6661

Temperature of Circulating Blood and Other Tissues with Associated Observations of Capillary Activity. *

IRVING S. WRIGHT AND HARRY J. JOHNSON.
(Introduced by H. O. Mosenthal.)

From the Department of Medicine, New York Post-Graduate Medical School and Hospital.

Studies of tissue temperature have been made by means of fairly accurate instruments since the work of Becquerel and Breschet.[1] Interest in recording the temperature of the circulating blood in humans has been secondary to the development of scientifically accurate apparatus which has been devised on thermo-electric principles. Clark[2] developed a galvanometer deflection method of measurement. Harris and Marvin,[3] and Bradburn and Blalock[4] studied changes in the temperature of the venous blood of the hand after immersion in water at various temperatures—by means of direct measurement. Foged[5, 6] reported observations on 116 patients concluding that the temperature in the ulnar vein averaged 2.6 lower than the rectal temperature. He noted, in the presence of fever, an increase in this difference and also that the temperature of the surrounding medium affected the temperature of the venous blood. Other of Foged's observations will be discussed in reference to our findings.

Our observations made on humans were recorded by means of one recording and one non-recording potentiometer (Leeds and Northrup). The readings were taken by means of a thermocouple using the metals copper and constantan at the junction. This thermocouple was contained in the tip of a No. 20 hypodermic needle and the same thermocouple was used for all readings taken in one individual. Mouth temperature was used instead of rectal—making sure that the thermocouple junction rested firmly against the under surface of the tongue. This was done because (1) the relationship between mouth and rectal temperature has been fairly well estab-

* This work was aided by grants from the Josiah Macy, Jr., Foundation, the Oliver Rea Fund, and the Harriet Weil Fund.

1 Becquerel and Breschet, *Comptes Rend. de l'Acad. des Sciences*, 1835, **1**, 28.

2 Clark, H., *J. Exp. Med.*, 1922, **35**, 385.

3 Harris, K. E., and Marvin, H. M., *Heart*, 1927, **14**, 49.

4 Bradburn, H. B., and Blalock, A., *Am. J. Physiol.*, 1929, **91**, 115.

5 Foged, J., *Skandinav. Arch. f. Physiol.*, 1930, **59**, 109.

6 Foged, J., *Hospitalstidende*, 1930, **73**, 1079.

lished; (2) the use of the needle made rectal readings more difficult and possibly uncomfortable; (3) it was easier to be certain that the tip of the needle rested against known tissue in the mouth; (4) changing the thermocouple or from thermocouple to mercury thermometer would introduce another possible source of error.

The room temperature was maintained between 20.5°C. and 24.5°C.

Intravenous temperatures were taken from the median cubital vein, jugular vein, and branches of the saphenous vein; intramuscular reading from the gluteal or deltoid muscles which were found to be practically identical.

Series I included 30 afebrile patients, studying the relationship between the mouth, intravenous and intramuscular temperatures. In all cases, and in all veins above mentioned the intravenous temperature was lower than the mouth temperature. In the 13 cases tested, the mouth temperature was lower than the intramuscular temperature (with one exception—case 4, which may have been due to error in position of needle). The lowest intravenous temperature reading was 84°F. (28.8°C.) which was doubly checked. Mouth temperature was also low in this patient, 97°F. (36.1°C.) but the difference of 13°F. was the greatest observed. The least difference between mouth and intravenous was noted in case 21—1.2°F. The greatest difference noted between mouth and intramuscular was found in case 7—2.2°F.

Series II represented a study of the relationship between mouth, intravenous and intramuscular temperatures in febrile cases; 13 cases were studied in 3 of which only the mouth and intravenous temperatures were taken. In these cases the fever was due to a disease process, *i. e.,* pneumonia, Hodgkin's disease, rheumatic fever, pulmonary tuberculosis, bacterial endocarditis, etc. All the temperatures were increased, the same general relationship maintaining, namely: Intravenous lowest, then mouth, and intramuscular temperature highest. We can establish no consistent change in relationship. The consistently highest average held by the intramuscular temperature tends to confirm the belief that the muscles are local generators of heat by metabolic activity.

Series III represented a study of the relationship of the subcutaneous temperature to the mouth, intravenous and intramuscular temperatures. Eight patients were used. In our cases the subcutaneous temperature was found to be either higher or lower than the intravenous temperature, but never equal to the mouth or intramuscular temperatures.

CHART I.
Intra-arterial Temperature—Relation to Mouth and Intravenous Temperature.

Patient	Mouth Temp.		Intravenous Temp.		Intra-arterial Temp.	
	F	C	F	C	F	C
1	99.6	37.5	96.0	35.6	97.25	36.4
2	98.4	36.8	92.0	33.3	92.75	33.7
3	101.5	38.6	93.0	33.9	99.0	37.2
4	105.5	40.8	98.0	36.6	103.2	39.6

Chart I gives the figures of 4 intra-arterial temperatures taken in the radial artery and compared with the mouth and intravenous temperatures of the same patients. Foged reports 2 intra-arterial temperatures, one of which equaled the rectal temperature, and one of which was 0.3°C. lower than rectal temperature, and concludes that the arterial temperature is apparently the same as the rectal temperature. We have found the arterial temperature (in febrile and afebrile cases) to be between the intravenous and mouth temperature but not equal to the mouth (and hence rectal) temperatures.

Two cases were studied in which the temperature was taken in one area in the gluteal region beginning with the skin and progressing inward to the muscle, and comparing it with the intravenous and mouth temperatures. The skin was found to be higher than the subcutaneous temperature. The intramuscular was the highest in both cases, and the intravenous the lowest in one case and equal to the skin in the other.

