PART THIRD.

ON THE TREATMENT OF FEVERS.

FEVER FROM A THERAPEUTIC STAND-POINT.

SUMMARY.—Fever—Characteristics of Fever—Animal Heat—Clinical Thermometry—Its Importance from the Stand-point of Prognosis, Diagnosis, and Therapeutics—Anti-Thermic Medication—Pathological Physiology of Fever—Combustions in Fever—Products of Elimination—Phenomena of Combustion—Calorimetry—Theories of Fever—Vaso-Motor Theories—Theories of Traube, of Senator, of Marey—Theory of the Augmentation of Combustions—Nervous Theories—Calorific Centres—Thermic Nerves—Humeral Theories—Pyretogenous Substances—Therapeutical Deductions—Anti-thermic Medications—Physical Means—Subtraction of Heat—Cold Baths and Warm Baths—Subtraction of Blood—Bloodletting—Medicaments Acting on the Circulation—Digitalis—Ergot—Aconite—Veratrine—Antiseptic Medicaments—Quinine—The Aromatic Series—Phenic Acid—Salicylic Acid—Resorcin—Kairine.

GENTLEMEN: In this last part of my course of clinical therapeutics, I propose to consider the treatment of fevers, and the present lecture will be devoted to fever from a therapeutic stand-point. Such general considerations will be an excellent introduction to the study of specific fevers, which is to follow, and the utility of the subject will be a sufficient apology for the dryness and difficulty of many of the details into which I shall be obliged to enter.

Fever is characterized principally by two symptoms, elevation of the bodily temperature and increase of the pulse, the former symptom being regarded of preponderating importance as an essential element of fever, especially since the clinical thermometer came into use. The very etymology of the word (*febris*, *fever*, from *fervere* "to be hot"),[1] and the common consent of the medical profession from Hippocrates and Galen downward, sustain this view, and clinical thermometry constitutes, with auscultation and percussion, as Jaccoud very properly remarks, the foundation stones of the clinician's arsenal.

This study of morbid heat plays a preponderant part in diagnosis, prognosis and even in therapeutics, and physicians everywhere to-day, when they would judge of the intensity of a fever, and the effects of their remedies depend almost entirely on the examination of the temperature. You know how much we consult our clinical charts for daily information as to the fluctuations of the temperature, and the care which we take to have them absolutely correct, as constituting the basis of our treatment. The series of curved lines which I here pass before your eyes, shows you the march of the temperature in typhoid fever, in pneumonia, in small-pox, in intermittent fever, etc., and familiarity with this manner of registering the progress of the fever will

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[1] Fever (febris) comes from the word *fervere*, to be hot, to boil, ferment, glow; the Greeks employed the word *Πῦρ*, *Πυρετός*, whence the name *Πυρέξις*, pyrexia, applied to the febrile state.

Some authorities have thought that this word feber. *febris*, comes from *februrer*, to purge or to purify, but as Littré has remarked, this derivation is inadmissible.

enable you to a certain extent, by a sight of these curves, to tell what fever the patient has, and what are the chances of recovery.

Have we not gone too far in this direction and neglected the attentive study of the pulse? For my part I am convinced that it is generally best to make the study of the pulse and temperature march side by side. If the one furnishes us precise information concerning the state of the fever, the other enables us to determine the condition of the heart and the resistance of the patient. In many febrile maladies the heart undergoes profound alterations, which seriously modify its function and make our prognosis doubtful if not unfavorable.

Examine then the temperature, but do not neglect the pulse; study its frequency, its force, its form. Attempts have been made to establish a relationship between the pulse and the temperature, and Liebermeister and Lorain[1]

[1] Liebermeister's table, establishing the relationship between the pulse and fever, is as follows:

TEMPERATURE.		PULSE.		
		MINIMUM.	MAXIMUM.	MEDIUM.
98.6 F.	37. C.	45	124	78.6
99.5 F.	37.5 C.	44	130	74.
101.4 F.	38. C.	52	148	91.2
101.3 F.	38.5 C.	52	160	94.7
102.0 F.	39. C.	64	160	99.8
103.1 F.	39.5 C.	64	144	102.5
104.2 F.	40. C.	72	158	108.5
104.9 F.	40.5 C.	76	152	109.4
105.8 F.	41. C.	66	160	110.
106.7 F.	41.5 C.	88	160	118.5
107.6 F.	42. C.	114	168	137.5

LORAIN'S AVERAGES ARE AS FOLLOWS:

TEMPERATURE.	PULSE.
99.5 F.	70
101.3 F.	95
103.1 F.	120
104.9 F.	145
106.7 F.	160

According to Lorain, if the rectal temperature at 37.5 C. (99.5 F.), corresponds to 70, each rise of one degree will be accompanied by an increase of 25 pulsations. (*a*)

have given us precious indications in this regard; but these indications apply only to the number of pulsations, and tell us nothing about the volume or force of the pulse, which can only be made apparent by the touch and the sphygmograph.

