the various quantities of the special ingredients are added. Each ingredient is added separately under strict asepsis to the melted agar which is kept liquid in a hot water bath between the filling procedures. Upon adding the last ingredient, the tube is slanted to allow the content to solidify in this position.

Summary. The trypsinization of the leprous tissue bits, before attempting the cultivation of the contained *B. leprae,* affords at once the split-protein products which we consider the necessary nutrient factor. The trypsin apparently exerts no harmful effect upon the Hansen bacillus as evidenced by its unmistakable growth in 4 to 6 weeks. This initial *in vitro* multiplication occurs in the trypsinized leprous tissue without the aid of other nutrients and under ordinary atmospheric conditions at 37°C. Growth continues in subculture on ordinary agar medium as long as the trypsinized leprous material is transferred. The crucial point in the initial cultivation of *B. leprae* away from the influence of the host tissue is experienced at this juncture. After the complete utilization of the nutrient material of the trypsinized host tissue, the acid-fast bacillus of Hansen will only multiply in a medium that is enriched with sufficient quantities of protein cleavage products.

7339

Repeated Determinations of Pulse Wave Velocity on Normal Individuals.*

ROY H. TURNER AND W. A. SODEMAN.

From the Department of Medicine and the Hutchinson Memorial Clinic of Tulane University of Louisiana, New Orleans.

The existing literature concerning pulse wave velocity deals with the effect of disease, age, sex and physiologic and pharmacologic stimuli on pulse wave velocity in groups of individuals. Spontaneous fluctuations in the same individual from time to time have received slight consideration.[1, 2, 3, 4]

* Aided by grants from the David Trautman Schwartz Research Fund and grant No. 163 of the Committee on Scientific Research of the American Medical Association.

1 Beyerholm, O., *Acta Med. Scand.,* 1927, **67**, 203.

2 Bazett, H. C., and Dreyer, N. B., *Am. J. Physiol.,* 1922, **63**, 94.

3 Schafer, E. A., *Textbook of Physiology,* Vol. II, Edinburgh, 103.

4 Keyt, A. T., *N. Y. Med. J.,* 1878, **28**, 30.

TABLE I.

B.P.	Mean P.W.V. Meters/sec.	High - Low	
Subject 1, male, aged 39.			
112/74	6.84	6.16 - 7.28	C-R I*
110/80	13.3	11.48 - 17.2	C-R I
not rec.	7.38	7.30 - 7.91	C-R I*
" "	6.17	5.82 - 6.53	C-R I*
115/80	8.17	7.73 - 8.89	C-R I
115/80	8.41	8.1 - 8.95	C-R I
118/90	6.64	6.17 - 6.93	C-R I standing
116/84	6.33	6.18 - 6.70	C-R I lying
108/76	8.47	7.75 - 8.96	C-R R
106/84	14.4	10.9 - 17.0	C-L I
Subject 2, male, aged 27			
117/88	10.9	9.8 - 11.5	C-R I
118/84	7.22	7.13 - 7.62	C-R I*
not rec.	9.05	8.45 - 9.6	C-L I
117/88	11.2	10.5 - 12.1	C-L I
118/88	7.62	6.82 - 7.83	C-L I*
not rec.	8.88	7.85 - 9.80	C-R R
110/85	6.45	5.8 - 7.24	C-R R*
Subject 3, female, aged 27			
102/80	5.75	5.20 - 6.64	C-R I
100/62	6.86	6.67 - 7.37	C-R I*
102/78	8.03	7.30 - 9.14	C-L I
100/62	7.00	6.67 - 7.37	C-L I
110/80	5.7	5.3 - 6.23	C-R R
90/70	6.63	6.23 - 7.07	C-R R*
110/80	8.41	7.58 - 9.63	C-L R
97/75	5.0	4.77 - 5.25	C-L R*
95/70	5.55	5.25 - 6.19	C-L R*
Subject 4, female, aged 22			
90/65	5.78	5.30 - 6.32	C-R I*
90/65	7.85	7.35 - 8.80	C-L I*
95/60	7.03	6.5 - 7.43	C-R R
95/60	5.72	5.47 - 6.12	C-L R
100/68	5.82	5.55 - 6.25	C-L R*
Subject 5, female, aged 45			
115/80	7.87	7.5 - 8.06	C-R I*
115/80	6.80	6.45 - 7.18	C-L I*
122/86	7.02	6.73 - 7.84	C-R R
122/86	8.30	7.23 - 9.40	C-L R
110/83	6.25	5.94 - 6.33	C-L R*
Subject 6, female, aged 24			
100/74	8.70	7.55 - 9.13	C-R I
102/72	7.55	6.45 - 8.65	C-L I
112/80	6.42	6.27 - 6.57	C-L I standing
106/75	6.93	6.00 - 7.6	C-L I lying
98/60	11.5	10.8 - 13.5	C-R R
98/60	9.15	8.15 - 9.64	C-L R
105/68	5.45	5.40 - 5.68	C-L R*

Mean P.W.V. is the average of 6 to 18 consecutive complexes in all phases of respiration.

