

clinical hemorrhagic tendencies. The levels of prothrombin as determined by the Russell viper venom modification of Quick's method cannot at all times be correlated with the clinical state of the patient and the dicoumarol therapy. The control of dicoumarol therapy by the Russell viper venom method for pro-

thrombin determination is a dangerous procedure. The results may be interpreted as being in a safe therapeutic level of prothrombin when actually the patient may be in a critical potential or actual hemorrhagic condition, this state being determined both by the Quick method and clinical observations.

16007

Electrocardiogram-Electroencephalogram Relations in Cardiac Arrhythmias.

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The initiating relationship of the brain, and in particular of the hypothalamus, to cardiac arrhythmias has been established on both experimental¹⁻⁴ and clinical⁵⁻¹⁰ grounds. Although a proportion and probably a majority^{11,12} of the arrhythmias may find their

prime origin in intracardiac derangements, numerous cases of various cardiac arrhythmias are on record^{13,14,15} in which the heart is normal. This study contains electroencephalographic data in cases of arrhythmic patients.

The electroencephalograms (EEG) were taken using the hypothalamic lead. Confirmatory electrocardiograms (EKG) were taken in each instance, except one, prior to the EEG. The EEG classification of Gibbs was used in determination of normalcy.† The results are summarized in the table, and tracings in Case 1-1a are reproduced in Fig. 1.

Interpretation. These cases may be divided

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TABLE I.

Patient			Other diagnosis	Arrhythmia by EKG	Preliminary EEG	Medication for conversion to normal rhythm			EKG with normal rhythm	EKG with normal rhythm
No.	Sex	Age				Abnormal	Quinidine	Normal		
1.	♂	32	None	Paroxysmal auricular fibrillation-flutter	Much fast 12-20 C/S	Abnormal	Quinidine	Normal	Normal	Normal
1a.	♂	32	None (Readmission)	Same	Same	Same	"	"	"	"
2.	♂	50	None	Paroxysmal auricular flutter	"	"	"	"	"	"
3.	♂	50	"	Paroxysmal auricular fibrillation-flutter	None taken	None taken	"	"	"	"
4.	♀	58	"	History of parox. tachycardia, 30 years	—	—	—	Abnormal	Questionable evidence of myocardial damage	Normal
5.	♂	50	"	History of parox. auric. fibrillation-flutter, 12 years	—	—	—	4.9 C/S, High ampl. Alpha	Borderline, Mixed frequency	Normal
6.	♂	50	Arteriosclerosis; cardiac hypertrophy	Auricular fibrillation	Normal	Normal	None attempted	—	8-22 C/S	—
7.	♂	63	Buerger's disease; amputations of 4 extremities	Extrasystoles, multiple foci. (Evidence of old myocardial infarction.)	"	"	"	—	—	—
8.	♂	55	History of rheumatic fever	Auricular fibrillation	"	"	Quinidine	Normal	Normal	—

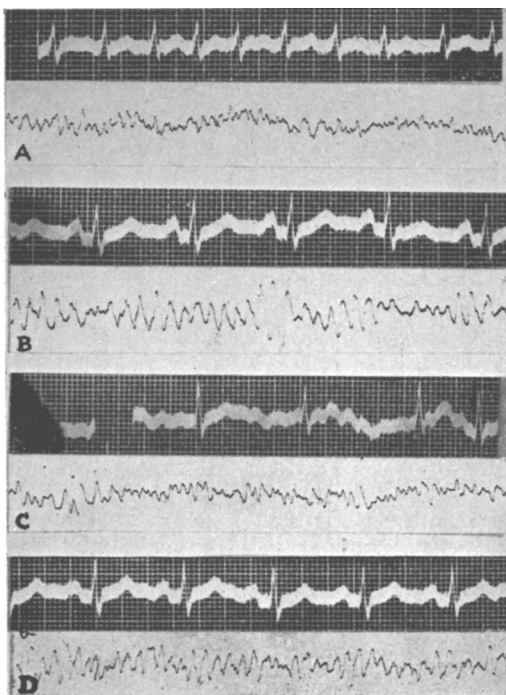


FIG. 1.

Electroencephalograms and electrocardiograms in a case of paroxysmal auricular fibrillation-flutter.

A. 3-20-47. C. 6-20-47 (Readmission).

B. and D. After conversion to normal cardiac and encephalic rhythm, using quinidine.

B. 5-8-47. D. 7-16-47.

EKG—Lead II. EEG—Left.

into 2 general groups: Those in which there is EKG history and physical evidence of myocardial damage, and those without such evidence. Cases 1, 2, 3, 4, and 5 fall within the second category. Reverse cerebral excitation is not supported by findings in cases 6, 7 and 8. EEG abnormality in a case with a history of long-standing paroxysmal arrhythmia without definite evidence of myocardial damage (Cases 4, 5) may or may not be pertinent to the cardiac arrhythmia. Experimental⁴ and clinical¹³ demonstration of the variety of possible neurogenic cardiac arrhythmias does not make feasible any separation at this time on the basis of type of arrhythmia. Factors not associated with the arrhythmia may cause an abnormal EEG and therefore the indication that an abnormal EEG may accompany a central excitation causing the cardiac arrhythmia is gained only from Cases 1-1a, and 2. Even here it will be necessary to investigate the possibility of cerebral circulatory insufficiency secondary to the cardiac arrhythmia.

Summary. Evidence is presented to indicate that electroencephalographic abnormality coincident with neurogenic cardiac arrhythmia may disappear with restoration of normal cardiac rhythm.

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Pressure Stimulation of Peripheral Nerves.

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Clinical experience suggests that pressure upon peripheral nerves or nerve roots is accompanied by pain, as well as by alterations in sensation and other neurological changes. The radicular pains caused by neoplasms of the spinal canal, the root pains arising from herniations of the intervertebral discs and the pain caused by pressure on the brachial

plexus of cervical ribs or thickened scalenus muscles are common examples. Pain occurs early and persists throughout the course of these conditions and has been attributed to the stimulating effect of the steady pressure against the nerve.

On the other hand, experimental physiological studies have repeatedly demonstrated that nerve fibers are relatively inexcitable to steady or even slowly changing stimuli. The classical studies on electrical stimulation of

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