for the visual axis *h-b-c* intersects the visual axis *g-d-c* at *c*, therefore point *c* is one point for the two eyes. Point *l* in the one eye corresponds with point *k* in the other eye, for the visual line *l-b-e* intersects the visual line *k-d-e* at *e*, therefore point *e* is one for the two eyes. Since *l* and *k* are corresponding retinal points it only remains for me to prove that *l* bears the same relationship to *h* that *k* does to *g*. The inscribed angle *c-b-e* is equal to the inscribed angle *c-d-e*, for each is measured by half the arc *c-e*. The angle *l-b-h* is equal to the angle *c-b-e*, for they are opposite angles; and the angle *k-d-g* is equal to the angle *c-d-e*, for they are opposite angles. Therefore, the angle *l-b-h* is equal to the angle *k-d-g*. The angle *l-b-h* is at the center of retinal curvature and is measured by the arc *l-h*, and the angle *k-d-g* is at the center of the retinal curve and is measured by the arc *k-g*. The arc *l-h* must be equal to the arc *k-g*, therefore *l* is related to *h* as *k* is related to *g*. The same truth is shown as to retinal points *j* in the one eye and *f* in the other eye. And this would be true however much the radius of curvature of the one eye might differ from the radius of curvature of the other eye. The similarity of location of corresponding retinal points is in degrees of arc and not in millimeters. What has been proved of *l* and *k* and of *j* and *f* as corresponding retinal points, could be proved of all corresponding retinal points lying in the horizontal retinal meridians. Using the same figure, the proof can be applied to points lying in the vertical meridians. With the two eyes fixed, rotate the plane of the circle on the chord *b-d* as an axis, and thus generate a surface of binocular single vision. As the rotation is effected what were the visual axes (*h-b-c* and *g-d-c*) become indirect visual lines, the retinal ends of which are made to move in the vertical retinal meridians. Elevating this plane in front 5 degrees depresses it behind 5 degrees; elevating it in front 10 degrees depresses it behind 10 degrees, and so on for any extent of rotation. The retinal end of the visual line *h-b-c* and the retinal end of the visual line *g-d-c* are made to describe arcs of equal degree. What is true of corresponding retinal points in the two horizontal and the two vertical retinal meridians must be true of corresponding retinal points in any two oblique retinal meridians that are similarly related to the vertical and horizontal meridians. Therefore, I conclude that Helmholtz taught the truth concerning corresponding retinal points. Correct as to corresponding retinal points, Helmholtz was in error concerning the normal relationship of the vertical retinal meridians to each other. He should have known that, in accordance with the supreme law of corresponding retinal points, the vertical axes of the eyes, which lie in the planes of the vertical retinal meridians, must be kept parallel with each other, except in cases of non-symmetrical oblique astigmatism. Except for the oblique muscles, the vertical axes of the eyes would not always be correctly related. The law relating to the oblique muscles is that they shall compel the vertical axes of the eyes to remain parallel with the median plane of the head, regardless of the position of the point of fixation in space.

DR. GEORGE T. STEVENS—I think that every one who has been a student of physiologic optics recognizes the diagram which has just been thrown on the screen by Dr. Savage as the familiar diagram of Müller. It has figured in text-books for many years. As a practical horopter it was and is an impossibility. It is hardly worth while to discuss the merits of Müller's horopter, which was rejected by experts long ago. Nor has Helmholtz' horopter been any more satisfactory. Dr. Savage has told us that there is no torsion in the changing relations of the eyes except such as results from defects. If this were true, if there are no physiologic torsions, we must abandon the law of Listing. That law has stood for many years and is likely to remain for still many more years. According to that law, in the example which I have chosen, with a depression of 35 degrees and a lateral angle of 5 degrees each eye, there must be torsion of 3 degrees or there will not be single vision. We shall never abolish Listing's law nor shall we find the eyes unchanged in their meridianal relations by their various adjustments. Dr. Savage tells us that he some time since pointed out that the doctrine of Helmholtz relating to the meridians was incorrect. That was not difficult, since I had demonstrated the fact clearly, but it had not been demonstrated until the clinoscope was introduced, when immediately the error was clearly shown.

