the scapulae.

In a second dog, right atriotomy was performed under inflow occlusion. During this procedure, a 4-cm length of the silver tubing, bent into a "U," was sutured to the interatrial septum in the region of the bundle of His. One leg of the "U" was placed parallel to, and about 0.6 cm above, the septal leaflet of the tricuspid valve; the open end of the "U" was directed toward the coronary sinus (Fig. 1). Polyethylene tubing (No. 200), tightly fitted to each leg of the "U," extended outside the chest which was closed as before.

The dogs were permitted to recover from surgery for 5 days before records were taken. During each experiment the animal was sedated with morphine sulfate, 1 to 2 mg/kg. Electrocardiograms were recorded on a Sanborn polyviso. The sinoatrial node and the bundle of His were cooled by passing a mixture of 95 percent alcohol and carbon dioxide ice through the silver tubing. One end of the polyethylene tubing was attached to a suction pump while the other end was immersed in a reservoir of the cooling mixture. Passage of the cooling mixture (initially at −10° to −20°C) through the coil inhibited impulse formation at the sinoatrial node. After suppression of the sinus pacemaker, the heart was paced at various rates by means of the electrode attached to the right atrial appendage. The stimulus intensity was between 5 and 15 v and the stimulus duration 5 msec. To suppress atrioventricular conduction, the alcohol-carbon dioxide mixture, cooled to temperatures as low as −45°C, was drawn through the silver tubing. The temperature in the coil and "U" tube was varied by controlling the proportion of alcohol and carbon dioxide ice in the cooling mixture reser-

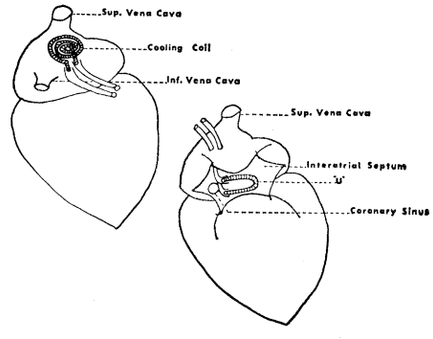

Fig. 1. Right aspect of heart showing cooling coil, sutured over the sinoatrial region (left) and bent into a "U" on the interatrial septum (right).

voir. The rate of flow through the coil and "U" was kept constant in each experiment. Temperatures at the outflow or in the tissue were not monitored directly.

The electrocardiogram in Fig. 2 shows that cooling of the sinoatrial node resulted in slowing of the sinus pacemaker, followed by the absence of P waves. Upon suppression of the sinus pacemaker a lower supraventricular pacemaker with a slower intrinsic rate became dominant. The heart was then paced by means of the electrode attached to the right atrial appendage at rates varying between 90 per minute and 64 per minute (Fig. 2). About 1 minute after cessation of flow of the cooling mixture through the coil, P waves reappeared in the electrocardiogram and the rate of the sinus pacemaker returned to the control value. This experiment was carried out several times on the same animal over an 8-week period. The sinoatrial node was cooled continuously for periods up to one half hour. No abnormal P wave changes were observed after cooling.

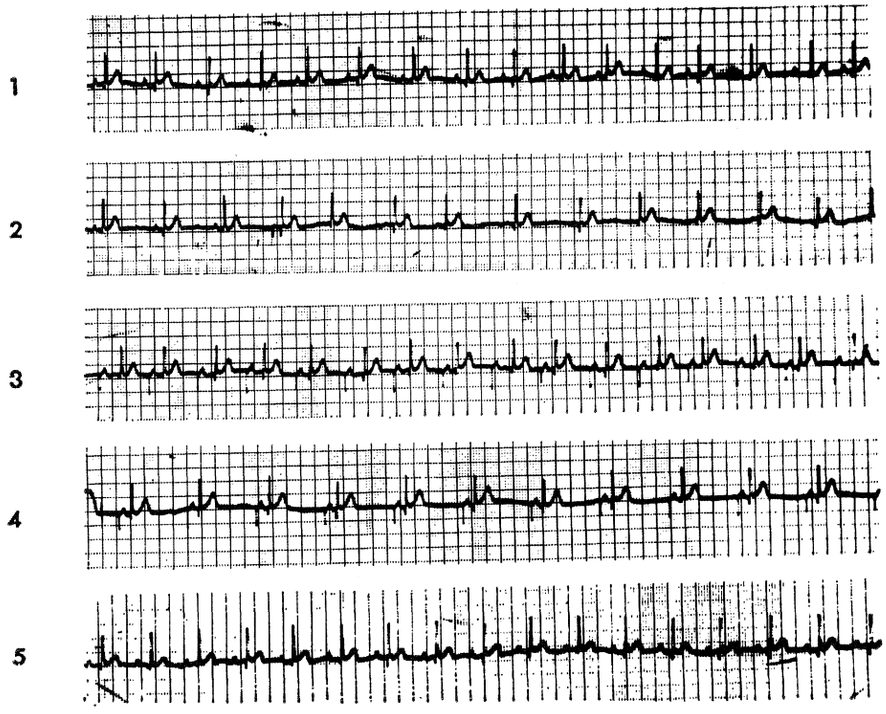

Fig. 2. Electrocardiogram obtained from lead 2 during cooling of the sinoatrial node. *1*, Normal sinus rhythm; *2*, suppression of P wave during cooling; *3*, atrial pacing at 90 per minute during sinus node suppression; *4*, atrial pacing at 64 per minute during sinus node suppression; *5*, reversion to normal sinus rhythm after cessation of cooling.