I need not insist further on clinical thermometry, it is a subject which belongs to the domain of general pathology and clinical medicine, and I hasten to the study of fever, properly so called.

We have seen that fever is characterized by elevation of temperature; numerous theories have been proposed to explain this excessive heat, and in the last twenty years more especially works have multiplied on this subject. Notwithstanding the patient researches of many experimenters, this great question of physiological pathology is not yet completely elucidated, and we are in reality reduced to an acceptance of the hypotheses of our predecessors concerning the febrile process.[2] Without going very far into the history of the doctrine of animal heat and fever, and referring you to the remarkable study of Lorain on the "Temperature of the Human Body" for more exhaustive information, I may say that the ancients regarded fever as the result of modifications effected in the blood, or of some trouble in the innervation of the circulatory system. We find both this humoral and this nervous theory maintained at the present day, supported, it is true, on other bases, and by arguments drawn from modern physiological experimentation, and it is at the same time difficult to say which one of these theories is the more probable. But in order to approach this discussion profitably it seems to me necessary to enter here into certain details which will enable us to explain the different definitions which have been

[2] According to Hippocrates, and more especially Galen, fever depends on augmentation of the heat of the body; the heat is formed in the heart and the blood is the vehicle of it. Against this febrile state Galen counsels refrigerant medications, such as ptisans, and in particular barley tea, and the usage of cold lotions; these views prevailed without disputation till the sixteenth and seventeenth centuries.

Bellini considers fever as dependent on an alteration of the blood. Sydenham sees in it only a movement of nature to expel peccant matters. Stahl partakes of the same view. Bœrhaave makes particular account of the pulse, which for him characterizes the fever.

Cullen places the seat of the fever in spasm of the extremities of the small blood-vessels. Nietski developes this idea and considers fever as peripheral spasm of the vascular system. Frederic Hoffmann has expressed the same notion in saying that fever is a spasmodic affection of the whole nervous and vascular system. Todd says that fever is an irritation of the nervous system.

At an epoch nearer our own, Broussais expunged fever from pathology. Fever was to him a miraculous product of the imagination of physicians. Boullaud makes of fever an *angeiotenia*, and Piorry, an angeio hæmitis, that is to say an inflammation of the walls of the blood-vessels.

(*a*) Lorain, Temperature du Corps Humain, t. 1, p. 30, Paris, 1877. Liebermeister, Deutsch. Arch. f. Klin., Band 1, 1866, p. 466.)

given of fever.[1] Does the increase of heat, which is the characteristic of fever, result from an augmentation in the combustions of the economy? This is the point to be settled. The solution of this problem of physiology has been sought in the examination of the urine, of the expired gases, in the state of the blood itself, and finally in calorimetry. Let us examine each of these points.

Till the last few years, there seems to have been general unanimity in the affirmation that the ultimate product of organic combustions, urea, augments with the temperature in fever patients, and that this augmentation is, so to speak, in the ratio of the hyper-pyrexia. The experiments of Moss, Murchison, Brattler, Claude Bernard, Hirtz, Catel and Unruh, were regarded as absolutely demonstrative.[2] Nevertheless, a more attentive examination of the question has shown that this augmentation is only relative, and its reality has been so far denied that Charvot has even maintained that there is no relation between the

[1] Numerous definitions of fever have been given, "Fever is a morbid state constituted by a variety of functional disturbances, and especially by exaggerated bodily temperature and acceleration of the pulse." (Compendium of Medicine.)

"The words fever, pyrexia designate a morbid state of a certain duration, characterized especially by increase of bodily heat and increased quickness of the pulse," by malaisé and divers other functional troubles. (Grisolle.)

"Fever is a pathological state constituted by increase of the organic combustions and temperature. This elevation is constant, and invariable to this degree that it serves to define and specify the fever. * * * Conversely, every individual whose temperature undergoes persistent augmentation, has fever." (Jaccoud.)

