ance of fibroblasts upon the cemental surface. These then undergo the usual fibroblastic development attended by lengthening of the nucleus and cytoplasm in a manner similar to that found in other portions of the body. The point of especial interest lies in the fact that growth of this tissue is begun at the tooth surface rather than from the alveolar surface. This is contrary to certain previous conceptions.

After reconstruction of peridental fibres has begun at the cemental surface, repair of bone commences. Osteoblasts appear. Cancellous bone is laid down about a fibrous net work. Fusion of fibres already formed precedes bone formation. Colloidal precipitation attends it as proven by the stain reaction of Van Giesen technique. Thus, following the genesis of the fibres at the cementum, the gap across to the bone is spanned and now the fibre becomes incorporated in the reforming alveolar process. Repair thus may proceed to completion.

This is a preliminary report.

[1] Hanford, W. H., Patten, C. O., Westbay, C., and Simonton, F. V., *Dental Cosmos*, 1923, lxv, 12-17.

[2] Fleming, W. C., A Clinical and Microscopical Study of Peridental Tissue Treated by Instrumentation. Paper delivered at Pacific Dental Conference, Portland, Oregon, June 25, 1926.

[3] Williams, Adrienne, *Dental Cosmos*, in press.

3465

Pulse Pressure, Its Probable Relationship to Stroke Volume.

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Numerous investigators have shown that in hyperthyroidism the tachycardia varies in degree directly with the metabolic rate. It seems reasonable to suppose that this increased heart rate is an adaptation for increasing the cardiac minute volume, since the minute volume is known to be increased in this condition. Previously[1] I have pointed out that the pulse pressure also varies directly with metabolism. In Table I are shown the values obtained in 1000 observations of the pulse rate, blood pressure and metabolic rate made simultaneously under basal conditions. It will be observed that the rise in pulse pressure with increasing b.m.r. is due to rise in systolic pressure, since the diastolic pressure remains practically constant.

The pulse rate and pulse pressure both increase in a parallel manner with elevation of the basal metabolic rate.

When gathering these data it was observed that some subjects showed a disproportionate increase in either pulse rate or pulse pressure, with little or no change in the other. This suggested that some subjects might increase the minute volume by increasing the rate of output per minute, while others accomplished the same increase in total blood flow by increasing the volume ejected into the aorta at each systole. Pulse pressure might then be a rough measure of stroke volume.

Recently there came under observation two individuals with complete heart block, as shown by electrocardiogram, neither of whom suffered limitation of activity imposed by circulatory insufficiency. One was a woman, 47 years old whose slow pulse had excited interest since girlhood. P. R. 36-44, blood pressure, systolic 180, diastolic 80. The other was a man, 70 years old. P. R. 28-30. Systolic pressure, 200-240; diastolic, 70-100. Neither had aortic insufficiency.

The normal diastolic pressures in these subjects and the lack of any circulatory embarrassment, with moderate cardiac enlargement suggested that the blood pressure changes were adapted to permit an adequate minute volume even with a pulse rate reduced to about half the normal. The wide pulse pressure in these cases I interpret as

TABLE I.

The mean values for blood pressure and pulse rate as they vary through the normal and pathologic ranges of basal metabolic rate.

Basal metabolic rate	Number of observations	Blood Pressure		Pulse pressure	Pulse rate
		Sytolic	Diastolic		
120.1 to 130	1	166	89	77	159
110.1 to 120	1	170	60	110	132
100.1 to 110	1	154	70	84	121
90.1 to 100	2	152	77.5	74.5	116
80.1 to 90	3	136.7	78	58.7	122
70.1 to 80	12	148.6	70	78.4	111
60.1 to 70	32	138.2	72	66.1	109.1
50.1 to 60	48	133.7	71.2	62.3	104.4
40.1 to 50	76	130.8	73.2	58.5	100.8
30.1 to 40	110	126.4	73.2	53.1	93.3
20.1 to 30	123	122.9	71.0	51.1	83.3
10.1 to 20	142	118.0	73.0	45.1	79.8
0.1 to 10	152	110.3	68.5	42.4	72.5
-9.9 to 0	138	107.7	69.2	38.8	68.9
-19.9 to -10	117	108.2	71.3	36.8	65.7
-29.9 to -20	87	106.9	73.3	32.9	62.7
-39.9 to -30	5	97.2	66.4	30.8	58.4
Total	1000				

indicating increased stroke volume and believe that, with a normal diastolic pressure, pulse pressure variations indicate variations in stroke volume and that a reciprocal relationship exists between pulse pressure and pulse rate if the minute volume remains constant. This is a preliminary report.

[1] Read, J. Marion, *Arch. Int. Med.*, 1924, xxxiv, 553.

3466

The Use of the Cathode Ray Oscillograph for Electrocardiography.

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In order to time the point in the cardiac cycle at which a number of rapid roentgen ray films of the heart are taken, it was wished to develop a small portable electrocardiograph with no string or delicate part to be damaged by the currents from the X-ray circuit. The cathode ray oscillograph, as first used by Erlanger and Gasser,[1] has proved quite satisfactory for making electrocardiographs. Gasser[2] found that with his amplifier the heart currents gave lower curves than similar voltages in nerve preparations, due to the low resistance of the body as compared with the resistance of the amplifying tube. The apparatus with which this record was made differs from that of Gasser and Erlanger (a) in that the same source of current is used for cathode ray tube and amplifiers; (b) the sensitive film moves over the face of the tube giving a continuous curve instead of standing curves; (c) four amplifying tubes are used, the first three with amplification constant 20, the last a UX112 tube with a constant of 7; (d) one lead goes directly to the filament of the first tube, the other passes through a 2 microfarad condenser to the grid, which connects to the filament through a one megaohm leak. The apparatus gives curves with a motion of one cm. per one millivolt input and at rates of motion of film up to fifteen cm. per second.

[1] Erlanger, J., and Gasser, H. S., *Am. J. Physiol.*, 1924, lxii, 496-524.

[2] Gasser, H. S., personal communication.