It is difficult for the residents of the Bitter Root Valley to believe that the tick lives exclusively as a parasite on animals, on account of the widespread and more or less uniform distribution of the arachnid in nature. It is thought it must live to some extent at least on vegetable or on dead insect material. There is as yet no direct evidence to support this suspicion.

It is true that among the foothills the tick seems to be omnipresent, yet the uniformity of its distribution even in these localities is open to doubt. It is not uncommon to learn from a ranchman that more ticks may be found on one hill than another which is in close proximity. Horses and cattle roam practically all the foothills to some extent, especially during the fall and winter. During their residences in such places they collect innumerable ticks, probably in the form of larvæ chiefly. The young ticks grow and moult on the host and when they reach adult form one would be safe in assuming that large numbers drop off, or are rubbed off against bushes and small pines by the animals. A high wind may be an important factor in disseminating ticks, for a ranchman related his experience in driving home one evening in the spring during a violent windstorm. He felt particles striking his face and clothing, and on reaching home found a large number of ticks on his clothing. From my own experience I can state that ticks climb small pines to at least the full height of a man. It is probable that they ascend higher than this, and they are so light that without doubt they could be easily carried by a strong wind for some distance.

It is another fact of importance that the tick has increased greatly in numbers since the valley has been well settled and the quantity of the stock has increased. This is agreed to by all who have lived long in the valley.

CONDITIONS IN THE LO LO VALLEY.

The conditions in the Lo Lo Valley during the past two years are very important. Lo Lo Creek is tributary to the Bitter Root River, and its valley for some years has been considered as badly infected. In 1906 this locality had no infections, and there was hope that the disease had died out there. If the disease is kept alive by ticks which have bitten infected men, there would be some ground to hope that a year without an example of infection in man might result in the disappearance of the disease. If after a year of no infections cases did appear in the following year, three possibilities present themselves in the way of explanations: 1, Certain ticks may live for two years instead of one year, the latter being the usual period of life; 2, the virus may be transmitted through two generations of the tick without its renewal from an infected host; 3, each generation of ticks may acquire the disease afresh each year from some susceptible host other than man (see below). There is not sufficient ground at present for considering a fourth possibility, that the tick becomes infected from decaying organic matter.

As stated, no cases occurred in the Lo Lo Valley during 1906, and but one has appeared in 1907. It is hoped that the first two possibilities can be decided experimentally.

SUSCEPTIBILITY OF THE GOPHER AND OTHER ANIMALS.

The third possibility mentioned above, that spotted fever may be kept alive in a susceptible host other than man, has received some support this year. Assisted by Mr. P. G. Heinemann, I have found that the indigenous gopher or ground squirrel⁵ may be infected by inocula-

tion and that one attack confers immunity to second inoculations. Its susceptibility is much less than that of the guinea-pig and is not perfectly uniform. The disease was again conveyed to the guinea-pig from the inoculated gophers. I do not maintain at this time that the gopher exclusively is responsible for the maintenance of the disease, although this is possible.

The susceptibility of other animals found in the valley is being investigated. Dr. William M. Chowning cooperated with us in the inoculation of a horse, which received about 60 c.c. of blood subcutaneously from infected guinea-pigs. After a brief incubation period, the animal ran a short but rather high course of fever. The horse resisted entirely a second inoculation of 60 c.c. of infected blood from man and monkey, the experiment being conducted by Mr. Heinemann and myself. Assisted by Mr. Heinemann, a second horse was inoculated with 10 c.c. of infected blood from man. No distinct reaction followed. A calf, 2 weeks old, failed to react following the inoculation of over 30 c.c. of blood from infected guinea-pigs.

INFECTED TICKS IN NATURE.

Although the experimental transmission of Rocky Mountain spotted fever by means of the "wood-tick" attracts the gravest suspicion to this arachnid as the agent of infection under natural conditions, the theory of tick-transmission can not be considered as proved circumstantially until infected ticks are discovered in nature. Assisted by Mr. Heinemann,⁵ it has been possible to demonstrate that infected ticks do exist under natural conditions on the west side of the Bitter Root Valley. The ticks were collected from vicinities in which the disease appeared this year, partly from horses and cows and partly from the woods.