"The best definition of fever," says Prof. (Sée), "is that which is founded on he primordial phenomenon which dominates the entire situation. It is that of Galen: *Calor præter naturum*."

The most complete definition of fever, and the one most conformable to the results of experimental physiology is that of Lereboullet—"Fever is characterized by a notable elevation of the temperature of the body, regulated under the influence of the nervous system, and is due to an exaggeration of the interstitial combustions, being determined most generally by an alteration of the blood." (Lereboullet, Dict. Encyclop., article Fever.)

[2] At the beginning of the researches concerning the elimination of urea in fevers, everybody seemed agreed in regarding the elevation of temperature as coincident with increased elimination of urea; thus Moss claimed that the curve of urea excretion is parallel with that of the temperature. Murchison declared that the temperature rose in the ratio of the quantity of urea in excess of the normal. Brattler, going still farther, maintained that one might calculate the temperature by the quantity of urea eliminated and that a temperature of 40° C. (104° F.) corresponds to an elimination of 40 grammes of urea (about 600 grains). Desnos, in his *thèse d'agrégation* of 1866, admits the complete parallelism between the secretion of urea and the elevation of the temperature.

But since this time, by dint of a more careful examination of the subject, and thorough study of the influence of alimentation and the function of certain organs on urea, it has appeared to observers that this augmentation does not follow the hyperthermia. Charcot and Robin maintained even that this secretion is below the normal during the fever. Liebermiester has shown that the augmentation of urea does not exist except in the first periods of febrile affections; afterwards this augmentation gives place to diminution.

However, despite these reserves, and especially as a result of making proper account of alimentation, the majority of observers are of the opinion that there is augmentation of urea. Claude Bernard acknowledges this augmentation, and asserts that febricitants eliminate on an average one and a half times as much urea as persons in the state of health. Senator admits that the combustion of albuminoid matters is augmented during the entire duration of fever. Hirtz has found this augmentation in cases of traumatic fever. Catel has arrived at the same results.

In fine, Unruh claims that the total excretion of nitrogen is one and a half times as much in the febrile process as in the normal state when the individual is fasting.

Anstie affirms that the augmentation in the production of urea, does not always necessarily accompany febrile phenomena, and he cites a certain number of cases where the quantity of urea has been below the normal. According to Zuelzer the sum total of azotized excretions is augmented during the fever and diminished during fasting and convalescence. The relative proportions of nitric acid and phosphorus during fasting remain in the urine in the normal limits of the quantity existing in the muscular substance and in the cerebral tissue. According to this experimenter, in the febrile state it is especially the denutrition of the muscles which is at stake, and in the state of fasting and of convalescence, that of the nervous tissue predominates. (*a*)

production of urea and febrile thermogenesis, and that the theory which explains the excessive febrile heat by exaggeration of organic combustion is not admissible. On the other hand, Charvot has affirmed that the quantity of extractive matters[3] follows with great exactness the thermal curve.

The views of Charvot have found few partisans, and despite the strong facts which he alleges in their support, the majority of physiologists and clinicians admit an exaggeration of urea production during the febrile process; not by any means, be it understood, by comparing the figures with those which are observed in the case of the healthy and well-fed individual, but with those which indicate the quantity of urea voided by persons who are fasting.

The same discussions which arose with reference to the quantity of urea excreted by fever patients have been revived when it has been a question of determining by the study of the gases of respiration the modifications which are effected in the combustions of the economy, and while Leyden, Silujanoff, and Frænkel affirm that there is an augmentation of carbonic acid, Wertheim

[3] The extractive matters in the urine of febricitants have been especially studied by Charvot. According to him, the quantity of extractive matters diminishes in inverse ratio to the abundance of urea, so that the curve follows precisely that of the temperature, since, according to him, the quantity of urea undergoes a notable diminution in fever. He admits, also, a diminution of the solid matters, and in particular, of chloride of sodium. Hœpffner arrives at similar conclusions, for, according to him, the curves of urea and of extractive matters take altogether different directions. (*b*)