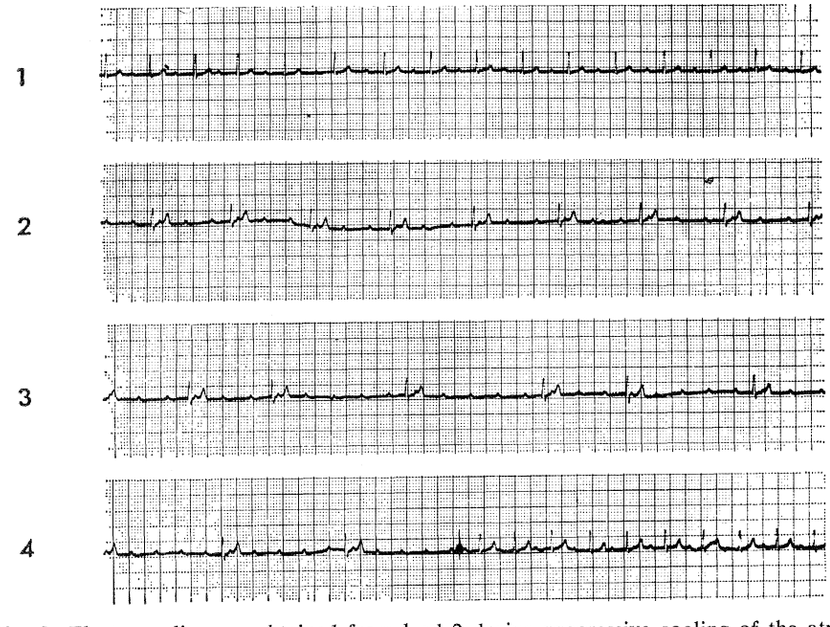

Fig. 3. Electrocardiogram obtained from lead 2 during progressive cooling of the atrioventricular nodal region. *1*, Normal sinus rhythm with a prolonged interval between the P and R waves; *2*, 3 : 1 atrioventricular block; *3*, 3 : 1 to 5 : 1 atrioventricular block; *4*, 5 : 1 atrioventricular block, and reversion to normal sinus rhythm after cessation of cooling.

Prior to cooling of the bundle of His, a sinus rhythm with a prolonged interval between the P and R waves was present (Fig. 3). The prolonged P–R interval probably was a result of surgical trauma to the atrioventricular node. Figure 2 shows that progressive cooling of the loop resulted in increasing degrees of inhibition of atrioventricular conduction. When the cooling mixture in the reservoir was at −45°C, a stable 5 : 1 block (where only every fifth atrial beat was conducted to the ventricle) was maintained. Almost immediately after terminating the flow of cooling mixture through the loop, atrioventricular conduction reverted to the state prior to cooling. This experiment was carried out several times on the same animal over a 4-week period. The atrioventricular node was cooled continuously for periods up to one half hour. There were no alterations in the interval between P and R waves after cooling.

Thus, our results show that in the unanesthetized animal, after suppression of the sinus pacemaker by cooling, heart rate can be regulated within the physiologic range by direct atrial stimulation; localized areas of the specialized conduction system can be blocked reversibly. This experimental method could be used in further studies of heart rate, arrhythmias, and conduction disturbances in the intact animal.

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5. One of us (B.F.H.) is the recipient of a career scientist award of the Health Research Council of the City of New York under contract No. I-203.

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Sleep Pattern of Tooth-Grinding: Its Relationship to Dreaming

Abstract. *Simultaneous recordings of brain waves, eye movements, and masticatory muscle potentials throughout the night demonstrate a temporal relationship between episodes of bruxism (nocturnal grinding of the teeth) and periods of rapid eye movements indicative of dreaming.*

Bruxism is the forceful contraction of some of the muscles of mastication occurring during sleep and frequently accompanied by loud grinding or clicking noises. Bruxism affects people of all ages; the incidence in normal populations is unknown. In completing a health questionnaire, 10 percent of the students entering the University of Chicago gave responses indicating possible nocturnal tooth-grinding. Bruxism has deleterious effects on the teeth and supporting structures and its noise often disturbs the sleep of roommates.

