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THE CALCUTTA JOURNAL OF MEDICINE:

A MONTHLY RECORD OF THE MEDICAL AND AUXILIARY SCIENCES.

तदेव युक्तं भैषज्यं यदारोग्याय कल्पते।
सचैव भिषजां श्रेष्ठो रोगेभ्यो यः प्रमोचयेत॥
चरकसंहिता।

That alone is the right medicine which can remove disease;
He alone is the true physician who can restore health.
Charaka Sanhitá.

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MILKMAID MILK

EVERY
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THE
CALCUTTA JOURNAL
OF
MEDICINE

Vol. xxvi.] August 1907. [No. 8.

MEDICAMENTS OF LEUCORRHŒA.

By Dr. Chiron.

(Continued from p. 274).

Ignatia. Flow white, puriform, acrid, gnawing, preceded by uterine cramps.

Iodium. Leucorrhœa extremely acrid, gnawing and corroding the skin and linen. Females slender, and delicate. Strumous antecedent, great heaviness of the body and *excessive palor.* Chronic congestion or inflammation of the uterus and ovaries (right side has the preference). Induration of the uterus. Carcinoma in the neck of the uterus.

Kali Ars. Leucorrhœa abundant, acrid, brown, very corrosive. Uterine pain. Great feebleness with perspiration at the least movement. Melancholic character.

Kali Bichrom. Leucorrhœa yellow, viscous, thick, coming out in long filaments. Painful weakness in the loins and pressure in the epigastrium. Painful sensibility in the vagina as if the vaginal mucous membrane is sensitive. Itching of the vulva. Dyspepsia and habitual constipation. Uterine subinvolution. Particularly indicated in fat females, with light blonde hairs.

Kali Carb. Mucous leucorrhœa with pruritus and burning heat in the vulva. Leucorrhœa yellowish, acrid, corrosive.

Acute painfulness; cutting across the lumbar region, extending to the extremities. The menses have sharp smell and are very acrid, excoriating the thighs with great soreness of the back and pricking pain in the abdomen. Chronic metritis. Anæmia in old, fatty and lax females.

Kali Ferrocyan. Leucorrhœa purulent, abundant, yellowish, creamy, non-irritant. Inferior part of the abdomen is sensitive to pressure. Menstruation retarded.

Kali Iod. Leucorrhœa coming in small quantity, acrid, corrosive, with itchiness of the vulva and sensation of excoriation of the pudendum. Flow white, milky, or resembles smegma. Leucorrhœa yellow or green of a putrid odour. Flow of mucus from the vagina. Coryza acrid and violent from slight cold. Diarrhœa with pain in the sacrum. Scrofula. Secondary syphilis. Chronic rheumatism. All symptoms aggravate by rest and ameliorate by movement.

Kreasotum. Leucorrhœa whitish, not painful, flowing as menses and preceded by pain in the loins with heat of the face. *The menstruation which has since terminated, reappeared without cause, therefore it proves injurious that it came far in advance.* Leucorrhœa milky, acrid starches the linen and spots yellow. Flow having fleshy colour and bad odour. Flow of blood and mucus, when she raises herself. Leucorrhœa acrid, fœtid, ichorous, corrosive, with violent pains of pricking, burning and excoriating in the pudendum, between the lips and thigh. Sometimes abundant flow of black and coagulated blood. Frequent desire to urinate, preceded by white flow which colours the linen yellow. Inefficacious desire to urinate, when it is satisfied, it is accompanied by coldness and milky leucorrhœa. Great feebleness; least effort causes profuse sweat. Young blonde girls, very tall, of melancholic and irritable disposition.

Lac Caninum. Leucorrhœa flowing only during the day, aggravates by walking and in standing position. Flow whitish and aqueous which increases at the time of the menses. Pain in the loins without a fixed position passing from one side to the other.

Lachesis. Leucorrhœa preceding menses, abundant, mucous, *stiffens the linen and colours it greenish.* Leucorrhœa with irregular menses, very feeble, and of short duration. The patient can not bear any pressure, even of his cloth on the uterine region. Great prostration, particularly after an effort or an exercise. The patient finds herself miserable in the morning she awakes or at night if she rises; during the night she feels chilly in bed; during walk she often complains of flushes of heat. Uterine congestion with prolapsus uteri. Excoriation in the neck of the uterus; the neck of the uterus is very sensible to touch and bleeds easily.

Lilium Tigrinum. Leucorrhœa abundant, brownish, clear yellow often excoriating, spots the linen brown. Leucorrhœa with sensation of pressure towards the base of the uterine region, increases from mid-day to mid-night, afterwards diminishes till the following mid-day. Sensibility in the hypogastric region. Leucorrhœa abundant and acrid after cessation of the menstrual flow. Loss of appetite. Painful urination. Uterine displacement, particularly flexion and prolapsus due to relaxation. General debility.

Lycopodium. Leucorrhœa abundant comes at interval. Itchiness increases at each menstrual period. Pricking pain from the right to the left side of the abdomen. Gurgling in the left illiac fossa. Sensation of fulness after eating; first course; abdominal fermentation.

Magnesia Carb. Chronic leucorrhœa. Flow clear white, slightly abundant; flow especially after mid-day, during walk and in the sitting position. Pinching around the umbilicus. Uterine cramps. Itchiness at the pubis. Flow acrid, mucous, preceded by colic.

Magnesia Mur. Leucorrhœa abundant, flows especially after stool or in the morning after passing urine, also after bodily exercise. Uterine and abdominal cramps. Flow white, thick in the interval of the menstrual epochs and followed by sanguinolent discharge. Abundant vaginal flow of thick aqueous mucus. Bruised pain in the loin and hip.

Magnesia Sulph. Leucorrhœa burning particularly during movement. Leucorrhœa abundant thick as the menses with bruised pain in the lower part of the back and thigh.

Mercurius Sol. Leucorrhœa worse at night (from 8 to 10 p.m.), prurient, burning, corrosive, with flakes of mucus or purulent discharge. Leucorrhœa of purulent aspect with ulceration of the internal and external parts. Buttons or tubercles in the labiæ. Urine of high colour deposits sediment resembling starch or chalk. Scorbutic appearance of the gum. Tonsils increase in volume.

Mezereum.—Leucorrhœa chronic, corrosive, malignant, resembles the white of egg. Violent irritation of the vagina. Blennorrhœa of the urethra with pricking and burning during urination. Uterine ulcer with sensation of burning, smarting and pricking. Albuminous flow sometimes tinged with blood by the vagina. The menstruation comes as a fortunate event and lasts a long while with leucorrhœa and prosopalgia. Anal prolapsus. Constipation.

Murex Purp.—Leucorrhœa greenish or sanguinolent *only during walk* alternating with mental symptoms and pain in the sacrum. Abundant leucorrhœa with sensibility of the uterus. Menstruation irregular, abundant frequent with clots. Great desire, excited. *Violent sexual excitement provoked by slight contact with the parts.* Nymphomania.

Muriaticum Acid.—Ulceration of the genital parts with emission of bad odour. Leucorrhœa with pain in the back. The menses are in advance. During the menses, pain in the anus.

Natrum Carb.—Flow white, preceded by colic or intestinal pains, day and night; flow white, thick, or yellowish, comes during urination or after passing urine. Menses late, rare resembling the washing of meat. Aversion to mankind, irresistible sleep; weak digestion; great fatigue after little exercise. Induration of the neck of the uterus.

Natrum Mur.—Nocturnal leucorrhœa. Leucorrhœa excessive, abundant, mucous, white, transparent and thick. Acrid leucor-

rhœa with itchiness and smarting pain in the vulva and crampy pain in the abdomen. Leucorrhœa with headache, colic and mucous diarrhœa. Flow yellowish or greenish, particularly during walk in the morning. Uterine prolapsus with sensation of soreness in the urethra after passing urine. Itching in the vulva. Menses irregular, ordinarily abundant. Suppressed menstruation. Aversion to sexual connection. Irritable humour after sexual connection.

Natrum Sulph.—Leucorrhœa at the end of the menses, accompanied by great weakness. Bleeding from the nose during menses which are abundant and acrid. Flow mucous, acrid, corrosive in the vagina. Herpetic vulvitis. Blennorrhagic leucorrhœa yellowish green. The back seems to be broken. Oppression on walking.

Nitricum Acid.—Leucorrhœa after the menses, flesh-coloured, green, or fœtid or filandrous and corrosive. Violent itching of the sexual parts; it makes its presence always towards night, sometimes on walking. Acute pain in the vagina from below upwards. Cold aggravates the leucorrhœa and pruritus. Epistaxis at night. Menses in advance, abundant and resemble dirty water. Syphilitic ulceration with tendency to rapid destruction of tissues, irregularly formed, and of fatty or greenish colour with corrosive flow. Swelling of inguinal glands. Mercurio-syphilitic inflammations; condyloma.

Nitrum.—Leucorrhœa clear, white, stiffens the linen, with breaking pain in the loins.

Nux Moschata.—Vicarious leucorrhœa in the place of menses. Leucorrhœa bloody and muddy. Leucorrhœa with prolapsus of the vagina and uterus. Females always awake with dry tongue. Hysteria. Globus hystericus. Weakness with palpitation of the heart followed by sleep. Physometry.

Nux Vomica.—Leucorrhœa fœtid, spots the linen yellow. Flow white, mucous. Sensation of pressure in the neck of the uterus. Prolapsus of the uterus and vagina. Constipation. Dysmenorrhœa with pain in the sacrum and constant desire for passing stool. Urine frequent; urine deposits brick-red sedi-

ment at the bottom of the vessel. Menses in advance and of long duration.

Palladium.—Leucorhœa glairy, transparent, resembles gelatine, worse before or after menses. Pain and feebleness as if the uterus is coming lower down. Pain in the back and hips with coldness of the extremities. Pain and swelling of the right ovarian region. Acute pain as of thrust of knife in the uterus, diminishes after stool and sleep.

Petroleum.—Leucorrhœa abundant, albuminous, with voluptuous dream, burning in the genital organs, with flow of a small quantity of blood. Menses in advance.

Phosphorus.—Leucorrhœa inflammatory, smarting, corrosive, gnawing and vesicating. Leucorrhœa milky. Flow white in the place of the menses. Painful sensation of feebleness in the abdomen; constipation; stools knotty and difficult; patient tall slender and of phthisical constitution. Sensation of heat in the inferior part of the back; vertigo on rising in the morning. Amenorrhœa with chlorosis; consequence of masturbation. Nymphomania. Metritis.

Phosphoricum Acid.—Leucorrhœa after menses, abundant, yellowish and itching, onanism with all its consequences. Great weakness with complete indifference to all things. Ulcer of the uterus. Flow abundant, putrid, sanguinolent with pruritus. Uterus sensitive. Nervous feebleness with chilliness and profuse sweat. Menses in advance and abundant with pain in the liver.

Phytolacca.—Leucorrhœa thick tenacious, irritant, exclusively found in the neck of the uterus. Painful menstruation accompanied by erosion or ulceration of the neck of the uterus.

Platina. Leucorrhœa periodical, *aqueous only in the day,* flows especially after passing urine or on rising from his seat. Leucorrhœa before or after menses which are profuse and occur frequently. Voluptuous itching of the genital organs with anxious oppression and palpitation of the heart. Great sensibility of the genital organs. Vaginismus. Nymphomania.

Medicament perfectly agreeable to females with dark hair and rigid fibre.

Podophyllum. Leucorrhœa mucous, thick, transparent with sensation of pressure in the base of the uterine region. Prolapsus of uterus. Suppression of menses. Patient nervous, feeble, miserable and capable with difficulty of anything whatsoever.

Psorinum. Leucorrhœa of insupportable odour, very thick, with enough pain in the loins and great weakness. Breast swollen and painful. Sycosis.