(*a*) Anstie, On tissue destruction in the febrile state and its relations to treatment (the Practitioner, March-May, 1874).—Moss, Americ. Journ., p. 384-388, 1861.—Murchison, the Continued Fevers of Great Britain, London, 1873.—Brattler, Contributions à l'urologie, Munich, 1858.—Desnos, thèse agrég., 1866, p. 48.—Charvot, Température, pouls, urine dans la crise et dans la convalescence de la pneumonie.—A. Robin, Essai d'urologie clinique.—Senator, Untersuchungen über den Freberhaften process und seine Behandlung, Berlin, 1878.—Cl. Bernard, Leçons sur la chaleur animale, p. 422.—Hirtz, article FIEVRE, Dict. Jaccoud.—Catel, De l'urine dans quelques affections fébriles chirurgicales (thèse de Paris, 1874).—Unruh, Rétention des matériaux de combustion incomplète dans la fièvre (Arch. f. path. anat., p. 48, 1869).—Zuelzer, Bemerkungen über einige Verhaltnisse des Stofftreohesels in Fieber und Hungersten (Berl. Klin. Wochens., No. 27, page 381, 1837).

(*b*) Charvot, Température, pouls, urine dans la crise et la convalescence de quelques pyrexies (thèse de Paris, 1871, No. 180).—Hœpffner, De l'urine dans quelques maladies fébriles (thèse de Parfs, 1872).

and Senator maintain that there is not this increase,[1] and the later engaged a few years ago in a vehement dispute with Pfluger *apropos* of the experiments of Colasanti on this subject. The experiments of Liebermeister seem to me to decide this question, and to-day it can hardly be disputed that there exists during fever an augmentation in the exhalation of carbonic acid. Only this augmentation does not show itself except at the commencement of the pyrexia, and does not appear to last as long as the elevation of the temperature.

The examinations of the gases of the blood which have been made by Mathieu and Maljean, while showing us a notable diminution in the respiratory power of the blood in febrile animals, would have furnished a powerful argument to the opponents of the doctrine of exaggeration of combustions in the febrile process, if one did not remember how much the respiratory and circulatory movements are increased in individuals affected with fever.[2] I pass rapidly by the other alterations of the blood, alterations concerning which little is known,[3] only mentioning in this connection the interesting researches of Prof.

[1] Leyden was the first to remark the increased excretion of CO_2 in fever, which increase may attain five per cent. Silujanoff remarked the same increase in animals in which he produced septic fever; but we are indebted to Liebermeister for the most precise observations in the human subject, from which it is demonstrated that the exaggeration of CO_2 corresponds to the rise of the temperature, but does not continue with it, for while the temperature keeps elevated, the production of CO_2 decreases.

Fraenkel and Leyden in their experiments on animals, in which they had determined fever artificially, always observed an augmentation in the production of CO_2; Wertheim, however, claims directly contrary results from his personal observations.

In his experiments on febrile guinea pigs, Colasanti had noted a large increase in the consumption of O, and in the production of CO_2, but in tabulating his results he made allowance for the difference between the temperature of his animals at the time of the experiment and the surrounding atmosphere; admitting, as he properly did, that the temperature of the room exercises a considerable influence on the consumption of O and the excretion of CO_2. *Apropos* of these experiments a long discussion arose between Senator and Pfluger, the first claiming that the correction by Colasanti was inapplicable to the case, the other resolutely maintaining that always in fever there is augmentation of oxidizing processes.

[2] Mathieu and Maljean have always noted in fever patients a remarkable diminution in the respiratory power of the blood. In animals there is the same diminution. Mathieu and Urbian have shown that the proportion of O and CO_2 decreases as the temperature rises. Geppert has examined the gases of arterial blood in animals in which he had produced fever. According to his experiments, the oxygen is not lowered, but CO_2 diminishes proportionally to the elevation of the temperature. He regards this lowering of CO_2 as not the cause, but the effect of the fever.

[3] Legerot affirms that the hæmoglobin of the globules does not possess the property of fixing oxygen during the febrile process. These globules are rendered incapable of administering to the internal combustions.

Richardson also thinks that the septic poisons prevent the absorption of oxygen by the hæmoglobin.

Manassein has observed in the blood of persons affected with fever a diminution of the volume of the red globules, while on the contrary, Laptschinsky and Kelsch have found an augmentation in their volume. (*a*)

(*a*) Geppert, Die Gaze des arteriellen Blute in Fieber (Zeitsch. f. Klin. Med., t. ii, p. 355).—Mathieu et Maljean, Etude clinique et expérimentale sur les altérations du sang dans la fièvre traumatique et dans les fièvres en général (Bull. et Mém. de la soc. de chirurgie de Paris, t. ii, 1876).—Mathieu et Urbain, Des gaz du sang (Arch. de phys., 1872).—Du Castel, Physiologie pathologique de la fièvre (thèse agrég., 1878).