A group of thirty-six males taken from the vicinity of Mr. Hickey's ranch produced the disease in guinea-pig 596, which presented a typical clinical course with scrotal hemorrhages, and died in twelve days after the ticks were first attached. The anatomic changes were characteristic of spotted fever in every detail, as it occurs in the guinea-pig, and cultures from the heart, liver and spleen remained free from discoverable bacterial growth.

I take the position that these experiments connect the tick unmistakably with the occurrence of spotted or tick fever of man in the Bitter Root Valley, and that without them the "tick theory" would lack essential support.

A CLINICAL STUDY OF BLOOD PRESSURE VARIATIONS IN DIABETES AND THEIR BEARING ON THE CARDIAC COM- PLICATIONS.*

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The pathologic conditions of the circulation in diabetes and their bearing on the prognosis of that disease have been made a subject for publication by a number of observers, including Jacques Mayer, O. Israel, Schott, von Schmitz, Ebstein and Leonard Weber. My purpose in reopening the subject for discussion is to consider the question from the vascular standpoint and to submit certain observations on blood pressure in diabetes which I have conducted, covering a period of two years.

⁵ The following experiments, with others, will be reported in detail, in conjunction with Mr. Heinemann in the near future.

* Read in the Section on Practice of Medicine of the American Medical Association, at the Fifty-eighth Annual Session, held at Atlantic City, June, 1907.

Reports differ widely as to the condition of the heart in diabetes. There are undoubtedly many cases in which the heart is normal or where the state of the organ corresponds to the general nutritive condition. It would be an amazing exception to the rule of toxic degenerations if the circulatory system could withstand without detriment the irritation of so prolonged and in many cases so intense a toxemia as characterizes this disease, and consequently it is not surprising that in a considerable percentage of cases some degree of cardio-vascular impairment is discoverable.

Saunby states that in 40 per cent. of his cases the heart was free from noticeable changes. In about an equal proportion it was found to be pale and soft, and in 13 per cent. enlarged. O. Israel found cardiac enlargement in 10 per cent. of cases observed. J. Mayer records an average of 21.6 per cent. of cardiac enlargements in his first series and 27 per cent. in his second series. Virchow's autopsy reports yield a total of 13 per cent. cardiac enlargements. The average for these assembled statistics is 17 per cent. cardiac hypertrophy in diabetes. No accurate particulars are forthcoming regarding the age of patients and duration of the disease. Some degree of renal change was a pretty constant accompaniment. In the severe toxic forms of diabetes the renal condition is either a functional hypertrophy or hyaline degeneration of the tubular epithelium. In the chronic milder type of cases the renal alterations are frequently of the nature of chronic nephritis.

In rapid progressive forms of diabetes occurring in young, spare individuals cardiac hypertrophy is seldom encountered, the myocardial changes being fatty rather than hyperplastic. The histologic changes in the heart wall in this type of case are neither pronounced nor definite. The myocardium may be pale and soft, or more rarely show marked fatty changes, with brown atrophy. Glycogen granules have been found between the muscle fibers (Frerichs). Degenerative conditions of the myocardium are probably much more rarely detected during life than after death, unless attention is called to their existence by pronounced symptoms. The acid intoxication and emaciation of inveterate forms of diabetes produce grave cardiac under-nutrition, with enfeeblement of the organ. The supreme result of this cardiac weakness was pointed out by von Schmitz, who described a condition of collapse or syncope from asystole, characterized by a very rapid, weak pulse, cold extremities, but without the peculiar panting respiration which marks true diabetic coma.

In seeking an explanation for the frequent existence of cardiac impairment in diabetes the state of the circulation is of the first importance. The introduction of methods for the clinical estimation of blood pressure furnishes us with the means for determining whether or not this factor is of importance in producing secondary cardiac defects in diabetes in somewhat the same manner as in other chronic intoxications. Very little light has as yet been thrown on this question. Such information as is forthcoming from a study of the literature consists mainly in dogmatic assertion unsupported by adequate statistics. Potain¹ stated in 1902 that the blood pressure is notably high in diabetes, surpassing even that observed in Bright's disease. This statement has found wide currency. Tiessier² states that in his opinion diabetes is one of the three great

