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Electrocardiographic Evidence of Cardiac Involvement in Acute Disease.

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Daily electrocardiograms were taken on patients suffering from acute disease who received no digitalis or quinidine. (Table I.) No such systematic electrocardiographic study has been made except in rheumatic fever,[1] pneumonia,[2] and acute rheumatoid (infectious) arthritis.[3] The recorded abnormalities were produced during the illness. Transient changes in lead III only were not included in the table.

For control data, 50 cases of acute rheumatoid (infectious) arthritis, 6 cases of ulcerative colitis and 9 cases of peptic ulcer were chosen. In the former 2 groups the patients were acutely ill with fever. In all the control patients no abnormalities in T-, QRS-waves, or in RS-T transitions, no auricular flutter or fibrillation, no heart-block were observed. Hence, one may conclude that an acute illness with fever and tachycardia does not produce significant arrhythmias, increased P-R intervals, T-wave inversions or marked RS-T changes unless the myocardium is involved.

Most marked heart-block and the most frequent arrhythmias were observed in acute rheumatic fever but a larger number of patients with this disease was studied than in any other. In acute disseminated lupus erythematosis, in periarteritis nodosa, in Malta fever, in capillary toxicosis, bronchopneumonia, typhoid and acute rheumatic fever, T-wave changes were most frequent. It is suggested that some of these diseases may have similar pathological alterations in the myocardium of vascular nature and that they may produce permanent heart damage.

A very fast pulse occurred in many cases with severe myocardial involvement. In 7 cases of pulmonary tuberculosis, for example, with rates above 120 beats per minute, 6 disclosed abnormal electrocardiograms. In this disease the occurrence of a heart rate of over 120 beats per minute should suggest myocardial involvement. However, a slow pulse did not indicate a good myocardium, for

[1] Cohn, A. E., and Swift, H. F., *J. Exp. Med.*, 1924, **39**, 1.

[2] Master, A. M., Romanoff, A., and Jaffe, H., *Am. Heart J.*, 1931, **6**, 696.

[3] Master, A. M., and Jaffe, Harry, *J. A. M. A.*, 1932, **98**, 881.

TABLE I.

	No. Patients	Aver. No. Records	Tachycardia	Bradycardia	Arrhythmias*	P-R over 0.20 sec.	Maximum P-R	T-wave Inversions	RS-T Changes
Acute rheumatic fever	63	30	68%	21%	37%	53%	.56	28%	85%
Typhoid	15	25	87	27	13	33	.24	27	33
Acute Lupus Erythematosis	6	16	100		17	33	.24	50	50
Typhus	14	9	64	28		35	.28	7	14
Lobar pneumonia	45	15	69	51	10	11	.24	15	93
Bronchopneumonia	8	15	50	13			.20	37	13
Pulmonary Tuberculosis	27	11	42	7	3	11	.24	15	11
Gonorrheal Arthritis	16	11	31	6		19	.26	19	13
Capillary Toxicosis	3	14	33					63	33
Periarteritis nodosa	4	7	75					100	
Malta Fever	2	27	100					100	
Trichinosis	4	8	50			25	.22	33	
Erythema nodosum	14	13	50	21	7	14	.23	14	
Malaria	7	7	29	43	14	14	.34		29
Erythema multiforme	5	9	40			20	.22		
Urticaria	6	9		16					
Purpura hemorrhagica	6	9	16	16		16	.21		
Controls									
Ulcerative Colitis	6	9	50	16	16				
Peptic Ulcer	9	9							
Rheumatoid Arthritis	50	30	34	20	2	10	.22		

* Premature beats, sino-auricular block, nodal rhythms, auricular flutter, or fibrillation, heart-block with dropped beats.

bradycardia and heart-block were observed in patients with marked electrocardiographic abnormalities.

The increased A-V conduction time in typhus fever (Brill's disease), occurred usually at the time of the crisis. A similar observation was found in lobar pneumonia.[2]

Unlike the observations in acute rheumatoid (infectious) arthritis one not uncommonly found significant electrocardiographic changes in patients with gonorrheal arthritis.

Cases of erythema nodosum and erythema multiforme revealed very little evidence of myocardial damage and hence their relation to acute rheumatic fever is probably remote.

In none of the diseases studied was a definite progressive and continuous change observed from an RS-T abnormality to an inverted T-wave as is seen following an acute coronary artery occlusion. This may serve as a means of differential diagnosis between acute coronary artery occlusion and other conditions which may produce severe chest, sternal or precordial pain such as pneumonia or pulmonary artery occlusion.

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Induction of the Olfactory Placode by the Forebrain in Rana pipiens.

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Bell,[1] using embryos of *Rana esculenta,* attempted to show the relation of the brain to the development of the olfactory placode. He transplanted "that part of the brain which forms the olfactory lobe" to other regions of the body without obtaining any induction of an olfactory placode. His conclusion was that the olfactory placode developed independently of the brain. He did not, however, preclude the possibility of an early stimulus for olfactory placode formation being imparted by the brain to the ectoderm above it.

Recently we have been able to show that the part of the forebrain median and slightly anterior to the optic vesicle of the intermediate neurula stage of *Rana pipiens* has the power to induce an olfactory placode in the ectoderm of the flank (Fig. 1).

[1] Bell, E. T., *Arch. f. Ent. Mech.*, 1907, **23.**