Pulsatilla. Leucorrhœa aqueous acrid and burning or milky, *sometimes without colour,* sometimes with swelling of the vulva and flows especially after the menses. Leucorrhœa mucous, thick and white, especially when lying or before and after menses with abdominal colic. Leucorrhœa and symptoms of hysteria with great sexual excitement caused by masturbation. Leucorrhœa of young women at the time of puberty or when the menses are suppressed from cold water or by exposure to cold and humid atmosphere. Female patients, chilly, weep for nothing, without thirst.

Ranunculus. Flow which is at first mild becomes afterwards acrid and corrosive.

Ruta. Leucorrhœa acrid and smarting after the cessation of the menses.

Sabina. Leucorrhœa gelatinous, thick, yellowish, sanious, fœtid with pruritus, corrosive, excoriating the skin of the thighs, with drawing in the lower part of the back. Leucorrhœa during pregnancy and confinement with pricking and pain in the thigh. Leucorrhœa accompanied by suppression of menses. Leucorrhœa exists only a little during the day preceding or following the menses. Inflammation of the ovary or uterus after abortion. Increase of sexual desire. Pricking pain in front and behind the vagina.

Sanguinaria. Endometritis with polypus or ulceration of the neck of the uterus, accompanied by abundance of hæmorrhage with leucorrhœa which is fœtid, corrosive and brown. Leucor-

rhœa follows the menopause, at that time the menses have not completely stopped.

Sanicula. Leucorrhœa with the smell of brine. Sensitiveness of the uterus. Pressure in the lower part of the abdomen as if all the organs of the base will escape by the vulva. Desire to support the parts.

Sarracenia Purp. Leucorrhœa aqueous or milky, thick, white with spasmodic pain in the uterus. Pulsating pain in the uterus as if there is tumour or dropsy; uterus swollen as if full of cysts particularly on the right side; neck of the uterus swollen and hot. Miliary eruption and heat in the vulva.

Sarsaparilla. Flow white mucous and profuse during walk. Pain after passing urine; urine deposits grey concretions. Before menstruation, humid eruption in front of the sexual organs. Menses late and slightly abundant.

Secale Cornut. Leucorrhœa offensive brownish, resembles cream. Liquid brown, flow arises from various morbid state of the neck of the uterus or of the organ itself. Gelatinous leucorrhœa alternates with metrorrhagia particularly in slender females who suffer from excessive menstruation and prolapsus. Burning pain in the uterus which is sensitive to touch. Cancer and gangrene of the uterus. Cachexia. Numbness and coldness of the skin.

Senecio. Leucorrhœa in short females, with headache, insomnia and urinary troubles. Afterwards the menstruation commences; the thoracic and vesical symptoms ameliorate. Menses retarded, suppressed.

Sepia. Leucorrhœa with great itching of the vulva or vagina, or pricking in the uterus, which regularly comes before or after the menses, and does not continue for a long time. The flow is thick and yellow or mucous, aqueous, milky or greenish. Milky leucorrhœa only during walk. Leucorrhœa worse after coition. Constipation with knotty and difficult stool, slowly evacuated and with great effort. Pressure in the anus. The urine is sometimes of a putrid odour and presents deposit which resembles reddish argillaceous clay difficult to detach from the

vessel. Painful sensation and continuous emptiness at the pit of the stomach. Gonorrhœa after the acute period; induration of the neck of the uterus and of the organ itself; retroversion. Prolapsus of the uterus. Well suited to children, weak and debilitated females, with dark hair, with fine and delicate skin, and who are extremely impressionable.

Silicea. Leucorrhœa acrid, coming out at intervals and at times preceded by colic around the umbilicus. Flow white, aqueous, with violent pruritus or flow especially after passing urine. Flow of a quantity of white water from the uterus, with violent itching in the pudendum, in place of the menses. Ulceration of the neck of the uterus. Painful itchiness of the whole body. Constipation with the peculiarity that the fecal matter comes to the front of the anus, but recedes to the rectum and after much effort it can be expelled. Abscess of the labiæ. Itchiness of the vulva and vagina.

Stannum. Leucorrhœa yellowish or greenish, very debilitating. Flow of transparent mucus from the vagina. Menses in advance and profuse. The patient can neither speak nor laugh in a high voice without experiencing feeling of weakness in the chest which arrests their force.

Sulphur. Flow white preceded by drawing or pinching around the umbilicus, aqueous or yellowish, or coming out especially in the morning after rising. Flow white mucous, fifteen days after the menses. Leucorrhœa corrosive, burning, or smarting as on applying salt. Sensation of coldness of the feet, even when they are not cold to the touch; heat at the summit of the head. Flushes of heat terminate with slow perspiration and a feeling of weakness and exhaustion. Hunger and exhaustion at 11 a. m. Unsound sleep; the patient is depressed in the morning frequent access of weakness during the day.

Sulphuricum acid. Flow white, abundant, acrid, gnawing and burning. Leucorrhœa without colour, milky or transparent. Menopause.

Syphilinum. Leucorrhœa abundant, aqueous, acrid, with acute pain as of thrusts of knife in the ovary. Flow acrid, causing violent itchiness and inflammation of the external organs, worse at night in the heat of the bed. Parts are very sensitive.

Tarentula. Leucorrhœa acrid, yellow, persistent. Obstinate uterine neuralgia. Pain in the sacrum and leg; vulvar pruritus; nymphomania. Abundant menstruation with frequent erotic spasm. Constant sensation of fatigue. Fear of death. Anæmia.

Thuja. Leucorrhœa abundant thick, greenish with violent pain in the ovary and left inguinal region. *Leucorrhœa between*

one menstrual epoch and another. Flow of mucus from the urethra. Vagina very sensitive; swelling of the labiæ with burning pain when they are touched or on walking; ulceration of the internal parts of the vulva; *verrucous excrescences* with pricking and burning on passing urine. Crampy pain in the vulva and perænium on rising from seat which extends to the abdomen.

Tabacum. Flesh-coloured leucorrhœa, fifteen days after the menses.

Tartarus. Leucorrhœa viscous, white and mucous.

Trilitium. Leucorrhœa abundant, exhausting, sanguinolent with atony, prolapsus and chronic engorgement of the neck of the uterus. Leucorrhœa abundant, yellow, and filandrous. After menses, creamy and yellowish fluid. Metrorrhagia during the menopause.

Ustilago Maydis. Leucorrhœa offensive, abundand, yellow, flow of brownish liquid mixed with black clots. Leucorrhœa albuminous and excoriating. Hypertrophy of the uterus. Uterine displacement with abundant hæmorrhage. Acute pain constant or intermittent in the left ovary which is swollen and very sensitive. Menorrhagia during the menopause.

Viburnum. Leucorrhœa excoriating, thick, white and abundant produces smarting pain and itchiness of the genital parts. Crampy pain in the base of the abdomen, extending to the anterior muscles of the thigh. The pain comes suddenly and is very violent. Menses very late, rare, only remain some hours and are of bad odour.

Zincum. Leucorrhœa mucous, thick, mostly vaginal, acrid, sanguinolent with ulceration of the neck. Profuse during stool, often preceded by colic. Pricking and pinching in the pudendum. All the symptoms are associated with restlessness, depression, chilliness, spinal sensitiveness, and trembling of the feet. Consequences of masturbation.

(*To be concluded*).

Meteorological Observations taken at 8 A.M. at the Indian Association for the Cultivation of Science, Calcutta.

For the Month of July, 1907.

Date.	Barometer.	Wind.		Temperature.		Humidity.	Cloud.	Rainfall in inches of past 24 hours.
		Direction.	Velocity per hour in miles.	Maximum.	Minimum.		Proportion.	
1	29·473	S	4·9	92·5	81·0	73	6	0·10
2	29·515	S	4·2	90·2	81·0	88	8	0·12
3	29·537	S	3·9	91·2	81·0	82	9	0·18
4	29·581	S	4·5	91·0	81·2	87	8	0·61
5	29·571	S	4·7	91·0	82·5	94	10	0·15
6	29·571	S	4·8	89·8	77·0	90	9	0·63
7	29·584	S	6·1	90·0	84·0	90	8	*Nil.*
8	29·624	S	6·3	92·6	74·8	87	8	1·33
9	29·670	S	3·4	86·5	79·0	85	10	0·15
10	29·731	S	4·4	90·0	79·5	80	9	0·01
11	29·682	S	4·4	93·0	75·4	94	9	1·32
12	29·623	S	3·9	89·0	77·0	87	7	0·43
13	29·607	S	4·3	94·0	79·5	72	4	*Nil.*
14	29·631	S	3·2	97·0	83·2	77	4	"
15	29·683	S	9·3	95·8	83·5	81	6	"
16	29·651	S	4·9	95·0	83·0	74	7	"
17	29·587	S	5·3	95·0	82·2	80	7	"
18	29·644	S	5·4	95·0	83·0	77	8	"
19	29·510	S	5·2	95·0	83·0	77	7	"
20	29·512	S	3·8	95·0	76·5	94	5	0·75
21	29·498	S	2·6	92·5	80·0	80	7	0·08
22	29·414	E	2·0	94·0	80·5	90	6	1·60
23	29·492	S	2·4	92·5	81·0	96	8	0·14
24	29·462	S	3·0	91·2	81·4	88	7	0·01
25	29·456	E	3·1	92·0	82·4	80	7	*Nil.*
26	29·482	E	4·6	91·5	81·0	88	6	0·31
27	29·487	S E	4·6	90·8	81·4	94	8	0·16
28	29·462	S	3·4	92·8	81·5	74	7	0·11
29	29·483	S	2·2	92·8	80·5	87	8	0·79
30	29·478	W	2·6	92·2	81·5	73	8	0·05
31	29·482	S	2·3	92·6	81·8	82	6	*Nil.*
Mean	29·554	S S E	4·2	92·4	80·7	84	7	Total 9·03

Remarks: The mean atmospheric pressure in the month of July being 29·554 shewed an increase. During the last month

it was 29·507, manifesting continuous decrease commencing from January. As in the month of June, the mean direction of the wind was E. S. E. Its mean velocity was 4·2. The mean maximum temperature was 92·4 and the mean minimum was 80·7, shewing a difference of 11·7. It is to be observed that the difference between the two mean temperatures was gradually being reduced. The difference in the month of June was 14·2 and that in May it had been 18·5. The mean humidity was 84 in contrast to 82 of the last month. The total rainfall was 9·03 inches in comparison to 17·67 of the month of June, shewing weak monsoon in this part of Bengal.

As to the mortality from cholera, during the week ending the 29th June, it was 68. In the week ending the 6th July, the death came down to 46. In the next week ending the 13th July, it further reduced to 26. During the week ending the 20th July it increased to 38 and in the following week ending the 27th July, the mortality went up to 65. It may be said that gradual decrease and increase without complete cessation are the pevailing characters of the mortality from cholera. The slight rainfall on the 8th and 22nd of the month could make no impression on the disease.

During the week ending the 29th June, the mortality from plague was 28. In the week ending the 6 July, it reduced to 18. In the next week ending the 13th July, the death numbered to 18 or remained exactly the same in number as in the last week. In the week ending the 20th July it came down to 11 and in the week ending the 27th July it increased to 16. On the whole the ravage of cholera was more than that of plague.

Smallpox was not altogether absent. The mortality ranged from 9 to 2 in a week. The mortality from fevers was rather on the increase. In the weak ending the 6th July it took away 115 persons. During the next week ending the 13th July the mortality was 110. In the week ending the 20th July 100 persons took leave from their earthly homes. In the week ending the 27th, death carried away 86 persons. The fact is that fevers took away more persons than any other disease in the town.

Death from bowels complaints ranged from 38 to 25 in a week. The mortality rather increased during the last part of the month.

The total number of deaths during the above mentioned four weeks of the month of July was 1,573, among the population of 8,47,796, shewing the ratio of 24·05 during the period.

EDITOR'S NOTES.

Coccus Cacti.