hypertensive diseases, classing it in this regard with aortic insufficiency and interstitial nephritis. He qualifies this sweeping statement by excluding the type of lean diabetes, that is so often complicated with pulmonary tuberculosis, in which he states we may observe hypotension instead of hypertension. Vaquez³ reports that he had never found an elevated arterial tension in diabetes. Ott⁴ reports observations on two cases, one with systolic pressure of 225 mm. in an old man with albuminuria and glycosuria; a second in a man of 40 with 6 per cent. of sugar, having a systolic blood pressure of 105 mm. Hensen includes in his records⁵ two cases of diabetes with hypotension. Janeway⁶ states that he has seen both high and low blood pressure in diabetes, the latter in severe cases, with marked emaciation and diacetic acid in the urine, the former, especially in the milder forms of the disease, in stout, elderly people, where chronic nephritis or arteriosclerosis existed.

The foregoing statements and observations practically cover the question as it stands to-day. It is apparent that they do not furnish an adequate basis on which to judge the matter, for besides lacking support from statistical observations, they are conflicting in character and leave the question in all essentials an open one. The observations submitted herewith are offered as a contribution toward its more accurate solution.

My observations cover a series of twenty-five cases of diabetes of different types, at all ages, taking them as they came under treatment for the disease. The majority of the cases were observed in private practice, a few in hospital and dispensary work. The instruments employed were Stanton's sphygmomanometer, with a 12

TABLE OF DATA OF DIABETIC PATIENTS.

Number	Sex	Age	Body Weight Pounds.	Maximum Gly- cosuria per ct.	Acetone Bodies	Albuminuria	Maximum Systolic Blood Pressure mm.	Minimum Systolic Blood Pressure mm.	Average Systolic Blood Pressure mm.	Complications
1	F.	21	134	1.68	0	0	120	108	114	
2	F.	46	113	4.4	0	0	140	130	137	Gallstones.
3	F.	54	185	3.33	0	0	140	140	140	
4	M.	28	136	4.4	+	+	105	95	100	Alcoholism.
5	M.	46	122	3.33	+	+	125	85	100	Carbuncle, neuritis, one attack of epi- lepsy, acetona; Death in coma.
6	M.	56	176	2.75	0	+	156	135	147	Arteriosclerosis, myo- carditis, cerebral thrombosis.
7	M.	24	136	4.	+	+	105	80	95	
8	F.	71	186	1.66	0	0	150	145	147	Arteriosclerosis, card. hypertrophy.
9	F.	50	152	2.	0	0	125	120	122	
10	F.	56	195	6.66	0	+	228	200	210	Chron. neph., card. hypertrophy, hemor. retinitis, gallstone.
11	M.	56	205	1.	0	0	115	115	115	
12	M.	48	207	2.50	0	0	125	120	122	
13	M.	36	135	5.	+	+	110	90	100	Death in coma.
14	M.	29	133	3.75	+	0	102	100	100	
15	F.	35	149	3.	+	0	115	105	110	
16	F.	55	137	3.	0	+	165	140	150	Chron. neph., card. hypertrophy.
17	M.	54	160	3.	0	+	165	130	145	Chron. neph., retin- itis, double mitral lesion, Graves' dis.
18	M.	60	185	1.33	0	0	145	140	142	Arteriosclerosis.
19	M.	76	210	1.66	+	+	173	165	170	Arteriosclerosis, myo- carditis, one at- tack card. syncope.
20	F.	75	160	3.	0	0	140	140	140	Gallstones.
21	F.	21	107	3.	+	0	125	105	115	Graves' disease.
22	M.	13	76	5.	+	0	90	80	90	
23	F.	26	115	4.	+	+	125	80	95	Death in coma. Sis- ter of patient 13.
24	F.	56	150	2.	0	0	170	165	168	Uterine fibroids. Op- eration.
25	M.	46	237	2.50	0	0	110	105	107	Chron. appendicitis. Slight pyrexia.