DENGUE IN THE ISTHMIAN CANAL ZONE.

INCLUDING A REPORT ON THE LABORATORY FINDINGS.*

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AND
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Of the several tropical diseases whose etiology is yet in question, dengue undoubtedly stands at the head of the list in importance. It has long been considered an infectious disease, as the writings of Ornstein,[1] Eulenberg,[2] Graham[3] and Hirsch[4] attest, while we find Leichenstein,[5] Malie,[6] von Duringe[7] and a half dozen others presenting almost indisputable evidence that it is contagious, in the technical sense of the word, as well.

TABLE 1.—Differential leucocytic count in dengue, showing changes in various stages of the disease.

Stage of Disease.	Poly-nuclears.	Large Mono-nuclears.	Trans-itionals.	Small Mono-nuclears.	Eosino-philes.	Mast Cells.
On admission	32.2	7.9	36.6	23.	0	.3
First drop	41.	5.4	6.7	46.8	0	0
Quiescent period	40.6	6.3	5.8	47.	.3	0
Second rise	50.8	7.6	5.2	33.6	4.	0
Second drop	52.5	5.9	5.4	25.1	11.	0
Convalescence	50.	5.3	4.2	30.3	9.7	0

McLaughlin[8] of Texas, in an exhaustive series of studies begun as far back as 1885, undoubtedly establishes the clearest claim to the discovery of the causative agent, but in spite of his clear and concise description of the method of procedure employed, very few investigators have been able to verify the presence in dengue blood of the micrococcus which he found in such large numbers.

Our lack of exact knowledge concerning the morbid anatomy of this infection is principally due to the extremely low mortality, the death rate being 1 per cent. (Hare) or less.

TABLE 2.—Differential leucocytic count in dengue, showing changes in various stages of the disease.

Stage of Disease.	Poly-nuclears.	Large Mono-nuclears.	Trans-itionals.	Small Mono-nuclears.	Eosino-philes.	Mast Cells.
On admission	52.4	3.8	7.1	36.3	.4	0
First drop	54.	3.9	6.4	34.3	.3	.1
Second rise	69.4	6.9	5.6	17.8	0	.3
Second drop	52.1	6.9	2.4	38.2	.3	0
Convalescence	33.1	6.3	9.3	45.9	3.	0

Consequently we are forced to depend, in a great measure, on examinations of the blood and excreta for information concerning the pathologic changes which are occurring.

During the past spring and summer, while stationed on the Isthmus of Panama, it was our good fortune to be brought into intimate contact with some 200 cases of the disease, and, although no startling discoveries were made, the laboratory findings may serve to direct some future investigator toward the true cause.

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* A preliminary report appeared in THE JOURNAL, Dec. 17, 1904, p. 1869.

1. Zur Dengue, Frage, Deutsche med. Wochenschrift, 1890.
2. Realencyklopedie, 1886, v.
3. Journal of Tropical Medicine, July, 1903.
4. Chrysochoos in Smyrna.
5. Nothnagel's Specielle Pathologie und Therapie, No. 4, vol. i.
6. Diction. Encyclop. des Sciences Medicale.
7. Monatschrift für praktische Dermatologie, 1890.
8. New Orleans Med. and Surg. Jour., 1885, n. s., xiii; THE JOURNAL A. M. A., 1886, vi; Boston Med. and Surg. Jour., 1886, cxiv; Texas Med. News, 1895-6.

The results from only typical cases are given; in those instances where there was an abortive second rise or if there was the slightest doubt concerning the possible presence of malarial organisms, as evidenced by periodicity, the microscope or the effect of quinin, the record was not used. The selections could be carefully made as all laboratory data for every patient admitted to the hospitals at both Bos Obispo (after the identity of the disease became fully established) and Empirado was systematically placed on file from the time of admission until convalescence was well established.

In spite of the fact that repeated searches were made for micro-organisms in the blood, such as those described by McLaughlin, various stains and methods (Wright, eosin and methylene blue, Ehrlich's tri-acid and Romanowsky-Nocht) being employed, at no time was their presence demonstrated.