Hayem on what he has described under the name of the "hæmatic crisis in fevers," a crisis characterized essentially by an exaggerated production of hæmatoblasts, coincident with the defervescence of acute diseases, and I come to the physical demonstration, the most clear in my own mind, of the augmentation of combustion in fever. I refer to the application of calorimetry to this study. Liebermeister, and his pupil, Kernig, have thus shown us by precise experiments, that the febrile individual gives forth more heat than the healthy individual.[4]

All these things concur then, as you see, gentlemen, in clearly establishing this first point, that in individuals affected with fever, there is increase of organic combustions. But this does not suffice of itself to explain the febrile process; there must be some disturbance in the regulation and distribution of heat in the economy.

Vulpian has devoted one of the most interesting chapters of his splendid work on the Vaso-Motors, to the study of the distribution of heat in the healthy individual, and in the febrile individual. He has put in clear light, the capital role of the capillary net-work, and the influence of the vaso-motor apparatus in the calorification of different parts of the body, showing us that whenever the capillary vessels of the skin become contracted, there is an augmentation of the central heat with lowering of the temperature, of the skin, and superficial subjacent tissues; when, however, there is dilatation of the cutaneous and subcutaneous vessels, we have elevation of the temperature of the skin and subjacent tissues, and a lowering of the central heat.[5]

It is by virtue of the harmonious reciprocal play of the vaso-motors of the central organs, and of the periphery, that man maintains in the normal state, and under the influence of divers external influences, his temperature at a figure almost unvarying.

These points being once established, we may now approach the critical study of the different theories invoked to explain fever.

[4] There are two processes of calorimetry—that by cold baths and by warm baths. In the process by cold baths, the water of the bath serves as calorimeter; in the other process, the patient becomes his own calorimeter. The experiments of Liebermeister and Kernig have shown that in the human organism heat production is in the ratio of the loss of heat. The more the loss, the greater the production, and vice versa. It is also proved that a healthy man, placed in a cold bath, evolves the more heat the lower the temperature of the bath, and that a feverish person radiates more heat than a healthy person. (*b*)

[5] Vulpian, Lecons sur. l. appareil Vaso-Motor, Paris, 1875, t. II, p. 188.

(*a*) Legerot, Etude d'hématologie (thèse de Paris, 1874).—Richardson, Some new researches on the causes and origin of fever from the action of the septic poisons.—Manassein, De la diminution des dimensions des globules, Tubingen, 1872.—Kelsch, Contributions à l'analyse pathologique (Arch. de phys., 1875).—Laptschinstky, Centralbl., 1874, No. 50.

(*b*) Liebermeister, Die Regulirung der Warmebeldung bei den theiren von constanter temperatur (Deutsch. Klin., 1875, No. 40).—Kernig, Experimentelle, Beitrage zur Kenntniss der Warmeregulirung beim Menschen (thèse inaug., Dorpat, 1864).

When we take a general survey of all these theories, we see that they may be grouped in three principal classes.

In the first class we have the theory of Traube and Hueter; a theory which, while denying all augmentation in the combustions of the economy, explains the febrile process by modifications effected in the capillary net-work.

In the second class, the increase of combustions is admitted, but as an element absolutely secondary, the principal rôle being assigned to the vaso-motors; this is the theory of Senator and Marey.

In the third class the augmentation in the combustions constitutes the dominant principal fact, and according as this increase is explained by a trouble in the regulation of heat production, whether by the influence of certain parts of the nervous system, or by the modification in the vaso-motors, we have the respective theories of Liebermeister, Claude Bernard, and Vulpian. Let us examine each one of these theories.

Traube rejects absolutely the notion of augmentation of combustions, as explanatory of the thermal elevation, which results, he thinks, from the fact that in the period of rigor or chill, the capillaries of the periphery contract under the influence of the vaso-constrictor nerves, and the afflux of blood to the central organs becomes the sufficient and sole cause of the febrile manifestation.

Hueter has gone still further in this direction. Taking his stand on the experiments of Albert, which go to show that the mechanical arrest of the circulation in a vascular tract produces thermal elevation and fever, he maintains that the first cause of the febrile process is a mechanical arrest, by embolic infarctions, of the capillary circulation of the lungs and skin.[1]

These theories raise so many criticisms and objections that they have been virtually abandoned. In fact, they can give no explanation of the rise of temperature which precedes the chill in intermittent fever, and they have no sufficient application to continued febrile processes.