1. La pression arterielle de l'homme à l'état normal et pathologique, Paris, 1902.

2. Gazette des Hôpitaux, Nov. 8, 1904, 1243.

3. Gazette des Hôpitaux, Nov. 3, 1904, 1221.

4. Montreal Med. Jour., November, 1902.

5. Deutsche Archiv. f. Klin. Med., 1900, lxvii.

6. The Clinical Study of Blood Pressure, 1904, 236.

cm. arm-band for office readings, and Cook's modification of the Riva Rocci instrument, with 9 cm. arm-band, for bedside observations. A total of one hundred and fifty observations was made. In all of the cases except two (Nos. 3 and 22), numerous readings were taken. In many of the cases, especially those of the severe type, it was found exceedingly difficult to secure satisfactory estimations of diastolic pressure with the instruments employed, and for this reason only the records of systolic pressure are included in the table submitted:

SUMMARY OF BLOOD PRESSURE RECORDS.

Number of cases observed.....	25
Males	13
Females	12
Average age of patients	45
Average weight of patients (pounds)	156
Average systolic blood pressure (mm.)	127

Age is the most important factor in influencing the type of diabetes. It is well known that the disease is less severe and progressive in the stout and elderly than in young, spare individuals. The age of 50 years often proves to be the deciding factor between the severe toxic form of the disease and the chronic mild type. In the series under consideration thirteen patients were under 50 and twelve over that age. The average blood pressure of the former was 107 mm. and of the latter 150 mm. Their average weight was 138 for those under 50 years and 175 for those over that age. Age and weight are consequently seen to exert a marked influence in determining the degree of blood pressure. Acid intoxication (diaceturia) was observed in nine of the thirteen cases under 50 years of age and in only one of those over 50. The intensity of the glycosuria was apparently without effect in increasing the degree of blood pressure. Fifteen of the series observed presented a maximum glycosuria of over 3 per cent. The average blood pressure in these cases was 121 mm. In the remaining ten cases the blood pressure averaged 135 mm.

If we are justified in drawing any conclusion from these figures, it is that an inverse rather than a direct ratio exists between the degree of glycosuria and height of blood pressure. This is really no more than we might expect, in view of the fact that the milder types of diabetes are observed in elderly people who are apt to have arteriosclerosis and in consequence higher average arterial pressures.

Perhaps the most interesting point involved in the present inquiry is the effect of the acid intoxication of diabetes on the blood pressure. In my series there are ten cases which developed acetonuria and diaceturia. The toxemia proved to be permanent in all cases except one (No. 19), although it fluctuated considerably in intensity from time to time.

Number of cases with acid intoxication.....	10
Average age	33
Average weight	131
Average systolic blood pressure (mm.)	107
Number of cases without acid intoxication.....	15
Average age	53
Average weight	172
Average systolic blood pressure (mm.)	140

The marked difference in the average values between patients with and without acid intoxication and the apparent disposition to hypotension in the former type of cases suggests a direct depressive influence on the circulation, exerted by the acid toxins in diabetes. Before ascribing any such influence the matter is to be examined further. These patients are invariably asthenic in type and display the general causes of low pressure, i. e., emaciation, malnutrition, mental depression, which produce subnormal values in tuberculosis and the cachectic states generally. To these factors rather than

to the acid toxins direct I believe are to be attributed the falling blood pressure curve.

I cite a condensed abstract of weight, pulse, blood pressure and urine records of two cases carefully observed:

CASE 7.—Age 24. Diabetes first discovered in August, 1905.

Date.	Weight.	Pulse.	Blood pressure, mm.	Urine	
				Glycosuria, per cent.	Acetone Bodies.
Feb. 3, 1906.....	136	78	104	3.33	Absent.
Feb. 6, 1906.....	138	76	105	...	Absent.
Feb. 13, 1906.....	143	72	90	2.50	Traces.
Feb. 21, 1906.....	141	78	100	2.	Traces.
Feb. 24, 1906.....	143	66	90	1.66	Absent.
Feb. 28, 1906.....	141	72	108	2.25	Absent.
Feb. 20, 1907.....	138	63	80	4.	Marked.
Feb. 25, 1907.....	138	70	95	2.85	Marked.
March 1, 1907....	135	78	100	2.50	Marked.
March 4, 1907....	135	80	88	1.50	Marked.
March 6, 1907....	133	80	80	3.	Marked.

In the year elapsing between Feb. 28, 1906, and Feb. 10, 1907, I am informed by patient's family physician, he was free from acid intoxication, and remained in fair activity, following his occupation.

CASE 5.—Age 46. Disease first discovered October, 1905.