Occasionally the somewhat striking refractive changes incident to "stippling" (Schuffner) are somewhat puzzling, but it is hardly possible that this condition could have been mistaken for minute organisms by previous observers.

Our first blood examinations were all made from fresh specimens, encircled by vaselin rings to prevent evaporation. The use of unstained blood in searching for the plasmodium of malaria is not to be compared in value to the results obtained when reliably stained mounts are employed.

The non-pigmented, piroplasma-like bodies (?) seen by Graham were frequently found in many of the unstained specimens. Inasmuch as they were also present in blood from a large number of cases of epidemic catarrh examined while on board the U. S. S. *Illinois* at Newport News, some two years ago, it is probable that they are not foreign bodies, but that they are simply the result of necrobiotic changes within the red cell. A stained specimen from the same subject, no matter how careful the technic employed, invariably failed to show anything.

But little was learned regarding the mode of conveyance of the infection owing to the comparative rarity of the anopheles we were compelled to make use of various species of the culex:[9] *C. consobrinus* (Desv.) *C. stimulans* (Walk.), *C. tarsalis* (Coq.), and *C. taeniorhynchus*, the *Conchyliastes posticus* (Wied.) and the *Stegomyia fasciata*.

Young but well-grown insects of the above genera

9. Howard's classification of species is used.

TABLE 3.—Showing urinary findings at various stages of dengue.

Case.	Stage of Disease.	Color.	Specific Gravity.	Albumin.	Reaction.	Phosphates.	Chlorids.	Sulphates.	Urea.
1.	First drop	Light amber	1.032	no	acid	23.	13.	1.	1.404
	Second drop	Amber	1.015	no	acid+	10.	3.3	.5	1.026
2.	First drop	Amber	1.030	no	acid	20.	9.	1.	1.566
	Second drop	Dark straw	1.032	no	acid	16.	3.	1.	1.62
3.	First rise	Dark amber	1.030	no	acid	18.	9.	3.	1.728
	Convalescence	Straw	1.014	no	acid	6.	5.	1.	1.08
4.	First rise	Light straw	1.020	no	acid	10.	13.	1.	1.392
	Second rise	Amber	1.010	no	acid	8.	5.	.5	.977
5.	First drop	Amber	1.022	no	acid	12.	8.	.5	2.052
	Second drop	Amber	1.018	no	acid	10.	5.	1.	.648
6.	First rise	Light amber	1.020	no	acid	10.	6.	.5	4.32
	Second rise	Amber	1.032	no	acid	20.	10.	1.	1.638
7.	First drop	Reddish	1.026	no	acid	10.	4.	2.	1.756
	Convalescence	Reddish	1.020	no	acid+	11.	5.	.5	2.511
8.	First drop	Amber	1.030	no	acid	20.	5.	1.	2.862
	Second rise	Dark straw	1.022	no	acid	10.	5.	1.	2.322
9.	First drop	Dark amber	1.036	no	acid	5.	5.8	.2	1.646
	Second rise	Dark amber	1.026	no	acid	20.	15.	1.	3.074
10.	First drop	Dark straw	1.028	no	acid	15.	10.	1.5	2.886
	Quiescent stage	Amber	1.022	no	acid+	17.	7.	1.5	2.74

TABLE 4.—Showing number of red and white corpuscles, with hemoglobin percentage and differential leucocyte count at various stages of dengue.