CHART IIa
Effect of Fever Produced by Diathermia on the Mouth, Intravenous and Intramuscular Temperatures. (Hyperdiathermia Machine—General Electric)

Patient	Mouth Temp.		Intravenous Temp.		Intramuscular Temp.	
	F	C	F	C	F	C
1						
Normal	98.6	37.0	95.4	35.2	99.2	37.37
Peak of Fever	105.0	40.5	103.5	39.65	106.0	41.15
2						
Normal	98.2	36.9	94.4	34.6	100.0	37.7
Peak of Fever	102.8	39.2	99.5	37.25	104.4	41.0

CHART IIb. (Chart IIa continued)
Effect of Fever Produced by a 36 meter Wave Length Condenser Machine. (Hyperthermy spark gap—Lepel)

Patient	Mouth Temp.		Intravenous Temp.		Intramuscular Temp.	
	F	C	F	C	F	C
1						
Normal	98.0	36.8	96.0	34.6	98.0	36.8
Peak of Fever	104.4	40.2	102.5	39.2	104.5	40.3
2						
Normal	98.5	36.99	95.0	35.0	99.0	37.25
Peak of Fever	103.0	39.4	101.5	38.47	103.8	39.85

Chart IIa presents the mouth, intravenous and intramuscular temperatures of 2 patients in whom fevers were produced by means of the Hyperdiathermia Machine (General Electric). The temperature rises in each area reached the peak in 30 minutes after taking the normals.

Chart IIb presents the same statistics on 2 cases in which the fever was induced by means of a 36 meter wave length condenser machine (Hyperthermy spark gap—Lepel). The results are very similar in all readings.

CHART III.

Mouth and Intravenous Temperature Changes in Relationship to Changes in Capillary Activity during Fever Produced by *B. typhosus* Vaccine (30 million) Intravenously.

Time	Mouth Temp. F	Mouth Temp. C	Intravenous Temp. F	Intravenous Temp. C	Capillary Activity (Nailfold)
12 noon	98.0	36.6	92.75	33.7	
12:10	98.4	36.8	91.8	32.8	Few capillaries seen per field. Straight or slightly tortuous loops. Flow moderately rapid. Moderate dilatation. Occasional stoppage.
1:10	98.6	36.9	92.0	33.3	
2:30	99.9	37.7	93.0	33.9	
3:00	100.4	37.9	92.5	33.6	
3:30	100.4	37.9	92.5	33.6	
4:00	100.2	37.8	93.5	34.2	
4:20	100.0	37.7	95.0	34.9	
5:00	100.0	37.7	96.5	35.8	
					Estimated to be 4 times as many capillaries active per field. Flow rapid. No stoppage. Greater dilatation. Arterioles and venules seen with anastomoses active.

Chart III represents the mouth and intravenous temperature as taken on a recording potentiometer during the induction of fever by means of the intravenous injection of 30 million *B. typhosus* in vaccine. Both the mouth and intravenous temperatures rose but the intravenous temperature was observed to lag, showing no definite increase until 4 p. m., 3 hours and 50 minutes after the first increase noted in the mouth temperature. The thermocouple was left in the vein throughout this experiment so that the first changes could be noted. After 4 p. m. the mouth temperature started to decline but the intravenous temperature still continued to mount until the experiment was discontinued at 5 p. m. (4 hr. and 50 min.).

This intravenous lag was not noted in the preceding series, Chart II, and where fever was induced by other methods. We have not made continuous observations on rising temperature due to disease processes.

The intravenous temperature is subject to fluctuations of 2-4°F.

within a period of 2 minutes. These were in the form of a sudden drop and restitution to former level. When the intravenous temperature began to rise these fluctuations became more frequent, occurring as often as every 10 minutes.

This chart also illustrates diagramatically the great increase in capillary activity with the production of fever. The lower blood temperature would incline us to believe that the purpose of this was not a direct increase of local heat but rather a means of supplying the cells, especially of the muscles, with an increased food supply for metabolic activity and associated heat generation.

6662

A Comparison of the Value of Ferrous Iodide Administered Directly and Indirectly.*

HAZEL C. CAMERON. (Introduced by A. J. Carlson.)

From the Agricultural Experiment Station, West Virginia University.

Ferrous iodide has been administered orally as a tonic for many years in the practice of medicine without any evidence of toxicity when therapeutic doses are not exceeded. Recently, however, "toxicity" on direct oral administration has been ascribed to it in vitamin A deficient rats[1] in explanation of the negative results obtained by Mason,[2] Cameron,[3] and Mendel[4] when ferrous iodide was used as a substitute for vitamin A.

In all of these experiments, dilute ferrous iodide was given *by pipette or syringe* to rats depleted of vitamin A, a method which prompted the criticism of toxicity. In the following experiments, comparison is made of the effects of ferrous iodide when given by this method and when given as a part of the diet, thus avoiding any possible toxic effect of direct oral administration.

In these experiments dilute syrup of ferrous iodide alone or combined with linoleic acid has been given daily, mixed with a small

* Published with the approval of the Director, West Virginia Agricultural Experiment Station, as Scientific Paper No. 121.

1 Chidester, F. E., *Collecting Net.*, 1932, **7**, 229.

2 Mason, Karl E., *Anat. Rec.*, 1931, **51**, Supplement 1, Abst. 91.

3 Cameron, H. C., *Science*, 1932, **76**, 18.

4 Reed, Lucille L., Mendel, Lafayette B., and Vickery, H. B., *Science*, 1932, **76**, 300.