The following is from the *North American Journal of Homœopathy*, of July :

"This is a seldom-used remedy and easily overlooked if its characteristics are not now and then brought to mind. Its specific action is renal : Pressive, sticking, drawing pains from kidneys to bladder ; urine brown-red pungent, strongly acid, with heavy sediment. Not alone this syndrome, typical of catarrh of the renal pelvis, is cured, but various other disturbances, non-febrile and with the characteristic urine, call for the remedy. It acts specially also upon the mucosae of the respiratory tract—though, ropy albuminoid, difficultly expectorated mucus. Pertussis with the above indication and scanty, red sedimented urine. The 2x—3x are commended.—Dr. H. Kesselring.—*Homeopathische Monatsblatter.*"

The indications of Cochineal is clear to conform it to use. Coccus Cacti can cure whooping cough when the cough is worse in the morning. The vomited mucus hangs in long strings from the mouth. Frequent scanty stools with much mucus is relieved by its use. The general recommendation is the low dilution 2 or 3 decimal.

The "Faking" of Butter.

The *Public Health* of July has the following interesting note on the adulteration of butter :

"The remar ks made by Sir E. Strachey in introducing the Butter and Margarine Bill into the House of Commons indicate the necessity for further legislative powers. The evidence before the Select Committee had shown how it paid to adulterate butter. One witness stated that overtures had been made to his company offering a process of adulteration which should not be open to detection, but which would increase their present profits by £5,000 a year. In a case taken against Bridgewater Creamery Company only recently, it was said that the profits made by adding lardine to butter were £5,650 in fifteen months, so that there was a very real need for this Bill, which aimed at stopping the adulteration of butter at its source. Previous Bills were aimed at the seller or retailer ; this measure went to the source."

Adulteration of food has assumed a character which shows the degrading principle of tradesmen. We do not know where and how it will cease. The adulteration is an attempt to slow poison. In that view of the matter severe steps should be taken to prevent it. The American and European trade for adulteration has proved disastrous to the whole world. Unless rigorous measures are adopted, fine seems to have failed as a deterrent punishment. Imprisonment with fine is the only alternative. Even then we are doubtful how far the step will be successful.

Paratyphoid and Meat-Poisoning.

The *Public Health* of July writes :

"The first clear diagnosis of a case of paratyphoid fever, that is, of a bacterial septicæmia clinically like typhoid but associated with a bacillus quite distinct from Eberth's, was that of Gwyn in 1898, where a "paracolon" bacillus was cultivated from the blood of a patient with all the clincal features of typhoid fever. Cushny (1900) in America, and Schottmüller (1900-1) in Germany, by a systematic application of the method of blood-culture in cases of "typhoid," showed that paratyphoid was not an uncommon disease in these countries. During the past five years cases have been reported from many parts of the world—including India and Japan. Recently Boycott has published an account of three fully established cases of infection with *B. paratyphosus B.*, which were met with in England during 1905 while examining a series of 176 specimens of blood sent in for Widal's test in suspected cases of typhoid. For detailed description of these cases, together with a useful bibliography on paratyphoid infections, the original paper should be consulted. Bacteriologists are by no means in agreement with regard to the systematic grouping of the organism concerned in the production of these fevers; but the following classification appears to be the one which reflects the most recent views of authorities on this subject : (1) *B. paratyphosus A*, (2) Gärtner's meat-poisoning bacillus, (3) Hog-cholera group, including *B. typhimurium*, etc., (4) *B. paratyphosus B.* The A type of paratyphoid bacillus is easily distinguished from the other three groups by cultural methods, is more nearly related (especially) culturally to Eberth's bacillus, and according to universal experience is much less frequently met with than *B. paratyphosus B.* Gartner's bacillus is differentiated from *B.*

paratyphosus B and the hog-cholera group by the agglutination reaction with sera prepared from known cultures.

The differences, however, in agglutinability between *B. paratyphosus B* and the hog-cholera group are so slender that at times it is impossible to distinguish them, yet many authorities who merge them in one group acknowledge that there is a certain amount of contrast between the two. Schmidt is of the opinion that hog-cholera, mouse-typhoid, and *B. paratyphosus B* cannot be differentiated, morphologically, culturally, experimentally, or by agglutination, and places them together under the hog-cholera group. Bock has demonstrated the close relationship between hog-cholera, mouse-typhoid, and the Kaen type of meat-poisoning bacillus, and although he places the B type of paratyphoid bacillus along with these as showing "no essential differences" culturally or in pathogenicity for animals, he admits that there is a certain amount of "contrast," and that the relationship between the first three mentioned is much more intimate than between these three and *B. paratyphosus* B. Boycott considers the distinction between the B type of paratyphoid bacillus and the hog-cholera group, although slight, is real. In the classification of Bohme not only is *B. paratyphosus B*, but also the Aertryck type of meat-poisoning bacillus and *B. psittacosis* found along with B. *typhimurium* in the hog-cholera group, which is differentiated from Gartner's bacillus by the immune-serum reactions. In contradiction of Bonhoff's statement, Bock also reports that Gartner's bacillus is easily differentiated from the others by the agglutination test.

In Germany and America it is estimated that about 10 per cent of cases clinically diagnosed as "typhoid" are in reality paratyphoid.

In England, Boycott, from the past year's experience, puts the proportion at 3 per cent, but admits that this is probably too low. In paratyphoid, intestinal ulceration is more frequently absent, and the prognosis is much more favourable, the case-mortality of paratyphoid being 1 to 4 per cent as compared with 17 per cent for typhoid (London). It is therefore desirable for the patient's, as well as the bacteriologist's, satisfaction that an accurate diagnosis should be made. Although for diagnostic purposes a bacteriological examination of the blood is preferable, and occasionally absolutely necessary, for differentiation and identification a diagnosis of paratyphoid can generally be made from the agglutination reactions of the serum with known cultures of the various organisms (Boycott). Basing their observations on forty cases of true typhoid, Grunberg and

Rolly report that 70 per cent of all cases gave the group agglutination reaction with paratyphoid bacilli in a dilution of 1 in 30 or higher and in 33 per cent the paratyphoid bacilli were agglutinated in a higher dilution than the typhoid bacilli. They therefore maintain that a bacteriological blood examination alone under certain circumstances can show the true nature of an illness. In reply to Grunberg and Rolly, Korte and Steinberg, having investigated the group agglutination reaction in seventy cases of true typhoid (for a positive result a dilution limit of 1 in 40 being required), state that in all cases the typhoid bacillus gave the reaction in higher dilution than the paratyphoid bacillus, and explain the results of Grunberg and Rolly by (1) their not having estimated the exact agglutination limits, (2) their having used the macroscopic test which is not so exact as the microscopic, especially at the limits of agglutinability, and (3) the occurrence of "zones of inhibited agglutination," which are more marked in the macroscopic test. Similarly, Manteufel says that in his experience paratyphoid bacilli seldom gave the group reaction in dilutions over 1 in 50; in all cases higher limits were got with the typhoid bacilli, and in no case did agglutination occur with paratyphoid bacilli when absent with typhoid bacilli. Korte and Steinburg observed that occasionally the typhoid bacillus failed to agglutinate with serum in low dilutions (zones of inhibited agglutination), while if the serum were more diluted, agglutination appeared; and the same authors also noted that this phenomenon was materially misleading only in the macroscopic method.

According to Falta and Noeggerath, the agglutination-preventing bodies (*Hemmungskorper*) which cause these zones of inhibited agglutination, appear generally towards the end of illness, and to prevent error it is advisable to use a dense emulsion of bacilli and test the serum to its agglutination limits. Although anomalous results are occasionally obtained, irregularities in agglutination results can generally be attributed to one or other of the following: (1) errors in technique and judgment of results, (2) a tendency to spontaneous agglutination of some strains (therefore use a strain of known agglutinable capacity), (3) the ultimate limits of aglutinability, especially by the microscopic test, of the different organisms not having been attempted, (4) zones of inhibited agglutination, (5) mixed infection. For the diagnosis of mixed infections, Castelleni's test of absorption or exhaustion of agglutinins is necessary. Castellani found that an excess of typhoid bacilli would

remove not only primary or homologous (i.e., typhoid), but also secondary or heterologous (e.g., coli) agglutinins from the serum of a rabbit inoculated with typhoid bacilli; while from a serum elaborated in response to inoculation with both B. *typhosus* and B. *coli*, neither typhoid nor *coli* bacilli alone, but only both together—simultaneously or successively—would remove all agglutinins. An instance of mixed infection by typhoid and paratyphoid, established by Widal's Pfeiffer's, and Castellani's tests, has been recorded by Gaehtgens. Ficker's paratyphoid diagnostica are two new bacterial suspensions—substitutes for living bacterial cultures, of B. *paratyphosus A* (Brion-Kayser) and B. *paratyphosus* B. (Schottmuller), respectively, specially designed to be of service for the agglutination test to practising physicians. Minelli reports favourably on these proprietary preparations. Stuhlinger by killing paratyphoid bacilli with chloroform, or better still by aseptic autolysis (in normal saline at 37 for two months), has obtained very good paratyphoid diagnostica.

A case of infection by B. *paratyphosus A* (Brion-Kayser), where the bacillus was isolated from the blood, urine and fæces, has been recorded by Kayser: and Brion lays stress on the fact that paratyphoid, like typhoid, is a bacillæmia rather than an intestinal disease.

Meat-poisoining bacilli, which, it will be observed, find a place in almost all groups in the above classification, have been divided by Levy and Fornet into two broad classes—(1) those like B. *botulinus* (sausage poisoning), that produce nervous symptoms, and (2) those that have an accentuated action on the gastro-intestinal tract. They give an account of a family of seven that was infected with B. *paratyphosus* B. the symptoms being very like those of typhoid: the bacillus was isolated from the excreta of all seven, and was agglutinated by the serum of the patients in dilutions of 1 in 200 to in 1 10,000. An instance of sausage poisoning, caused by B. *paraytphosus* B. has been reported by Kreehl. The fever ran a course like that of a moderately severe case of typhoid; the bacilli were found in great numbers in the stools from the beginning of the illness, and agglutination (1 in 1,000) appeared on the eleventh day of illness and only with *B. paratyphosus B.* Curschman recognizes the gastro-intestinal and nervous forms of meat poisoning, and regards *botulinus*, outside man, as a pure saprophyte. Gartner's bacillus, on the other hand, generally occurs in the flesh of cattle that have been sick during life. Schmidt,

Pottevin, and Klimenko, all draw attention to the significance of sickness among domestic animals at a time when meat-poisoning and typhoid-like illnesses of man occur.

Pottevin isolated a bacillus very like Gartner's from a ham which had infected four members of a family of seven. The blood of the four patients agglutinated this bacillus in dilutions of 1 in 50 to 1 in 500, while tests with the serum of other persons, even in a dilution of 1 in 25, were negative. The bacillus was pathogenic for young cats, producing, after ingestion with milk, a diarrhœa which persisted for about three weeks. Klimenko isolated B. *paratyphosus* B. from a perfectly healthy adult dog, and believes that this may throw some light on the occurrence of sporadic cases or even epidemics of paratyphoid fever. The bacillus was pathogenic for mice, rats, guinea-pigs, rabbits and young dogs, causing fever and diarrhœa, and retained its vitality in milk for a long period being still alive and virulent after one year and four months."

The bacteriological differentiation generally speaking will not help us in private cases. The clinical demarcation has not been given. As far as our knowledge extends typhoid and paratyphoid can be differentiated by the mild nature of the latter, principally diarrhœa. In typhoid, there is good deal of intestinal ulceration, consequently severe diarrhœa is its character. Paratyphyoid may have diarrhœa of mild nature or constipation. Delirium or other symptoms may be almost the same. The aggravation of the symptoms depends also on the nature of treatment.

On the other hand, meat poisoning bacilli have severe diarrhœa simulating cholera. The stools are generally green but may be choleraic. Other symptoms, as fever, delirium etc., are much less in comparison to the other two.