The theories of Senator and Marey are much more seductive; in that of

[1] Albert claims for his experiments a demonstration that arrest of the circulation in a vascular district causes elevation of temperature and fever; the arrest being the result of emboli, which may be purely mechanical, producing fever of an intermittent type, or septic, with fever of a continued type.

According to Hueter, fever is due, not to over-production of heat, but to lessened distribution. The body has two surfaces of refrigeration, the cutaneous surface and the pulmonary surface. The less the quantity of blood flowing through the vessels of a refrigerating surface, the less the loss of heat, and the greater the general temperature. In fever there is arrest of the circulation in these two refrigerating surfaces, and fever ensues, with tumefaction of the spleen, kidneys, and liver. All of Hueter's experiments were made on frogs.

Senator has examined the auricular vessels of albino hares in health and in fever, and has shown that during febrile heat there is no permanent paralysis or tetanus of the vessels. He thinks that the theory of Hueter is not well founded. (*a*)

(*a*) Albert, Untersuchungen über das Fieber (Ber. der natvrwiss. Med. Veruns zu Innsbruck, 1873).—Hueter, Ueber den Kreislaufund die Krieslauffstœrungen in der Froschlunge versuche zur Behandlung einer mecanischen Fieberlehre (Centralb., No. 5, 1873).—Senator, Weitere Beitrage zur Fieberlehre (Centralb., No. 6, 1873, p. 84).

Senator we find the same explanation as that given by Traube of the mechanism of the chills and the totality of the febrile phenomena, but he admits the augmentation in the combustions of the economy resulting from the enhanced destruction of albuminoid matters.[1]

The theory of Marey is very much like that of Senator, but to the initial constriction of the capillaries of the periphery, cause of the central thermal elevation, there succeeds, according to him, a paralytic dilatation of this capillary net-work, which brings the heat to the surface, "leveling down the temperature." In the theory of Marey, as in that of Senator, there is augmentation in the combustions, but this is a secondary matter, the primordial phenomenon and the most important is the trouble brought upon the vaso-motor circulation of the periphery, whether by spasm, as Senator thinks, or by alternations of spasm and paralysis, as Marey thinks.[2]

These theories which are much nearer the reality, do not at the same time give us a sufficient explanation of the elevation of temperature which precedes the chill, and the only theory which is in harmony with all the facts, is in my opinion that of Liebermeister. According to Liebermeister, the augmentation in the combustions plays the principal part; it is admitted without dispute, and you have just seen upon what solid proofs it rests. But it is not enough to admit the fact of increase of combustions; the original cause demands explanation, and here certain opinions come in which we must discuss.

Just here Liebermeister, to whom we owe such important works on this part of general pathology, has furnished us precious data. After having shown us by the examination of the urine, of the expired gases, and especially by the application of calorimetry, that there is augmentation of heat-production in fever, he has well brought to light this other fact that this elevation of itself does not constitute fever, and there is needed besides a modification in the regulation of heat. Let me explain.

In the healthy and normal state of man, you may by artificial processes raise the internal temperature, but as soon as those means cease to act, the

[1] According to Senator the febrile elevation is due (1) to exaggeration of heat-production, arising from increase of interstitial combustions; (2) to retention of heat, by contraction and shrinkage of the cutaneous arterioles. Buss adopts the theory of Senator in explanation of fever, which he considers as essentially constituted by a disorder in the deperdition of heat. This disorder is attributed to the presence of irritant agents in the blood which determine the contraction of the peripheral vessels, and hence, augmentation of heat in the ratio of the lessened deperdition. (*a*)

[2] Marey. Medical Physiology of the Circulation of the Blood. Paris, 1883.

The important part of Marey's theory is the leveling of the temperature, which depends on a paralytic dilatation of the peripheral capillaries. At the commencement of the attack and during the first period, there is constriction of the capillaries of the periphery; the blood is forced into the viscera, and the central temperature rises. Then the cutaneous capillaries dilate, and the blood, at a high temperature, flows to the peripheral parts and distributes its heat.

(*a*) Senator, Untersuchungen über den Fieberhaften process und seine Behandlung (Berlin, 1873) — Buss, Ueber wesen und Behandlung des Fiebers (Stuttgard, 1878).