Date.	Weight.	Pulse.	Blood pressure, mm.	Urine	
				Glycosuria, per cent.	Acetone Bodies.
Jan. 3, 1906.....	122	90	125	3.33	Traces.
Feb. 10, 1906....	116	90	120	2.	Traces.
Feb. 26, 1906....	121	90	120	1.25	Traces.
March 12, 1906..	120	78	120	1.66	Traces.
March 19, 1906..	122	96	110	2.50	Traces.
April 2, 1906....	121	90	105	1.21	Moderate.
April 23, 1906..	121	84	110	2.	Moderate.
May 7, 1906....	121	90	120	2.	Moderate.
May 24, 1906....	121	78	112	2.	Moderate.
June 10, 1906....	120	72	100	1.66	Moderate.
July 12, 1906....	118	84	110	2.	Marked.
Oct. 9, 1906....	114	76	110	2.	Marked.
Nov. 8, 1906....	114	78	120	2.50	Marked.
Nov. 29, 1906....	110	90	120	2.	Marked.
Dec. 10, 1906....	110	90	100	2.50	Marked.
Jan. 8, 1907....	110	90	110	2.66	Marked.
March 13, 1907..	100	90	105	3.	Intense.
May 21, 1907....	?	96	100	3.	Intense.
June 1, 1907....	Death in coma.				

If these records are carefully examined it will be found that while undoubtedly the blood pressure falls progressively during the stage of acid toxemia, it does so in association with a falling weight curve and increasing weakness—evidences of malnutrition which underly hypotension in chronic nutritive diseases generally. I agree with Janeway in thinking that the progressive emaciation and cardiac weakness cause the hypotension in severe cases of diabetes rather than the acid intoxication *per se*. The point is difficult to determine and perhaps not essentially important, inasmuch as the acid intoxication and malnutrition of inveterate diabetes run hand in hand.

In the tabulated record a note is made of the various complications found in many of the cases. It is of importance to appraise the influence of these various factors on the blood pressure record. In a certain number of cases chronic albuminuria was present, unassociated with diaceturia. These cases number five in all, only one of them displaying a slight and transient diaceturia (No. 19). Without exception these five cases gave definite clinical indications of arteriosclerosis or chronic nephritis. The average blood pressure was 164 mm. In five other cases albuminuria developed in small amount late in the disease, associated with prolonged intense diaceturia (acidosis). In these cases the average systolic blood pressure was 98 mm. This observation would indicate that albuminuria occurring as a result of toxic irritation of the kidneys from acid toxins in the urine and hyperfunction has no effect in increasing the blood pressure. The high blood pressure value of the first group is attributable to the complications causing the albuminuria (arteriosclerosis, chronic nephritis).

The only high blood pressures observed in the series herewith reported, with one exception, were noted in association with arteriosclerosis and chronic nephritis. Four cases (Nos. 6, 8, 18, 19) having marked arteriosclerotic manifestations gave average pressure values of 151 mm. Three cases (Nos. 10, 16, 17) complicated with chronic nephritis averaged 168 mm. One patient with uterine fibroids had a systolic blood pressure of 168 mm.

Nowhere in these records do we find the excessive hypertension declared by Potain and Tiessier to characterize diabetes. The only case displaying excessive pressure is Case 10, a female with chronic diabetes association with gallstones and chronic interstitial nephritis. The high blood pressure in this instance is fully accounted for by the complications. Indeed, if we are to accept the indications of this investigation, complications are the only factors making for high pressures in diabetes, and especially is this true of arteriosclerosis and chronic nephritis, both of which are hypertensive diseases. The average systolic blood pressure of cases with arteriosclerosis was 151 mm., and of those with chronic nephritis was 168 mm. After deducting from the series reported the cases complicated with arteriosclerosis and chronic nephritis, we find the average systolic blood pressure of the remaining eighteen to be 115 mm.—a normal average. It is well known that a strong predisposition exists in chronic forms of diabetes to the development of arteriosclerosis. The frequent association of the gouty and obese diatheses with this class of cases may help to explain this tendency to degenerative arterial change. Obesity or gout pre-existing for years before the development of diabetes may carry the individual well along the road to cardiovascular degeneration before glycosuria is discovered. In some cases there undoubtedly coexists the causes of cardiac hypertrophy from associated chronic nephritis—a not infrequent complication.