Case.	Stage of Disease.	Red.	White.	Hb.	Polynuclears.	Large Mononuclears.	Transitionals.	Small Mononuclears.	Eosinophiles.	Mast Cells.
1.	First rise—top	5,385,520	3,132	100+	31.2	7.9	37.	23.3	0	0
	Second rise—top	4,425,000	3,156	100	29.4	6.3	7.	51.8	3.1	0.3
2.	First rise—top	5,040,000	5,422	100	73.5	1.3	9.4	17.1	3.2	0
	Convalescent	4,604,000	5,154	95	30.5	6.6	12.	40.	12.	0
3.	First rise—top	4,056,000	4,433	100	83.5	2.1	1.2	7.1	.7	0
	Second rise—top	5,040,000	1,866	100+	41.5	2.9	7.9	24.7	22.9	0
4.	First rise—top	4,395,510	6,422	100	73.8	6.4	2.2	16.7	.8	0
	Second rise	5,320,000	8,310	100+	34.4	1.9	9.	52.3	2.4	0
5.	First rise	3,875,000	2,488	100	50.7	7.4	5.1	36.8	0	0
	Second rise—top	5,904,000	4,022	100	34.9	8.8	3.3	7.2	7.2	0
6.	First rise	4,557,776	3,444	95	50.	5.2	6.9	36.3	1.6	0
	Second drop—beginning	5,266,900	7,778	100	30.9	10.2	9.2	45.3	4.4	0
7.	End first drop	5,172,228	4,800	100+	39.7	9.5	5.	44.9	.9	0
	End second drop	5,734,000	4,320	100	26.5	9.1	6.5	57.4	.5	0
8.	First rise—top	5,776,000	3,688	100	44.8	5.3	6.9	40.9	1.9	0
	Second rise	5,104,000	4,266	100+	30.	5.6	6.9	52.8	3.8	.9
9.	First rise—top	5,532,000	4,111	100	65.3	6.1	8.	19.5	.5	.3
	Second rise—top	5,040,000	3,622	100+	51.9	6.6	5.	33.5	4.	0
10.	First rise—top	4,152,000	4,344	95	69.4	6.9	5.6	17.8	0.	.3
	Convalescent	4,504,000	3,977	100	38.1	6.3	9.3	43.9	3.3	0
11.	First rise—top	4,942,216	3,622	100	62.5	4.2	8.8	24.3	.2	0
	Second rise	5,320,000	5,510	95	38.7	5.2	9.2	44.2	2.7	0
12.	First drop	4,640,000	4,210	100	40.	5.	8.8	45.7	.4	0
	Second drop	5,693,332	3,866	100+	40.3	3.6	8.9	47.	.2	0
13.	First drop	5,306,664	5,311	100+	42.3	4.2	6.9	46.5	0.	0
	Convalescent	5,404,400	7,310	100	23.2	7.2	6.2	51.4	12.	0
14.	First rise—top	5,040,500	3,576	95	61.3	2.4	9.2	25.8	.6	0
	Convalescent	5,404,000	5,866	100	49.9	4.4	5.2	31.1	9.4	0
15.	First rise	4,351,610	3,110	100	58.5	5.5	7.8	37.9	.3	0
	Convalescent	4,408,888	4,288	95	30.	5.2	6.8	53.	5.	0
16.	First drop	5,440,000	3,954	100	56.4	4.7	7.	31.	.9	0
	Second drop	4,524,440	3,266	100	53.6	5.8	4.5	24.	12.	0

were taken from the breeding jars and allowed to feed on dengue patients. Each group was then confined in a separate cage for periods of time varying from five to fourteen days, then taken out and carefully dissected on a glass slide.

The stomachs and salivary glands were then examined, both directly and after being treated with a solution of sodium chlorid and various simple stains. No bacteria, piroplasma or zygote forms were found.

The inoculation experiments were unsatisfactory and open to criticism, for the volunteer subjects were not only exposed to the bites of other mosquitoes at any time, but they were also brought into almost daily contact with dengue cases.

CASE 1.—Bitten by *Culex tarsalis* and *Culex consobrinus* that had been allowed to feed on a 4-day dengue patient 10 days previously. No fever appeared in 14 days.

CASE 2.—The experiment was repeated with similar virgin insects that had fed on first, second, third and fifth day patients and that had then been allowed to remain in isolation for from 8 to 14 days. The man bitten by the fifth-day mosquito was taken down with typical dengue 6 days later.

CASE 3.—Bitten by a similarly infected stegomyia which had been isolated 14 days previously. Patient, fourth day. An attack of dengue followed, but not until two full weeks had elapsed.

CASE 4.—The same procedure was carried out with a young *Culex stimulans* which had been infected 11 days prior to the experiment from a 3-day case. No infection resulted.

Other sources of a possible origin for the epidemic were investigated. Scheube[10] and Clayton[11] have fully covered the essential meteorologic factors in a few brief phrases: high temperature, moisture, low levels, lack of air space, dirt and overcrowding.