Any hard and fast line of distinction is not possible between typhoid and paratyphoid. The general indications are given above. It is certain that we get more paratyphoid cases than real typhoid. In over-populated towns of India, the two diseases are frequently observed.

Mercurius corrosivus.

The following record of cure of rectal fistula by Mercurius Corrosivus, mentioned in the *North American Journal of Homœopathy* for August is interesting :

"Case 1. Boy, æt. 3, with a complete rectal fistula, a rare occurrence in children. Operated the year before, but, as often with rectal fistula, the trouble had returned. Although the child appeared sound, the result of medication seemed to point to luetic heredity; for mercurius corr. 5x healed in a few weeks, and for two years there has been no return.

Case 2. A blooming, healthy woman (eine blühendegesunde Frau) with no possible specificity apparent. Mercurius corr. ix healed the fiistula in a few weeks."

Some Cough Remedies.

The following is from the *North American Journal of Homœopathy* for August :

"Ammonium Mur. Persistent cough, tickling in trachea or larynx, dry in morning, loose in afternoon and night, when it becomes spasmodic, ending sometimes in gagging or vomiting. Stitches in left chest or hypochrondrium. Coughs so severe that he coughs up blood.

Ammonium carb. Cough worse about midnight with asthma about 3 A.M., dry cough like a feather in throat, accompanied by a constricted feeling in chest.

Silphium. Cough loose, expectorates copiously of grayish mucus, asthmatic breathing, worse at night, copious acrid discharges from anterior or posterior nares, causing smarting sticking, sensation in throat and soft palate.

Sanguinaria. Marked soreness and rawness in larynx and under sternum, cough paroxysmal, no relief till some mucus is raised, worse at night, stitches in right chest, and hypochrondrium. Later in the stage of catarrhal conditions expectoration becomes yellow or brownish yellow.

Rumex. Cough excited by a tickling irritation behind the upper end of sternum, dry, harsh, shaking, excited by pressure on sternum, inhaling cold air and worse at night, great difficulty in raising a small quantity of thick, tenacious mucus.

Hyoscyamus. Voice husky as of mucus in throat, dry cough of a spasmodic nature, comes on at night or excited by talking,

or laughing, during coughing a constricted feeling in larynx. Sensation as if the palate had dropped down.

YERBA SANTA. Copious quantities of mucus in chest, worse on right side, cough like whooping cough, coming on during dentition in scrofulous children, cough immediately becomes dry after exposure or when fever develops. Breathing does not become asthmatic like in silphium.

STICTA. Croupy cough during beginning of influenza, oppression of the chest, hard, racking cough, with pains reaching from the sternum to spinal column.

SENECIO AUREUS. Loose cough with copious expectoration of thick, yellow, sweet mucus, often streaked with blood, rawness and soreness in chest, especially when accompanied with delayed menstruation, flushes of heat to head and chest, worse in afternoon and night."

These are not ordinary cough remedies. They are to be used with reference to their special indications. In *Ammonium Mur.* the cough is aggravated after a meal or cold drink. The spitting of blood is preceded by tickling in the throat. *Ammonium Carb.* has cough only at night, or only by day, or in the evening, before going to sleep, or in the morning from 2 to 5 A. M. The cough of *Hyoscyamus* is worse on lying down, almost completely removed by sitting up, worse at night, worse after eating, drinking or talking. Cough from elongated uvula. *Rumex* has cured cases of cough with tickling behind the top of sternum, trachea sore to external pressure, excoriated through its whole extent as also the fauces. Cough exhibited by pressure on throat pit, cough violent with scanty difficult expectoration. *Silphium* has copious expectoration of stringy, frothy, light-coloured mucus. *Sticta* is for hard dry barking cough following cold.

Variolinum as a Prophylactic.

We take from the *North American Journal of Homœopathy* for August, the following note:

"A Leading member of the faculty of the Harvard Medical School stated recently that it could no longer be said that the dominant school was without a therapeutic law inasmuch as the law of immunity had been adopted as a principle. In the development of this principle the school has come perilously near, to say the least, to homœopathy and immunization is by no means a

new idea recently evolved in old school ranks. Every student of Hahnemann's writings knows that he called attention to the well-known fact of natural immunity to certain diseases acquired through one attack of these maladies, and he developed the idea of immunity artificially produced by the administration of drugs, notably in the case of belladonna and scarlet fever. The most recent advances in immunization bear close relation to the use of nosodes which have been used more or less widely in the homœopathic professsion for a number of years.

The most familiar example of prophylaxis is the use of vaccine virus as a preventive against smallpox, and so widespread has been its use, and so satisfactory, on the whole, have seemed to be the results that the majority of homœopathic physicians have not thought it necessary or advisable to inquire if there were any superior method of prophylaxis.

The internal administration of variolinum has been advocated for smallpox prophylaxis, however, for many years by a number of homœopathic physicians, and in the State of Iowa this method has the support of the majority of our branch of the profession practising there; and three district courts have decided that prophylaxis by this method complies with all the requirements of law.

In a paper read before the Bureau of Sanitary Science at Jamestown, Dr. Charles Wodhull Eaton, of Des Moines, Ia., by invitation represented the homœopathic physicians of his State in presenting to the American Institute of Homœopathy the "Facts about Variolinum," as elicted by them. In that paper he pointed out that variolinum was a preparation of the virus of smallpox and not cowpox, as is vaccine virus. He then discussed the reasonability of the use of variolinum, showing that this depends upon whether an individual can be rendered immune to a given disease by the administration of the virus of that disease, and upon whether the virus can be effective if administered by the mouth. Then comes the test of experience. By correspondence with other physicians in Iowa soliciting conservative figures borne out by actual case records, Dr. Eaton presented the following statistics: Number of people to whom variolinum had been administered internally as a prophylactic against smallpox, 2,806; number of these actually known to have been exposed to smallpox after taking variolinum, 547; number who had smallpox after taking variolinum, 14. Dr. Eaton called special attention to the fact that the number of pro-

phylactic administrations and of subsequent exposures was far larger than the above figures would indicate, since the figures dealt with cases on record only. One correspondent suggested that the number of known exposures was probably less than ten per cent. of the actual number of exposures.

These figures demand some consideration; certainly they cannot be ignored. Did they emanate from some European authorities high up in the councils of the dominant school they would be widely quoted. They certainly show that prophylaxis against smallpox by the internal administration of variolinum deserves further investigation and possibly submission to a scientific testing out under the strictest regime of modern science.

What sanitarians advocate and are aiming at is prophylaxis —vaccination is only a means to that end. If the internal use of variolinum is as efficient a means of prophylaxis as vaccination as ordinarily understood, the choice of the means can well be left to the patient or guardians of the child; and if preference is shown for the newer method it will be a feather in the cap of homœopathy."

Whatever difference there may be between vaccininum and variolinum, it is one of quantity and not of quality, as vaccininum is nothing but the virus of smallpox when it occurs on cows. The transition is not enough marked. The intensity of the vaccinal virus may be less than that of the smallpox. In other words it is a difference of dilution or trituraton. In India, during smallpox epidemics both the nosodes have been tried as prophylactic with success in most cases. For ourselves, we were content to use vaccininum as variolinum may increase the danger of getting smallpox from overdose. Whereas in that consideration vaccininum will be the safe remedy to use.

CLINICAL RECORD.

Foreign.

CLINICAL CASES.

By Dr. Stonham.

Case I. Backache.—*Magnesia Mur.*

E. W., aged 45. Operated on some years ago for removal of appendix.

November 23, 1906.—Came complaining that for the last two nights, directly on lying down in bed, she had a severe pain in the upper sacral region; the pain she described as a sore pain, causing her to draw up her legs—"it is agony if she puts them down straight." She can only get ease by lying on the front and right side of the stomach, < lying on the left side. This pain lasts some hours; she finally gets to sleep and wakes without it. The pain makes her very hot, burning hot all over; she does not perspire at the time of the pain, but when she wakes up after the sleep which follows it she finds herself bathed with perspiration. She has no pain through the day. Bowels have acted quite regularly and sufficiently. Catamenia one week ago; were less profuse than they used to be, but there was nothing abnormal about them. No leucorrhea lately, but till a month ago she had a yellow discharge, worse at night. Has slight piles. Stools have been lumpy lately—hard lumps, conglomerate. *Mag. mur.* 3x ♏v, 4 hours. The pain at once began to get less and was quite gone in five days.

The general hyperesthesia, the < from lying on affected part, the < on lying down at night and > while getting about during the day, the burning and heat, and the character of the stools were the indications for *Mag. mur.*

Case II Diphtheria—*Lachesis.*

E. M., boy, aged 5. Was called to see him on May 13, 1907, and found him lying in bed very prostrate and with a pulse of 132 and temperature of 107° F. I was told that this was the seventh day of his illness, and that he had been gradually getting weaker and would take no food. The only complaint he had made was that it pained his throat to swallow. On examining the throat I found a dirty-looking membrane on the l. tonsil, the rest of the throat being clear. Cervical glands not enlarged. He was given

Lach. 12, a few drops in a tumbler of water, a dessertspoonful to be taken every hour, and a swab was taken from the throat for bacteriological examination.

The next day, May 14th, much better. Temp. normal and pulse 100. Appetite returning. Much less prostration. Membrane gone. Continue medicine.

May 15th. Temp. normal, pulse 80. No return of membrane. Eating well and quite lively. Convalescence uninterrupted.

The bacteriological examination showed the specific diphtheria organism to be present. The great prostration and the situation of the patch on the left tonsil indicated *Lachesis.*

CASE III. DIPHTHERIA.—*Mercurious Cyan., Apis,* ETC.

C. C., boy, aged 9. He was taken with a shivering fit while at school on the morning of Thursday, May 9th. After dinner he complained of his legs giving way, and was kept at home from school in the afternoon. All day Friday he was very feverish and was light-headed; he complained of sore throat and took but little food. His mother gave him *Acon.* and *Bell.*

On Saturday he woke early and said he was hungry and wanted to get up. He had a good breakfast of bread and milk and came downstairs about midday; throat felt better; he ate vegetables and gravy for dinner and an ordinary tea. Was given two doses of *Mercurius sol.* 3x. He slept well till 4 A. M., on Sunday, when he woke complaining of thirst and dryness and soreness of the throat.

I saw him for the first time at 9-30 A. M., of Sunday, May 12th, the fourth day of the illness. His temperature was then 104° and pulse 122. Both tonsils were covered with a dirty membrane and the breath smelt extremely fœtid and of characteristic diphtheritic odour. He was given *Merc. cyan.* 30 every hour.

In the evening, May 12th, 9 P. M., temperature had fallen to 101° and pulse to 112. He had felt better and had been able to swallow food well. A swab from the throat was taken for examination and sent to Dr. Watkins. I may say here that the report when received showed the presence of the specific diphtheria organism. Repeat med.

May 13th.—In the morning better. Temp. 100°; pulse 100. No extension of the membrane, which seems inclined to disappear in parts. Rep. med.

May 14th.—Not so well through the night. This morning the membrane has increased again and extended to the uvula, which is

much swollen; some slight nasal stuffiness indicating commencing extension to the back of the nose. Had a choking attack in the night. Throat pains on swallowing, and glands at angle of jaw are a good deal swollen on each side. Rep.

May 14*th*.—(evening, 5-30 P. M.).—No further extension of membrane and feels and swallows better. T. 100·2°, P. 88. I injected 2,000 units of *Antitoxin* (Burroughs and Wellcome). Continue *Merc. cyan.* 30.

May 15*th*.—Temp. 100°, P. 96. R. tonsil and uvula are covered with membrane; the uvula is very long and edematous; swallows better; nose less stuffy. *Lach.* 12 every hour.

(Evening.) Temp. 100°, P. 102. R. tonsil and uvula are much the same. L. tonsil is clearing. Has eaten better to-day. Bowels have acted. Rep.