Albuminuria associated with diabetes, when due to chronic nephritis or renal sclerosis, shows increased pressure values, although toxic albuminuria due to the acid intoxication of diabetes exerts no such influence. There is nothing to show that myocardial and vascular degenerations of the chronic type of diabetes are essential to that disease. All the evidence points to the fact that the complications, especially arteriosclerosis and chronic nephritis, bring about the cardiovascular secondaries. The cardiac hypertrophy noted by pathologists is attributable to these causes and not to the diabetes *per se*.

In the severe progressive forms of diabetes met with most frequently in young, spare individuals arteriosclerosis and cardiac hypertrophy are not frequent or prominent developments. We have seen that the cardiac condition is one of enfeeblement, the myocardial changes being fatty rather than hyperplastic. Marked hypotension characterizes the arterial condition in this form of diabetes—as low reading as 80 mm. being obtained.

These patients are invariably asthenic, emaciated and depressed, and there is much reason to believe that general adynamia rather than special toxic influences are at work to produce the falling blood pressure values of severe cases of this type. The cardiac condition is a progressive exhaustion, eventuating in certain cases in cardiac collapse. The enfeeblement of the organ under these circumstances merely constitutes a part of the general denutrition and weakness. From this standpoint the myocardial changes and "cardiac collapse" described by von Schmitz in inveterate cases of diabetes can hardly be viewed as essential diabetic phenomena, inasmuch as

we encounter very similar changes and a like mode of death in other severe exhausting diseases, such, for instance, as pernicious anemia and pulmonary tuberculosis. The attempt to establish the existence of certain cardiac conditions peculiar to diabetes—in other words, a "diabetic heart," has on examination little justification. None of the evidence forthcoming from an investigation of blood pressure conditions in this disease lends itself to such a theory.

The blood pressure observations submitted herewith furnish positive confirmation of the opinion expressed by Janeway in his excellent book* that "The disease of itself is without influence on arterial pressure; the occurrence of chronic nephritis or arteriosclerosis or angiosclerosis as a complication explains the hypertension, and the resultant emaciation and brown atrophy or fatty change in the heart causes the hypotension in severe cases."

103 State Street.

LATENT DIPHTHERIA.

A PUBLIC HEALTH PROBLEM.*

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CAUSES OF DIPHTHERIA PREVALENCE.

From the public health viewpoint, diseases are studied in order to prevent their spread. Effective prophylactic measures, based on scientific observations, have caused some infectious diseases to disappear from civilized communities, while others have become comparatively rare. Yet, despite the ever-increasing knowledge as to its bacteriology and methods of propagation, diphtheria still remains a fairly common affection. Why is this?

One reason is that only the well-marked cases are isolated and placarded, while persons suffering from the milder forms of diphtheria are permitted to walk the streets, attend school and frequent public places. Another unguarded avenue of contagion is kept open through the false sense of security given by the rules now in vogue as to disinfection. Fomites, which, as shown by Chapin,† Hill, Weichard and Welch, only rarely contain the germs, are required to be disinfected, while no attention is paid to the virulent bacilli which so frequently are present and persist for long periods in the noses or throats of persons with whom the patient has been in just as intimate contact. The fault lies partly with physicians who do not recognize the latent forms of diphtheria and partly with sanitary officers who fail to seek out and guard as sources of infection all those who are liable to transmit the disease.

THE AUTHOR'S CASES OF LATENT DIPHTHERIA.

I have seen twenty-seven cases of latent diphtheria, most of them in my capacity as assistant medical inspector of the Philadelphia Bureau of Health. I regard as diphtheria any pathologic condition, local or general, due to infection by specific diphtheria organisms, as defined by Williams, and apply the term latent to those forms unassociated with pseudo-membrane.

CASE 1.—J. S., a schoolgirl of about 12 years, presented enlarged and reddened tonsils on April 29, 1904, and was excluded from school for tonsillitis. A culture taken at the time

* Paper read at the fifty-sixth annual session of the Medical Society of the State of Pennsylvania at Bedford Springs, Sept. 11 to 13, 1906.

† Lack of space prevents the usual bibliographic references to journals and other sources of information.