We have studied the occurrence of bruxism during the sleep cycle. The subjects we used slept in a laboratory where electroencephalograms (EEG), eye movements, and electromyograms (EMG) from the masseter muscles could be monitored continuously each night. The EEG and eye movements were monitored by well-known techniques which permit discrimination of the four major stages of sleep (*1*) The EMG recording was obtained from two surface electrodes over the masseter muscle on a vertical line between the zygomatic arch and the inferior border of the mandible. A sensitive microphone above the subject's head carried the sounds of contacting teeth to the experimenter who noted their occurrence on the polygraph tracing.

Twelve subjects in whom bruxism was known to occur were observed for 1 or 2 nights each for a total of 14 nights. To define bruxism, we used an EMG recording of at least four times the amplitude of the recording of the basal muscle tone, recognizing that this definition might exclude minor masseter contractions. No masseter contractions which occurred during gross body movements were scored. Sounds of grinding were clearly unreliable indicators of bruxism because massive masseter contractions indicated that noiseless but very strenuous tooth-clenching could occur. The results of these studies are described herein.

Masseter contractions of any amplitude were rare during sleep characterized by high amplitude, slow delta EEG activity (sleep stages III and IV) in agreement with Takahama's report (*2*). Masseter contractions during these stages were usually a concomitant of other body movements and preceded by less than 2 minutes the onset of stage II or of stage I. Masseter contractions were infrequent in stage II of sleep, but not as infrequent as in stages III and IV.

Bruxism was most frequently associated with REM periods (emergent stage I with rapid eye movements) (Fig. 1). Because REM periods are associated with dreaming (*1*), a temporal relationship between bruxism and dreaming is indicated. The difference in the incidence of bruxism in REM and non-REM stages was significant at less than the .001 level (matched t test). Masseter contractions occurred at the rate of 20.9 per hour (averaged across nights) during REM periods as compared to 5.3 per hour during non-REM periods. Pilot experiments with four normal subjects showed a similar relationship between sleep stage and masseter contractions, indicating that symptomatic bruxism may represent an exaggeration of a normal phenomenon.

Heightened EMG activity from the masseter leads was observed in conjunction with virtually all large movements of the trunk or limbs. To decrease the possibility that this EMG activity might have represented a spread of electrical activity from other muscle groups, an additional measure was used. Two metal plates were fitted onto corresponding upper and lower molar teeth and arranged in a circuit such that a signal was registered on the polygraph tracing whenever the plates came into contact. Because, at rest, the lower teeth are normally separated from the upper teeth by a space of a few millimeters (*3*), a meeting of the upper and lower teeth could serve as another index of masseter contraction. Virtually all movements of the limbs and trunk were found to be accompanied by a meeting of the upper and lower teeth as well as by EMG signs of masseter contraction.

In the rapid phase of sleep of the cat (corresponding to the REM period in humans) Jouvet noted occasional sudden movements of the jaws and vibrissae (*4*) in addition to the eye movements. He also showed that the occurrence of REM periods demands the integrity of the nucleus reticularis pontis caudalis which is situated in the reticular formation of the brain stem

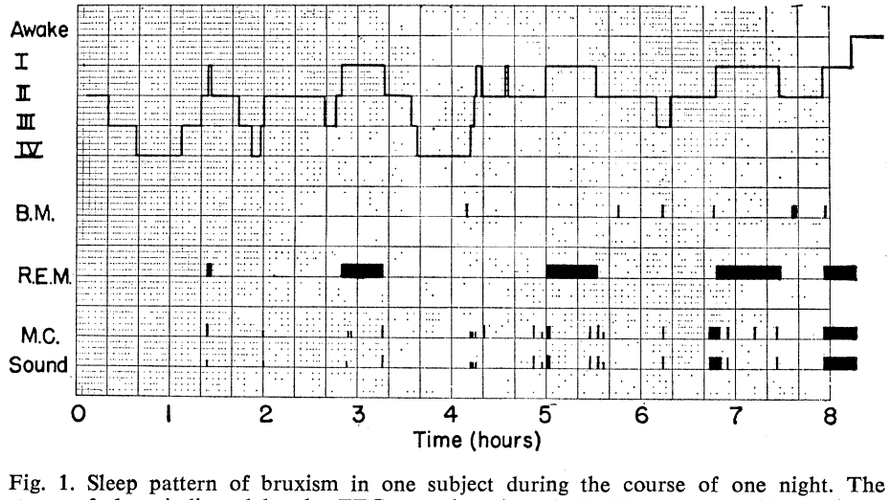

Fig. 1. Sleep pattern of bruxism in one subject during the course of one night. The stages of sleep indicated by the EEG are plotted as I, II, III and IV. Abbreviations: B.M. body movements; R.E.M., periods of rapid eye movements; M.C., masseter contractions; "sound" indicates sounds of bruxism.