May 16*th*.—Slept well most of the night. Temp. 98·4°, P. 80. Throat rather cleaner but still much edema of the uvula and tonsil. Pain on swallowing runs up into the ears. Taking food well. Unit dose of *Apis* cm.

May 17*th*.—T. 98°, P. 76. Slept well. Much less swelling of uvula and tonsils. Uvula clear of membrane and tonsils nearly so. Appetite good and scarcely any pain on swallowing. Is bright and cheerful. *S. V. R.*

May 18*th*.—Much better. T. 97·5°, P, 62. All membrane has gone except a speck or two on the right tonsil. Uvula no longer swollen. No pain on swallowing. Appetite very good. Bowels act daily.

May 20*th*.—Going on well. T. 98·4°, P. 62. Throat quite normal, knee jerks normal. Rep. *S. V. R.*

CASE IV. HEADACHE.—*Silica.*

J. L., aged 41. Female. Had rheumatic fever at 16, was laid up for months with it, and has had rheumatism in the knuckles on and off since, and also in the limbs after washing. Two months ago she began to have pain in the left side of the head, starting in the left temple and darting across the top of the head, worse in the morning soon after getting about, better towards evening; sensation of cold water on the vertex with the darting pains; the eyelids feel as if they must be closed. The darting pains seem to take her senses away. The only way she can get ease is to wrap the head up in flannel; when the head gets quite hot and perspiring the pains are better; she is very irritable with the pain and must get away by herself and lie down. Feet feel very cold and are clammy. The feet used to perspire offensively before she had the rheumatic fever but not since. Was confined three months ago with a dead child at full time, and lost much blood at the confinement. Appetite poor. Tongue, slimy white fur, bad taste. Bowels regular. Catamenia returned this week, lasted three days, and were moderate in amount. February 28, 1907, *Silica* 30 ♏iii *nocte maneque*. Result, speedy cure of the headache.

CASE V. NEURALGIA OF FACE AND FOREHEAD.
Stannum.

Mrs. G., aged about 30. Was confined with her second child on January 1, 1907; the confinement was a good one, and she nursed her child, the milk being sufficient and the child thriving. Has lately been without a servant and had extra work and worry. On February 27th the attacks commenced, for which she sent to me on March 4th. They had continued daily without intermission and consisting of neuralgic pains on the left side of the face, affecting the supra-orbital arch, the zygoma, and the left side of the forehead. The pain was mostly throbbing, but at the inner canthus there was a feeling as if a wedge were being driven in. The affected parts were red and a little swollen. It pains the eyes to look upwards. The pain comes on about 9 A. M., increases gradually till noon, remains at its height till 3 P. M., and gradually declines till 6 P. M., when it goes completely. No pain at night. While at its height the pain seems unbearable and there is giddiness. The pain is < heat of fire, > firm pressure, not > by lying down; not influenced by food or drink; a rather sick feeling and distaste for food when it is at its height. Tenderness over affected region remains after pain is gone. She had a similar attack last February and also at other times when run down, even from childhood. The attacks are more apt to occur in the spring.

March 4th.—*Stannum* 12 ɱv., 4 hours.

March 6th.—Had the worst attack she has had at all. Great agony from 12 to 3, could hardly contain herself. The pain moderated later in the day, but did not entirely go off as it usually does. She, however, had a good night's rest, but woke with still a little pain, which became slightly worse in the forenoon but did not increase after 12 o'clock. The face was less red and the eyes less swollen, and she was well enough to go out for a walk between 12 and 3. Discontinue med.

March 12th.—Only a few shooting pains in the face each day of no great severity. Face is, however, a little red, hot and swollen on the left side and she says the scalp is rather tender. Sleeps, eats, and feels well. *Bell.* lx, pil. ii t.d.s. There was no return.

The seat of the pain—the left side of the face—and the crescendo-decrescendo character of the pains were the indications for *Stannum.*

CASE VI. RHEUMATIC PAINS IN THIGHS.—*Kali Carb.*

Mr. S., aged 60. Has for two or three weeks complained of rheumatic pains in the thighs, < on the right side. They seemed to come on after exposure to damp and are of sharp character and affect the front and outside of the thighs from the hips to the knees; tenderness on pressure. They are > from walking, and become still worse the more he walks; there is a feeling as if the r. thigh, which is much the worse affected, would give way on walking. The pains disappear at night in bed. *Bryonia* was at first tried, with only slight relief. He was then given *Kali carb.*

30. Only two doses were taken, as the medicine, which was given mixed in a tumbler of water, was by accident thrown away, but complete relief was at once obtained. A week later a long walk brought back the pain in the right thigh to a slight degree, but another dose of *Kali carb.* 30 sufficed to drive it away.

Kali carb. especially affects the thighs and chiefly the right thigh from hip to knee. Its pains are sharp and shooting and it has the symptom "feeling as if the right thigh would give way on walking." According to Clarke's *Dictionary* the pains of *Kali carb.* are better while moving about and < at night, but I have always found the reverse to be the case with the sharp shooting pains in the r. thigh cured by *Kali carb.*—*Homœopathic World* July, 1907.

LATENT GOUT.

By G. Blackley, M.B.

Case 1.—Chronic catarrh—nasal, bronchial, gastric, and intestinal—neuralgia and eczema.

Mr. M., a retired stockbroker, came to me first in 1899 at the age of 70. He is a strict Jew and of very spare habit, weighing barely ten stone. Has always been very abstemious and careful in his diet. Father had gout. He himself has never had gout, but a few joints are distinctly nodose.

When first seen his chief complaint was supraorbital neuralgia, which, as it was associated with very profuse nasal catarrh I ascribed to blocking of the frontal sinuses. He had, at the same time, a few small patches of dry eczema on the shins. Since 1899 he has suffered in turn from catarrh in one or other form but most of all with rectal catarrh, accompanied by severe pain in the hypogastrium, and associated with slight fissure and pruritus. The urine has been free from albumen and sugar, but constantly deposits red sand and oxalate of lime crystals. The drugs which have helped him most have been lycop., ignatia, arsen. and opium. Occasionally gastralgia was only to be allayed by salicylate of bismuth.

Repeated visits to Harrogate, Llandrindod, and Eaux Bonnes have resulted in no permanent relief to any of the catarrhal manifestations.

Supraorbital and frontal headache are now always relieved, at least for the time being, by 0·5 gram doses of aspirin. The pain in the hypogastrium is best relieved by small doses of heroin.

Case 2.—Intestinal catarrh.

Miss P., aged 61, comes of a gouty stock, but has never had any pronounced arthritic attacks, although she has deformity of several of the finger joints. She is of very spare habit, weighing barely eight stones, and has always been a total abstainer. Came to me in 1898 complaining of constant looseness of the bowels; stools when

formed being of very small calibre. This condition of matters has continued with intermissions until the present, but usually yields slowly to verat. and arsen.

In March, 1904, came to me complaining of much pain and tenderness over the left sacro-iliac articulation and in both knees, which were very stiff in the morning. Sulph. 3 and later ledum were given. In June she spent a month at Aix-la-Chapelle, and came back very much improved in every way.

In 1905 began to complain of pain in left iliac fossa, especially after any little chill, the pain being followed by sickness and diarrhœa, the stools pale and containing mucus. Weight stationary. Digital examination afforded no special information. Hydrast., arsen., lycop., and ac. nit. all failed to give more than very temporary relief. In January last these attacks of relaxation began to alternate with constipation, during which the old pain in the sacro-iliac joint was very much to the fore. Bryon. gave a little relief, but the patient was obliged to walk in a half doubled-up position. I then bethought me of aspirin, giving 0·5 grm. t.d., with the result that the patient presently wrote for a fresh supply and proclaimed herself freer from discomfort, and better in every way than she had been for a long time.

Case 3.—Catarrh of the lower bowel.

Miss W., private secretary, aged 58, one of a large family, two of whom suffered from psoriasis, one from eczema, and one from gallstones. Mother was distinctly gouty, and had occasional eczema. Patient has never had gout or any skin affection. Is very thin, but not losing flesh.

Came to me in 1898 complaining of constant mucous discharge from rectum, with stools of very small calibre. On rectal examination no obstruction could be made out, and full-sized rectal bougie passed without difficulty. Hydrastis and arsenicum were given steadily for a week at a time, and afforded much relief, though the catarrh never disappeared entirely. Since 1899 the patient has had several attacks of influenza, which have caused much nervous prostration and serious heart weakness, but during the time that these have lasted the rectal catarrh is always in abeyance.

Case 4.—Bronchial catarrh; mental depression.

Mr. H. S., aged 65, American and a quaker, of no occupation. Has never had gout, but a brother, now dead, was repeatedly under my care with acute gout. Before consulting me this patient had for twelve years suffered much from depression of spirits and inability to concentrate his thoughts, and had been obliged to travel constantly and to lead an idle life. In 1897 he had a very long convalescence under my care from influenza. Many drugs were given at first, but the one which finally became our sheet anchor was lycopodium 3. In 1901 had bronchial catarrh with asthmatic attacks, during which time he was absolutely free from the depression usually present, and was decidedly more capable of sus-

tained mental effort. The only drug he took during the attack was senega ϕ.

Case 5.—Spasmodic asthma.

Miss N., aged 48, first seen in 1900. Mother living, aged 80, very stout, and has occasional attacks of acute gout. Daughter, who has never had gout, is also immensely stout. Has suffered from asthmatic attacks in the winters for several years. I treated this patient during two severe attacks occurring in two successive winters with very satisfactory results, the drugs used being largely emetin. 4x and arsen. 3x, usually given on alternate days.

Since 1901 the patient has paid several visits to the arsenical waters of Mont Dore, with the result that the winter following has usually been fairly free from asthma. This was the case last winter, although she is still increasing in weight; has had an attack of retinal hæmorrhage in one eye and has a fibro-myoma of uterus.

Case 6.—Asthma; neuralgia.

Mr. A., aged 53, solicitor, of Jewish descent and bilious temperament. Father suffered with asthma and mother from gall-stones. Patient has had attacks of spasmodic asthma from the age of puberty, and has in consequence been obliged to live in the heart of London, a night spent at Brighton or St. Leonards being invariably followed by threatenings of an attack. This patient is particularly susceptible to the influenza bacillus, and after an attack has always a long convalescence. On one occasion influenza was followed by severe neuritis of the posterior tibial nerve, which lasted for many months, during which, however, he was free fron asthma, although six weeks of the time were spent at Harrogate. This may possibly be explained by the fact that he was taking arsenic most of the time in addition to undergoing bath-treatment at Harrogate. He now goes every year. Since the commencement of 1903 he has suffered much from left supra-orbital neuralgia with discharge of much thick yellow mucus from left nostril. Sense of smell is gone. Sulphur relieves the neuralgia.

Case 7.—Neuralgia.

Mr. A. W., publisher, aged 52, was treated by me for syphilis about the years 1880-85. Has had several attacks of acute gout. Has been practically a total abstainer for many years past. In May, 1902, came to me complaining of right supraorbital neuralgia, coupled with slight giddy attacks, which appeared to proceed from left parietal region. R Sod. iod. gr. iii, t.d., and gelsem. ϕ night and morning. At the end of a fortnight he began to have some gouty twinges, ane the urine was scanty and hypheracid; gelsem. was replaced by lycopod. No arthritic attack ensued, and at the end of six weeks the only symptom left was described as a "swimmy" feeling in the head, especially on stooping. This was promptly relieved by ac. picric, a drug which experience leads me to regard as specially useful in many neuroses in gouty patients.

He now has occasional attacks of acute gout, and is fairly free from headache or giddiness.

Case 8.—Lichen circumscriptus.

Mr. A.., a Sheffield steel manufacturer, came to me in 1901 for advice for a lichenous rash which had tormented him for some years. The patient was a small man of bilio-sanguine temperament and great mental and physical activity. Family history was gouty, but he personally had never had gout. The legs, buttocks and arms presented numerous circumscribed patches of lichenous rash which were intensely irritable. I advised a sojourn at Ilkley for the sake of its pure water and air, but as the patient had made all arrangements for going to Harrogate for a course of treatment I allowed him to go.