So many cases had catarrhal symptoms of the respiratory tract (bronchitis, pharyngitis, tonsillitis and coryza) that it seemed to be more than a coincidence and strongly suggested the epidemic fevers of overcrowding so frequently seen on shipboard.

The striking resemblance to influenza is also seen in the leucopenia and the selective action of the toxin on the nervous system. Many who did not complain of pain or soreness in the throat had marked congestion, with white patches, or even ulcerated areas, of the pharyngeal mucous membrane, and the peculiar putrefactive odor observed in cases of epidemic catarrh was characteristic of these cases also.

Swabs from eleven throats were examined simply by rolling on a slide and, after fixing, staining with Loeffler's solution of methylene blue, to determine the predominating bacteria present. More than the usual number of leptothrices and streptothrices were seen and many groups of tiny diplococci, free or in epithelial cells.

In preparations from the pus in a case of tropical bubo, occurring as a sequel to dengue, the small diplococci were again found in large numbers, and were seemingly the only organisms present. The increase of small, mononuclear lymphocytes points to an invasion of the lymphatic apparatus.

Unfortunately, our outfit for making bacteriologic cultures was incomplete, and no efforts at either isolation or cultivation were possible.

It would be extremely interesting to carry through a series of cultures, especially on blood serum and blood-streaked agar, from the blood, enlarged glands and throats of typical uncomplicated cases.

10. Diseases of Warm Countries.
11. Encyclopædia Medica.

The appended table of urine examinations shows decided increase in phosphates. They often appeared as a heavy cloud in freshly voided specimens. No experiments were made at the time to see if clear urine would have resulted from the previous internal administration of acids. The amount of urine passed in twenty-four hours was decreased. Albumin was hardly more than a trace when present. Two cases in which it appeared were atypical ones in the early part of the epidemic.

The estimation of phosphates, sulphates and chlorids was made by the use of the centrifuge. The urea was measured by the modified Davy's method and is open to criticism, as the low percentages of the first group were probably due to lime employed not being so good as that obtained later.

The urine in 122 cases of dengue gave albumin in six instances, about 4.9 per cent. In 41 cases of malaria it was found five times, or 12.2 per cent.

Our conclusions are as follows:

1. Dengue is one of the few fevers in which a leucopenia persists from the first.

2. Blood examinations are of great value in differentiating between malaria and dengue. Even though no parasites be found, a slight leucocytosis with decided increase in the percentage of large mononuclears and transitionals is indicative of the former, while a leucopenia, with a normal differential leucocytic count or varying degrees of a small mononuclear lymphocytosis and a marked eosinophilia late in the disease, is characteristic of the latter.

3. Albuminuria is seldom seen in an ordinary attack of dengue, and then only in small amounts. The exact opposite is true of yellow fever.

4. The period of convalescence in dengue is almost invariably ushered in by a pronounced small mononuclear lymphocytosis which persists for several days.

5. It is suggested that the causative agent is a small diplococcus or a delicate bipolar staining bacillus closely resembling Pfeiffer's organism. It is probably transmitted by the respiratory tract, and its virulence is much increased by the presence of the essential meteorologic factors and by overcrowding.

Clinical Report.

BILATERAL FACIAL PARALYSIS,

WITH SENSORY AND REFLEX DEFECTS, POSSIBLY DUE TO LA GRIPPE.*

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This case was referred to me by Dr. J. C. Oliver of Cincinnati, with request for examination and opinion.

Patient.—The subject of the conditions here reported was a teacher, 51 years of age, married, and the father of three healthy children, aged, respectively, 20, 18, 11.

I saw him at the Presbyterian Hospital, Cincinnati, in March, 1904. He was about 5 feet 10 inches in height, well developed, fairly nourished, of dark complexion and hair. Not evidently anemic. Family and personal history good.

Present Illness.—His chief complaint was of headache, which was located in the occipital and both mastoid regions; it was dull in character and had persisted since its onset, five weeks before. He also complained of inability to close either eye, which was evident.

The onset of his trouble he attributed to an attack of al-

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