High-low = highest and lowest P.W.V. in each record.

C-R I = left carotid-right index fingertip; C-L R = left carotid-left radial; C-R R = left carotid-right radial; C-L I = left carotid-left index fingertip.

* = determinations in which conditions approached those used in determination of basal metabolism and cardiac output.

We have measured repeatedly during a period of 6 months the pulse wave velocity in 6 normal individuals of 22 to 45 years of age. A simplified modification of a technic previously described[5] was used, which employs carbon grain microphones and 2 Einthoven string galvanometers. Carotid and either radial or index fingertip sphygmograms, together with respiratory movements, were recorded simultaneously and measurements made from reference points on the ascending limbs one-fifth of the vertical distance from beginning of upstroke to the peak. Brachial blood pressure was taken immediately following the taking of the record. With a camera speed of 10 cm. per second measurements were made with accuracy to 0.002 second. Subjects were examined at approximately the same time of day, in the sitting position (unless indicated otherwise) with the arms resting horizontally on supports. Preliminary rest periods varied from 5 to 45 minutes. Room temperatures were fairly constant. Noises and other demands upon the subjects' attention were kept at a minimum.

Careful scrutiny of Table I shows that for the group as a whole there is no apparent correlation between pulse wave velocity and systolic and diastolic pressure. When pulse wave velocity for each subject was plotted against systolic and diastolic blood pressures the following relationships were noted:

Subject	Systolic	Diastolic	Subject	Systolic	Diastolic
No. 1	—	±	No. 4	±	±
No. 2	±	±	No. 5	+	±
No. 3	±	±	No. 6	—	—

+ = P.W.V. tends to increase with B.P.
— = P.W.V. tends to diminish with B.P.
± = No apparent correlation.

Most workers[2, 3, 6, 7] seem to agree that in normal arteries blood pressure is the principal factor which influences pulse wave velocity and that as blood pressure varies pulse wave velocity also varies and in the same direction. Some observers are not in full accord.[8, 9, 10]

We have noted in individuals with apparently normal arteries and with normal blood pressure pulse wave velocity which was extremely

[5] Turner, R. H., *Johns Hop. Hosp. Bull.*, 1928, **43**, 1.

[6] Hickson, S. K., McSwiney, B. A., *J. Physiol.*, 1924, **59**, 217.

[7] Laubry, C., Mougeot, A., and Giroux, R., *Arch. d. med. du Coeur*, 1922, **14**, 49, 97.

[8] Fulton, J. S., and McSwiney, B. A., *J. Physiol.*, 1930, **69**, 387.

[9] Dawson, *Am. J. Physiol.*, 1917, **62**, 613.

[10] Hafkesbring, R., and Ashman, R., *Am. J. Physiol.*, 1932, **100**, 89.

high even when the blood pressure was lower than usual for that individual. A related observation has been made before in this laboratory[11] with a less satisfactory method when pulse wave velocity in patients with hypertension actually increased following a drop in pressure produced by nitrites.

The observations here reported and others to be published later indicate to us that changes in arterial tone largely independent of the blood pressure may greatly influence pulse wave velocity in the brachial and radial arteries. Such a concept gives to the artery not a passive rôle, merely distended by its internal pressure, but ascribes to it an active dynamic rôle more in keeping with its known muscular equipment. Final interpretation of data such as here presented is impossible without knowledge of the changes in arterial calibre which is an important variable influencing pulse wave velocity.

It was noted also that as the conditions of the experiment approach more closely the basal conditions laid down for the determination of basal metabolism and cardiac output that pulse wave velocity became lower and less variable.

7340 P

Relation of Adrenals to Diabetes.*

FRANK A. HARTMAN AND KATHARINE A. BROWNELL.

From the Department of Physiology, University of Buffalo.

Frouin[1] found that pancreatectomy produced less glycosuria than usual, in dogs from which a considerable amount of adrenal tissue had been removed. Later work[2] has indicated an interrelationship between the adrenal and the pancreas. The adrenal cortex is necessary for the development of severe diabetes.[3] The medulla, however, may be significant in preventing insulin shock.[4]

If the cortex is necessary for the full development of diabetes, is its cortin-producing or some other function responsible?

11 Turner, R. H., and Herrmann, G. R., *J. Clin. Invest.*, 1927, **4**, 430.

* Aided by a grant from the Carnegie Corporation of New York to the Carnegie Institution of Washington.

1 Frouin, A., *Compt. rend. Soc. de Biol.*, 1908, **64**, 216.

2 Riddle, O., Honeywell, H. E., and Fisher, W. S., *Am. J. Physiol.*, 1924, **68**, 461.

3 Turcatti, E. S., *Rev. Méd., Rosario*, 1929, **19**, 269.

4 Boggild, D. H., *Acta Med. Scand.*, 1933, **79**, 458.