At the end of about a month he again presented himself, the rash being, if anything, more irritable, especially on the arms, which were always at their worst at night. I prescribed phenazone 1x, gr. i. *ter die*, and the local application of a liniment of chloral and camphor. Improvement began at once and proceeded steadily. I have usually found that patients with a gouty strain in them are particularly susceptible to the effects both of large and of small doses of antipyrin.

Case 9.—Psoriasis.

Miss G., aged 40, of nervous temperament, was sent to me by Dr. Byres Moir in 1894 on account of psoriasis. The patient's family history was distinctly gouty, but she had never had any distinct arthritic attacks herself. Under the treatment suggested by me, chiefly external, she got clear of the rash, but was not so well in general health. In 1898 she showed distinct gouty symptoms, and spent the winter in Bath at Dr. Moir's suggestion. In the following spring, the rash being again troublesome, Dr. Moir sent her to me, and I suggested substantial doses of liq. arsen. In July the patient had an attack of subacute gout in the ball of the foot, and the arsenic was interrupted. In September she went for a course of treatment to Llandrindod, and came back much improved in every way: the rash was no longer troublesome, and Dr. Moir reported that, in spite of this, the general health remained good.

Case 10.—Eczema seborrhœica.

Mr. N., aged 48, wine merchant, a Jew (but not a strict one) was sent to me first in 1898 by Dr. Byres Moir. He was then suffering with severe seborrhœic eczema of scalp, neck, axillæ and groins, for which I prescribed chiefly external remedies, the principal one being a lotion of resorcin, with the result that in a few weeks he was clear of all rash. Two years later, in the early spring, he returned with a similar, though less severe, attack, and acting on my advice, he spent the greater part of the month of April at Aix-la-Chapelle. Whilst undergoing treatment there the rash came out more, and the patient had slight attack

of acute gout in the left great toe. He returned home very much discontented with his stay at Aachen, and when in the following July he had a smart attack of impetiginous eczema on the face, scalp and neck, he was not complimentary in his references to that "beastly hole." Under antim. crud., however, and a very mild lead lotion, the rash rapidly subsided, so that by the end of the month the skin was clear. To the best of my knowledge it has remained so from that time.

Case 11.—Glycosuria.

Mr. D., aged 77, retired merchant, American; is very temperate, but fond of sweets. Father suffered from gout and bronchial catarrh, but lived to the age of 89. Patient first came to me in 1895, on his return from a trip to the States, saying he had been told that he was suffering from diabetes, and was taking pills prescribed by his old homœpathic physician in Brooklyn; these turned out to be of *codeia* in substantial doses. I encouraged the patient by telling him that the ailment was probably not genuine diabetes and would pass off. Since 1895 he has had *intermittent* attacks of glycosuria, the output of sugar ranging from 1 oz. to 3½ oz. in twenty-four hours. The attacks have usually lasted some months; there is never any loss of weight, and, except in the matter of sweets, the diet has not been restricted. The urine is always freely acid. The patient's only complaint which appeared to me in any way connected with the glycosuria—and I have noticed it in other cases—was of a feeling of discomfort in the œsophagus, especially at its lower end. This was always relieved by oxalic acid. The drugs given during the attacks of glycosuria were usually lycopod., ac. phosph. and jambul, but I cannot say that I found the two latter exercised any decided influence upon the glycosuria. In 1899 the patient again visited the United States, being away three and a half months, eating and drinking just what pleased him. On his return the urine was quite *free* from sugar (!). In 1903 he had an attack of glycosuria, during which the quantity of sugar was fairly constant at about 3 oz. per diem. I gave him aspirin, beginning with a does of gr. v. t.d., and gradually raising the quantity to gr. xx. t.d. The glycosuria was quite unaffected by it. A course of treatment at Llandrindod during the autumn of the same year was equally ineffective. Last October he returned to London, after spending fourteen months in travelling about the United States, during which time he had eaten moderately of sweets, but had been a total abstainer. The urine was normal in quantity (56 oz.), and there was a bare trace of sugar present. His only complaint was of giddiness on standing up in the morning and on stooping during the day. This was speedily relieved by ac. picric.

Case 12.—Glycosuria and insomnia.

Mr. L., a German merchant, long resident in England, aged 63, weighing 14 st. 5 lbs., who had had occasional attacks of acute gout, came to consult me first in 1903 for persistent insomnia,

with slight arthritis in thumbs. The urine was large in quantity (112 oz.—but the patient, like many Germans, was naturally a very "thirsty subject") it deposited urates on standing, contained sugar to the extent of 2½ oz. in the above quantity, and there was a trace of albumen present. The daily output of urea and uric acid were fairly normal, and the relative proportions of the two were as 43 : 1. Except as to quantity of liquid, which was much curtailed, the diet was but little interfered with. Treatment was begun by giving lycopod. and sulph. on alternate days, and this was succeeded by uran. nitric.

Four months later the patient visited Germany for some weeks, and took both wine and beer. On return he was better in every respect but that of sleep. The weight had fallen some pounds, and the daily output of sugar was much diminished in quantity.

By the end of the year, under the influence of lycop. and uran nitric., the quantity of urine had fallen to 66½ oz.; albumen and sugar were absent, and the weight had fallen to 13 st. 12 lbs. (*in puris naturalibus*). The insomnia remained as before, but was relieved by occasional doses of veronal. I strongly advised a visit to Carlsbad.—*Journal of the British Homeopathic Society*, July, 1907.

Gleanings from Contemporary Literature.

THE CROONIAN LECTURES ON PLAGUE.

By W. J. R. Simpson, M.D.

The Present Pandemic.

The present pandemic has no connexion with the plagues arising in Mesopotamia the chief features of which, as shown by Tholozan, were, during the latter part of the nineteenth century, those of comparative mildness, spontaneous cessation, and self limitation, irrespectively of preventive measures. It would appear that the strain of the Mesopotamian virus has become attenuated both in powers of attack and powers of diffusion and that it required a virus derived from a new source or from another endemic centre to produce a plague endowed with more virulent and diffusive qualities.

The Chinese endemic centre in Yunnan, from which the present pandemic is derived, is like the endemic centres in Arabia and India, between 5000 and 7000 feet high, and in this respect differs from some of the older endemic centres which are low lying. Yunnan has been known as an endemic centre of plague since 1870 but there are Chinese records which seem to indicate that the disease probably existed there for over 100 years, for mention is made of a strange and fatal rat disease prevailing at the end of the eighteenth century which also infected the inhabitants. There are no records discovered which make the endemic centre older than this and there is no evidence to show that the Black Death of 1348 arose from Yunnan. It is not known exactly when the present pandemic overflowed its boundaries and invaded the adjoining provinces of Kwangtung and Kwangsi. Plague had passed over the boundary several times during the Mahommedan rebellion in Yunnan and in 1867 reached Pahhoi, a small seaport on the southern coast of China, but there appears to have been no very extensive epidemic. As far as can be ascertained it was about the year 1890 that the disease began to show unusual activity. At that time the annual recrudescences in Mentze, one of the principal trading towns in the south-east portion of the province, became more severe and there was an extension of the disease to some of the towns situated on the West or Canton River and which have trade relations with Mentze and Canton. Gradually an extended area of the western parts of Kwangsi and Kwantung became affected and in January, 1894, Canton was attacked. Canton is the chief port as well as the largest and most important city in Southern China. It is only 80 miles from Hong-Kong, which, situated at the mouth of the Pearl River, contains a population that is mainly Cantonese, and so great is the intercourse between them that Hong-Kong has been styled the suburb of Canton. Hong-Kong became infected in May, 1894. Recrudescences of plague have occurred more or less in Canton and Hong-Kong since. Canton and Hong-Kong are the great marts and distributing centres for the produce of Southern China and have trade connexions with the southern parts of China, the neighbouring islands of the Pacific, and with India, Australia, Japan and America. They were accordingly favourably situated as distributors of plague to all those countries adjacent and distant with which they had commercial relations. Their ships carried infection to the seaports of other countries, and these in their turn infected other places. The course of the spread of plague has differed from all previous pandemics in that

its distribution has been by sea routes, in contradistinction to former pandemics which spread by land routes and coasting vessels. With the exception of India and one or two places in South Africa and America the infected localities are mostly on the coast, and the history of their infection is importation of the disease from some infected port with which they carry on commercial relations. Any circumstance which increases to an unusual extent the transport of goods from infected ports increases the risk of importation of the disease. Thus, the war in South Africa, with its enormous shipments of grain and fodder from the Argentine and from India, whose ports were infected with plague, introduced the disease into Cape Town and Port Elizabeth, where the rats in the docks were the first to become infected. The Russo-Japanese war was, fortunately for Europe, out of the zone of any badly plague-infected district.

Many places have been infected in different parts of the world, but none outside India have hitherto given rise to any very serious epidemic. Still, notwithstanding its apparent inability to develop into an epidemic, yet the disease has in many instances when imported into a locality shown a remarkable persistence as displayed by the annual recurrence of sporadic cases at the season of the year favourable to epidemic plague. The potentiality of plague becoming epidemic in such localities is there all the same and no country is safe while it retains infection.

The Epidemic in India.

At present, however, the chief interest of this pandemic lies in India. Imported into the city of Bombay in 1896 from Hong-Kong it broke out in epidemic form in September of the year, and by the end of April, 1897, when the first epidemic was over it had caused 11,000 deaths. Every year there has been a recrudescence and the total number of deaths from plague in Bombay since its appearance till the end of 1906 is 150,000. From Bombay city it spread to the Bombay Presidency, chiefly by coasting boats and by the railways carrying fugitives infected with plague to their native villages. By December, 1897, 50,000 deaths had occurred in the Presidency, and a few deaths in some of the other provinces. It has continued ever since in this Presidency and has up, to the end of April, 1907, caused 1,500,000 deaths. Gradually the disease has spread to the other provinces of India, affecting some severely and some lightly, and the grand total of deaths from plague in India, as shown in the following statement of annual deaths, amounts to over 5,000,000 :—

Total Recorded Annual Deaths from Plague in India.

Years.	British Territory	Native States.	Total.
Sept., 1896. to end of 1896	57,000	—	57,000
1898	89,200	27,000	116,200
1899	102,300	36,600	138,900
1900	73,500	19,200	92,700
1901	234,600	46,100	280,700
1902	445,200	126,900	572,100
1903	701,800	179,000	880,800
1904	938,000	203,300	1,141,300
1905	940,800	128,300	1,069,100
1906	—	—	332,000
1907 (first 4 months)	—	—	641,000
			5,321,800

The population of British territory is 232,000,000 and that of the native states 62,000,000. The 5,000,000 deaths represent accordingly one death in every 60 of the inhabitants of India. The mortality during the later years is very much greater than in the earlier, the deaths in the last five years and four month reaching over 4,500,000.

