MECHANISM AND CONTROL OF CENTRALLY INDUCED CARDIAC IRREGULARITIES DURING HYPOTHERMIA

PART I. CLINICAL OBSERVATIONS

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CENTRAL influences on cardiac rate and rhythm have been investigated in the operating room and laboratory because of their potentially dangerous effects during cerebral vascular surgery under hypothermia. In the course of these clinical and experimental observations, the effects of drugs and low body temperatures on brain-heart pathways and the action of the heart have also been considered.

Our clinical observations (Part I) are based on electrocardiographic findings during 40 intracranial operations of which 20 were done for cerebral vascular lesions: 19 for aneurysms of the circle of Willis and 1 for a parietal lobe angioma. Hypothermia was employed in 15 of these 20 cases. General anesthesia, consisting of intravenous Pentothal sodium and intratracheal oxygen-nitrous oxide, was administered for 37 of the 40 operations and local anesthesia for 3.

Ancillary experimental studies (Part II)² were carried out on over 30 cats to test the effects and thresholds of single and combined or conditioning stimuli of central and peripheral origin on cardiac activity. The influence of certain drugs and of lowered body temperatures upon the nervous system and upon the heart was also tested.

It is our hope that correlation of the clinical and experimental data so obtained will clarify some of the basic principles of brain-heart physiology and thus enable us to reduce or prevent cardiac complications of neural origin during surgery.

Historical. There are considerable data to indicate that cardiac activity in man may be altered by central influences. It is well known, for example, that increased intracranial pressure may induce bradycardia, presumably because of pressure on the vagal nuclei. Additional pressure in turn may lead to tachycardia, possibly because of vagal release or central compensatory mechanisms.

Disturbance of brain-stem function, as from hemorrhage or compression, may also lead to bradycardia or tachycardia, depending on the location and extent of the pathological process. Disturbances of hypothalamic function

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or of the visceral cortex\textsuperscript{10} may likewise affect the heart, as exemplified by palpitation and tachycardia in diencephalic\textsuperscript{9} or temporal lobe\textsuperscript{5} epilepsy. It has, moreover, been shown by White \textsuperscript{16} that electrical stimulation of the anterior hypothalamus in patients under ether anesthesia may induce bradycardia, but not when midbrain pathways are blocked by a tumor, or when atropine has been given. Mechanical or electrical stimulation of the anterior cingulate cortex\textsuperscript{12} or uncus\textsuperscript{6} in man may also alter cardiac rate or rhythm, independently of or together with blood pressure and respiratory changes.

While pain has been induced by manipulative stimuli applied to vessels of the circle of Willis in man,\textsuperscript{14} we have found that similar stimuli applied to the circle of Willis may induce alterations in cardiac rate or rhythm, particularly under abnormal conditions imposed by hypothermia, together with insufficient neural blocking.\textsuperscript{11}

It is our purpose therefore to present examples of this and related phenomena to illustrate how certain central stimuli may at times alter the rate, rhythm or configuration of the electrocardiogram.

\textbf{METHODS OF PRESENT STUDY}

(a) \textit{Anesthesia.} Approximately 1 hour before induction of anesthesia, each patient was given Demerol 100 mg. (h) and scopolamine 0.4 mg. (h). Immediately prior to craniotomy, moreover, the site of the scalp incision was infiltrated locally with 1 per cent procaine or Novocain in all 40 cases.

The 3 patients operated upon under local anesthesia alone were given additional Demerol by vein from time to time throughout the procedure to render them drowsy but not incapable of response. In one a posterior rhizotomy for tic douloureux was performed; in another, a subtotal temporal lobectomy for psychomotor epilepsy, while in the third no anesthesia other than local Novocain was given until exposure of a bleeding aneurysm of the internal carotid artery was completed.

All other patients were anesthetized with general anesthesia consisting of intravenous drip of Pentothal sodium supplemented by intratracheal administration of oxygen-nitrous oxide.

In all hypothermic cases cooling was induced by placing the patient on a Therm-O-Rite\textsuperscript{*} mattress and then packing the body with ice cubes. Body temperature was constantly recorded by an intragastric electrically recording thermometer.\textsuperscript{†} To some of these patients chlorpromazine was given in 10 mg. amounts to a total of 40 to 110 mg., while succinylcholine was administered to a few others. There was no apparent correlation between the dosage of these drugs and the reported cardiac effects.

(b) \textit{Electrocardiography.} Constant monitoring of cardiac activity was carried out in all hypothermic patients with a cardiotachiscope,\textsuperscript{‡} and in all but the first 3 hypothermic patients a recording electrocardiographic apparatus was also used, as for all other operations of this series. "Minor" elec-

\textsuperscript{*} Therm-O-Rite Products Corporation, Buffalo, N.Y.
\textsuperscript{†} Tele-Thermometer, Yellow Springs Co., Yellow Springs, Ohio.
\textsuperscript{‡} Instrument Laboratories, New York.