The fact that India is an immense country with nearly 300,000,000 inhabitants has often been considered as minimising the gravity of the situation which 5,000,000 of deaths would otherwise represent, and the argument is employed that in such a large country it is impossible to deal with the disease. 1,000,000 deaths a year in a population of 300,000,000 is viewed only as 1 death in 300 of the inhabitants and from that point appears not to have much influence on the vast population. It is once more the fallacy of averages. There is the old story of the man who assured that the average depth of a river was four feet, endeavoured to cross it and was drowned. He had not reckoned, that it might be shallow in some parts and deep in others. Plague is not epidemic over the whole of India. But, even if it were, and admitting that India, including Burma, is greater by 12,000 square miles than the whole of Europe, excluding Russia, Poland, and Finland, most people will allow the mortality is serious. 5,000,000 deaths from plague in Europe would be considered on this side of the Red Sea appalling in whatever way it was distributed. And if it happened that many of the countries in Europe were more or less free from plague and that the disease concentrated itself on three or four countries, such as France, Italy, Austria, and Great Britain and Ireland, causing in these nearly 4,500,000 deaths out of the 5,000,000, the mortality would be viewed as a catastrophe of the first magnitude. But if in addition to the loss of the 4,500,000 of inhabitants there were owing, to the recurrence of the disease, a prospect of several more millions being destroyed in those places already attacked, and that there was the further danger as the pandemic developed of the other countries—such as Germany, Holland, Spain, Greece, and Turkey—being attacked in the same way, then the situation of Europe would be similar to that of India to-day. This will serve to give some conception of the tragedy which is going on in India at the present time and of the future perils of that unhappy country. It is a misconception, fraught with the greatest danger, to suppose that in India the plague is only causing a death-rate of 3 per 1000, as was stated in the House of Commons during the debate on the Indian Budget. The figures giving the total number of deaths in the different provinces of India since September, 1896, exhibit a very different degree of incidence and severity in each. The Madras Presidency has escaped with a comparatively small number of deaths; so have most of the other provinces. Four provinces have hitherto borne the brunt of the epidemic in India out of the 15 presidencies, provinces, and States into which India is divided. These are the Bengal Presidency, with a little over 500,000 deaths; the Bombay Presidency, with a little over 1,500,000 deaths; the United Provinces, with nearly 1,000,000 deaths; and the Punjab, with over 1,750,000 deaths.

The annual returns for these provinces are approximately represented in the following figures :—

Year.	Bombay and Sindh, population 22,000,000.	Bengal population 74,000,000.	United Provinces, population 47,000,000.	Punjab, population 25,000,000.
September, 1895, to end of 1897 ...	57,000	.....	80	179
1898	104,000	166	116	1,800
1899	117,000	3,000	6	250
1900	38,000	37,000	116	500
1901	158,000	78,000	9,000	18,000
1902	217,000	32,000	43,000	222,000
1903	340,000	65,000	80,000	210,000
1904	281,000	75,000	179,000	402,000
1905	96,000	126,000	383,000	389,000
1906	71.000	50,000	57,000	98,000
To May 11th 1907 ...	52,000	50,000	223,000	432,000

The Epidemic in the Punjab.

The province of the Punjab which has lost nearly 1,750,000 of its inhabitants is in size less than one-twelfth the total area of India and it contains less than one-eleventh part of its population. It is slightly larger in area than Great Britain but is smaller than Great Britain and Ireland. Its population, including the Native States, is 25,000,000 without the Native States 20,000,000 against the 43,000,000 of Great Britain and Ireland, so it is not a large province, and the loss of 1,750,000 out of 25,000,000 can only be viewed as an appalling disaster. If plague had destroyed 3,000,000 of the inhabitants of Great Britain and Ireland in ten years it would have represented proportionately what the Punjab has lost during that time with its smaller population. The plague began very slowly in the Punjab and took six years before causing 250,000 deaths. There were 179 deaths in 1897, 1871 in 1898, 253 in 1899, 525 in 1900, then, as reported by the Sanitary Commissioner for the Province, all restrictions were removed and a new policy was introduced ; in 1901 there were 18,877 deaths and in 1902, 222,533 deaths. The great mortality has been during the past four and a half years and the greatest during 1907, when over 500,000 deaths occurred during the first five months, which is the epidemic season. 800,000 deaths from plague occurring in Great Britain and Ireland in five months would represent the intensity of the epidemic in the Punjab during the early months of this year. There is no comparison between 800,000 deaths and 54,000, which was the largest epidemic of cholera in England in 1854-55 and which was considered to be appalling in this country. During the week ending May 11th, when the climax of the present year's epidemic in the Punjab was reached, there were 60,000 deaths from plague in that province, which in the British Isles would be represented by 100,000 deaths in one week in an epidemic of the same intensity. The condition of affairs in this country with 100,000 deaths from plague taking place in one week in the British Isles as the climax of an epidemic which in the course of five months had destroyed 800,000 of its people would be similar to that now existing in the Punjab at the present time. The state of mind of the rest of the inhabitants of Great Britain and Ireland under such a catastrophe coming on the top of a devastation which had

previously destroyed over 2,000,000 would not be one of calmness and contentment. In the first 12 weeks of the year there were 145,000 deaths from plague in the Punjab; in the next six weeks 286,700 deaths. During these six weeks the plague deaths were as follows :—

Week ending	April	6th	...	...	34,651 deaths.
„	„	„ 13th	...	...	39,084 „
„	„	„ 20th	...	...	47,047 „
„	„	„ 27th	...	...	54,204 „
„	„	May 4th	...	...	51,305 „
„	„	„ 11th	...	...	60,400 „

These facts will dispel the view that the plague is a small thing in India scattered over a vast continent. The effect in the Punjab may be gathered from the following extract from the *Times of India* of June 1st, 1907 :—

A picture of some of the results of the terrible epidemic in the Punjab is given by the "Statesman's" Simla correspondent. To dismiss this epidemic (he writes) with the statement that the people of the Punjab have been dying from it for some time past at the rate of 50,000 a week gives but a faint idea of the deserted villages, the crops rotting upon the ground over wide areas for lack of men to reap them, and the breaking up of homes and family life inseparable from such a calamity. The people have learnt to quit their villages and to camp out in the open when the disease appears and the worst is now over for the year, since the hot winds of May invariably reduce the mortality. But the evil has attained such extraordinary magnitude that it is affecting the whole outlook of the people. In Simla carpenters have become difficult to procure, because the Jullunder district, where most of them lived, has suffered so terribly. The plague is an undoubted factor, though perhaps not the principal one, in connexion with the much-discussed unrest. It is also becoming important in changing the relations between population and sustenance since the survivors inherit the property of those who succumb and grow less inclined in consequence to work for themselves.

In another extract from the same paper it is stated that "natives of Rawalpindi who have relations in the plague-infected villages will not go to tend their sick; others have left the corpses or belongings of deceased relatives to the mercy of the village rather than risk plague. Firewood is not obtainable to burn the dead, so timber from the houses is being utilised and many Hindus are burying their dead." This last is against the religious views of the Hindus, but their necessity has brought it about.

The concentration in the Punjab does not mean an equal distribution of the disease over the whole province. The mortality falls with unequal intensity on the divisions and districts into which the province is divided.

Table I. gives the deaths registered in the rural circles of the Punjab with the death-rates of each from 1901 to 1905 inclusive. The statistics are not available for the great epidemic of 1907 or for the lesser one of 1906, but those which are available show the great incidence of plague on certain rural areas. In 1902 the district of Ludhiana was worst affected and had a death-rate from plague of over 70 per 1000. More than a fourth of the deaths in the rural circles occurred in this circle. Umballa came next with 30 per 1000 and Sialkot with 26 per 1000; one-third of the circles were not infected. In 1903 Gujranwala was the worst infected circle and had a death-rate from plague of 65 per 1000; Amritsar and Jullundur came next with a mortality of 30 per 1000 each; seven circles still remained unattacked. In 1904 only

one circle remained free from the disease. Shahpur had a death-ra from plague of 74 per 1000 of its inhabitants, Sialkot 48 per 10 Gurdaspur 46 per 1000, Ludhiana 45 per 1000, Gujrat 41 per 10 and Jullundur 35 per 1000. In 1905 the recurrence in the rural circ was not quite so severe as in 1904, still Rohtak had a death-rate fr plague of 52 per 1000, Gurgaon 40 per 1000, and Ludhiana 34 per 10 Similar rates are to be noted in the towns of the Punjab. For instan in 1902 Rupar, a small town in the Umballa district had a death-ra of 90 per 1000 of its population from plague. In Jamke, a small to in the Sialkot district, a death-rate of 116 per 1000 from plague w registered.

In 1902 the Sanitary Commissioner reports that in the minor tow of the Ludhiana district the ravages committed were fearful. Raik and Machiwara were almost decimated. Jagraon lost 11 per cent. its population and Khana no less than 14 per cent. In 22 villages t death-rate from plague ranged from 20 to 40 per cent. of the populatio These death-rates, or depopulation of villages, are similar to those whic are recorded as having occurred in some of the villages of the Bomba Presidency. There some of the villages lost 33 per cent. of thei inhabitants. As is always the case in plague, some places escape lightl in one year while others are almost depopulated.

Since 1903 special reports on plague in the Punjab have been discontinued and in that year the administration of plague was handed over to the Inspector-General of Civil Hospitals, an officer whose duties in ordinary times are always of an onerous character and who could have no time to devote to this extra work. Owing to these changes the information is henceforth scanty. In 1904, however, there can be gleaned from the few remarks made by the Sanitary Commissioner, when treating of the vital statistics of the province, the havoc which the plague caused in some localities. Thus he says: "As an instance of the fearful increase in the mortality caused by plague, it may be noted that the death-rate of the district of Shahpur in April when plague was most virulent was *twenty-three times* higher than in August, by which time the disease had entirely disappeared. The total number of deaths in April in that district was 17,889, of which no fewer than 17,012 were ascribed to plague, as against only 781 in August, including one death from plague. The death-rate for Shahpur for April was *415 per mille per annum*." With this rate in the district of Shahpur as a whole, it is safe to say that similar rates of 20 and 40 per cent. occurred in some of the villages as were recorded two years previously in the villages of Ludhiana. There was in 1904 an abnormal increase in the total urban death-rate which the Sanitary Commissioner states was accounted for by plague, and he mentions the high rate of 145 per 1000 in Hodal in the Gurgaon district and of 125 per 1000 in Miani in the Shahpur district, whose death-rate from plague was 100 per 1000 in each case.

If the totals for the five years in the rural circles be taken it will be seen that the Ludhiana district lost 100,000 of its inhabitants out of 586,000, or more than 1 in 6. Sialkot lost 103,000 out of 994,000, or nearly 1 out of every 9. Gujranwala lost 101,000, or nearly 1 in 8 of its population. I have not been able to add to these losses in the rural districts those of 1906, which was a comparatively light year, and those of 1907, which has been worse than any of the previous years, but the figures as they stand demonstrate a devastation on an immense scale, and which with the losses of 1906 and 1907, together with what is to come, means a mortality comparable with even that of the Black Death.

Though not able to give the statistics of the rural circles for 1906 and the earlier months of 1907, I am able to give them for the divisions and districts of the Punjab as a whole from the year 1901 to May 11th, 1907. There are five divisions in the British territory of the Punjab Province. These divisions contain a population of 20,000,000. Since 1901 they have lost 1,500,000 of their inhabitants—i.e., more than one-fourteenth of their total population. The Jullundur and Lahore divisions have suffered the most. Jullundur has lost 441,000 of its inhabitants out of 4,269,000—i.e., more than one-tenth; and Lahore has lost 665,000 out of 5,550,000, or nearly one-eighth. In these two divisions the incidence on some of the districts was very heavy; thus the district of Ludhiana has lost 140,000 out of 673,000 of its inhabitants, which is more than one-fifth of its population; another district of Gujranwala has lost 156,000 of its inhabitants out of 890,000, or slightly less than one-fifth of its population.

There are 29 districts in the Punjab and eight districts have lost less than 1 per cent. of their population, seven districts under 5 per cent. of their population, three districts over 5 and under 10 per cent. of their population, nine districts over 10 and under 15 per cent. of their population, and two districts have lost 17 and 20 per cent. of their population. With the districts as a whole giving such figures it is certain owing to the fact that plague is never evenly distributed, that some of the villages have lost half their population.

The Prevention of Plague in India the most Important Question for India and England at the Present Time.

The disease attacks the able-bodied and the strong, the bread-winners and protectors of the family. It is the sturdy peasants of the Punjab, the sowers and tillers of the soil, the producers, that are dying in such enormous numbers, and if they continue to die the effect, as has been the case in all epidemics, will be a serious disorganisation of the social and economic conditions of the province. Already in 1903 the Lieutenant-Governor of the province recorded that there had been an increase of crime and although the effects of the plague had not been immediately apparent he anticipated that trade, agriculture, and education, recruiting and other interests were bound to suffer. These anticipations and more have happened and the state of the Punjab has become socially and politically a serious cause of anxiety. It has recently been announced in the *Times* that there is a deficit of 18 lacs of rupees in the Punjab revenues of last year and that in many places the crops are standing unreaped because there are no harvesters. The silence of those unharvest fields, together with the figures of mortality, should bring to everyone's imagination a very clear realisation of the awful devastation of the Punjab. Nor are the effects likely to be confined to the Punjab, for there can be no moral and material progress of India as a whole when one of its smaller provinces is being more than decimated, and when there is danger of the disease attacking other provinces with a similar intensity.

It will be recognised from the foregoing facts that the dying millions in India present a problem of the greatest urgency and danger. The plague if left as it has been within recent years to take its own course bids fair in such circumstances to overwhelm not only India but also to be a danger to the world.

Modes of Dissemination and Channels of Infection.

Turning to the preventive aspects of plague the question arises. Are the principal modes of dissemination and the channels of infection known?

It is now accepted that the importation of plague into a healthy locality can be effected by an infected human being, or an infected rat, or infected clothes. Observations have been so numerous as regards these agencies as to remove all doubt on this point. Dr. E. E. Klein has further shown by some important experiments that infected grain may also be an agent in the importation of the disease. By feeding rats and guinea-pigs with grain which had been contaminated with old cultures of plague, and which was then dried, he was able to cause plague in those animals, so that grain which has been infected with infective material from plague rats or human beings may, on importation into a healthy seaport, give plague to healthy rats on shore which eat the contaminated portions of the grain. The association of corn ships with plague is a very old one. In recent years the association has been ascribed almost wholly to plague rats on board ship which have left the ship and infected healthy rats on shore, but now infected grain must be added as a likely agent.

Once imported, the dissemination of the disease is effected by similar agents as those bringing about importation. Rats, clothes, human agency, and food play their respective parts. It is now almost universally admitted that, except in pneumonic cases rats are the principal agents in the dissemination of the disease, though personal contact, as in typhoid fever, also plays its part. They bring the infection into houses connected with their subterranean passages. In Hong-Kong one could trace many of the subterranean passages with their infected rats and their connexion with the distribution of the disease in the houses. A systematic bacteriological examination of rats trapped and found dead proved that plague in the rats of the locality preceded plague in man. So constantly did this occur that as soon as plague-infected rats were discovered measures were introduced as in Cape Town but on a more extended and systematic basis, of treating the house or locality as plague infected. In other words, a policy of forestalling plague was adopted. The precedence of rat plague in relation to plague epidemics was observed in Bombay, the Punjab, Calcutta, South Africa, Australia, and Southern China. The value of the Hong-Kong observations lay in the direct evidence obtained by scientific and precise methods not only of the direct relationship which the precedence of rat plague bore to human plague but also of the important role which the rat plays in the spread of the disease from house to house and in groups of houses. Similar observations were made by Dr. J. Ashburton Thompson in Sydney, but under different conditions. The Sydney out-breaks are comparatively mild, the mortality being under 40 per cent., and consist mainly of bubonic cases with but few septicæmic cases. Probably the type in Sydney was the same as that met with in Cape Town, where one of the marked features was absence of cerebral symptoms and intestinal disorders, differing in this respect from the type met with in Poona in 1897, but which in 1907 seemed to me to have changed, presenting fewer nervous symptoms. The Hong-Kong outbreaks are exceptionally virulent, the mortality being over 80 and 90 per cent., and with a large percentage of septicæmic cases.

The problem of the dissemination of the disease by rats is not solved by simply proving that rats are infected with plague in a house or locality before human beings, or by the fact that if measures are taken to dispose of the infected rats human plague will not occur, and, *vice versâ*, if no measures are taken plague will attack some of the inmates of the house. The question naturally arises, How is the infection of the rat transferred to man? There are two views and the upholders of each have a tendency to claim that theirs is the only way. The more that is learnt of plague the more it is evident that there is no only way.

The Flea Theory.

Dr. Ashburton Thompson had exceptional opportunities of studying the small outbreaks in Sydney and on that study he came to the conclusion that Simond's theory that the flea on the rat, leaving the rat dead from plague and then biting man, transfers plague from the rat to man, explained the phenomena connected with plague. That theory, of which Dr. Ashburton Thompson is the champion, has recently received very valuable support in its favour from the very important experiments made by the Indian Plague Commission and which are the outcome of certain researches by captain W. G. Liston, I. M. S., to whom much credit is due for his excellent work in this direction. These experiments established first the accuracy of Simond's and Gauthier's and Raybaud's experiments as to the power of infected fleas from plague rats causing plague in healthy rats: 61 per cent. of the white rats, which are very susceptible to plague, and 52 per cent. of the Bombay rats experimented on contracted plague by the transference to healthy rats of fleas from infected rats. Similarly, guinea-pigs allowed to run free in plague houses in many intances attracted a large number of fleas, most of which were rat fleas, and 29 per cent. of the guinea-pigs contracted plague. Animals in cages protected from fleas by a layer of "tanglefoot" did not contract plague, while animals in cages not so protected developed plague to the extent of 25 per cent. Out of 247 fleas caught on the "tanglefoot" 60 per cent. were human, 34 per cent. were rat, and 6 per cent. were cat fleas. Plague-like bacilli were demonstrated in the stomach contents of one out of 85 human fleas dissected and of 23 out of 77 rat fleas. The commission carried the observations a stage further, and by the transference of infected fleas from guinea-pigs started an epidemic of plague among healthy guinea-pigs which was maintained by the introduction of fresh infected fleas.

That the flea is an important agent in spreading plague from rat to rat and in the maintenance of rat epizootic is evident from these experiments. The rat flea, variously named by different observers Pulex cheopis, Pulex murinus, and Pulex pallidus, and commonly found on the Mus rattus was the active agent in the transfer of the disease from rat to rat and from rat to guinea-pig. To the extent to which the Pulex cheopis is an agent in keeping up the epizootic in the rat, it will account for the epidemiology of plague but it is not a necessary deduction from this that Pulex cheopis plays the same active part in the transfer of the disease from the rat to man as it does from rat to rat, though it may be credited with an indirect influence in the maintenance of the disease in man owing to its being an active agent in the dissemination of the disease in rats and possibly in other animals.

There is much in favour of Pulex cheopis playing an active part in the transfer of plague from the rat to man. There can be no doubt that Pulex cheopis, unlike Pulex fasciatus, another common rat flea, bites man. Pulex cheopis is only found occasionally in ordinary circumstances on man. As a rule, it is either the human flea or Pulex canis that is usually found on man. Hilger identified 59 per cent. of over 2000 fleas which he found on man as being Pulex canis, and it may possibly have been this flea which, owing to its wide distribution on rats, dogs, cats, and man, gave rise to older views that dogs, cats, and fowls from infected houses spread plague even when these animals were unaffected. In certain circumstances Pulex cheopis may be found in numbers on man, such as the instance given by Liston, in which 46 per cent. of the fleas found on the persons of inmates in an infected house proved to be Pulex cheopis.

In Hong-Kong in 1902 I was able to infect two monkeys by placing rats which had died from plague and which were covered with fleas in the same cages as the monkeys but in compartments which prevented any possible contact between rat and monkey but which allowed of a free passage of fleas from rat to monkey. Both monkeys fell ill but recovered. The Indian Plague Commission has in a similar fashion on two occasions succeeded in transferring the plague of the rat to monkeys by the agency of fleas. As it is in the monkey so probably it is the case in man. No direct experiments can be made to prove this, but the accidental infection in man caused by the handling of rats dead from plague and on which Simond's first founded his theory would appear to bear the interpretation given them by the laboratory experiments.

On the other hand, taking the most liberal interpretation of the flea theory based on the experiments mentioned, it is doubtful whether the flea theory will account for more than a certain percentage of the fatal bubonic cases which may vary in different circumstances in different localities. Out of ten experiments with monkeys by the Indian Plague Commission eight failed and in my own experiments the monkeys recovered. Moreover, the fact that infants under one year of age are more or less immune to plague militates against the flea being as important an agent in the causation of human plague as of rat plague. The incidence on infants is very small. This has been shown by Lieutenant-Colonel C. J. Bamber, I.M.S., in the different Punjab epidemics. This immunity is shared with the aged. Fleas probably are not attracted to old people but the same cannot be said with reference to infants who sit, lie on, and crawl about the floor. The food of infants fed as a rule at the breast of the mother differs from that of the adults in that it is pure and not subject to contamination with plague virus and herein probably lies the explanation of the immurity of the infant.

The Food Theory.

The difference in the type of the disease in Sydney as contrasted with Poona and Hong-Kong has already been mentioned. More post-mortem examinations are made on plague cases in Hong-Kong than in any other part of the world, and it is on the observations there both on man and animals that the theory has been formulated that the ingestion of food contaminated with the plague virus is the cause of septicæmic cases of plague.

Wilm in 1896 found that in 20 per cent. out of 150 necropsies the mucous membrane of the stomach or intestines showed lesions with hæmorrhages, and he was successful in causing plague in fowls by feeding them with plague material and with pure cultures of the plague bacillus. He also succeeded in infecting a pig fed with the spleen of a man who had died from plague. In 1897 the German Commission in Bombay was successful in causing plague in rats, a mongoose, a squirrel, and monkeys by feeding experiments, but failed with mice, guinea-pigs, dogs, and pigs. The Austrian Commission was successful in similar experiments with guinea-pigs, rats, mice, and cats, but failed with dogs, pigeons, hens, and a mongoose.

In 1902 I had the opportunity of seeing with Dr. W. Hunter a large number of necropsies on plague cases in Hong-Kong and we were able to confirm the great frequency with which the intestines are the seat of primary hæmorrhagic lesions and the mesenteric glands swollen and hæmorrhagic. Later Dr. Hunter has given a detailed description of the pathological changes which are observed in the alimentary canal and which are practically present in all the septicæmic cases of plague. The stomach shows well-marked congestions and hæmorrhages; some of the

hæmorrhages may occasionally be of the size of a dollar piece. The changes met with in the small intestines are even more severe than those in the stomach. Petechial hæmorrhages and blood extravasations with necroses, forming not infrequently superficial ulcers, are common. Peyer's patches and solitary follicles are swollen and may be hæmorrhagic, and in some parts inflammatory changes extend through the walls of the intestine. The mesentery is frequently affected by extensive hæmorrhages and the contained lymphatic glands are enlarged and hæmorrhagic. The lymphatic glands standing in relation to the ileum and cæcum are almost always affected. They are frequently double their ordinary size and on section are œdematous, with minute blood extravasations into their parenchyma. With Dr. Hunter and Dr. Matsuda, a Japanese medical man lent to the Government of Hong-Kong by Japan, I was able to carry out a series of feeding experiments which established that poultry, calves, pigs, sheep, rats, and a monkey contracted plague by feeding. We failed to cause plague in dogs by feeding. These observations on men and on animals led me to the conclusion that septicæmic plague is in most cases contracted by the alimentary canal; an additional fact which lends support to this view is the frequency of abrasions and denudations of the alimentary canal in Asiatics. Dr. H. Fraser, who is engaged in a research on the condition of the intestines, showed me in his laboratory at Kuala Lumpur in the Malay State microscopical specimens of the intestines of Chinese and Indian in which he had found over 50 per cent. with abrasions or ulcers. Dr. Hunter has also shown that when premonitory symptoms of plague do manifest themselves the patients frequently suffer fron gastric and intestinal disorder with diarrhœa and in these cases examination of the blood shows a septicæmic infection.

More decisive than even the foregoing experiments on animals are those made by Dr. Klein in this country because of the convincing histological work which he has associated with them. He has proved beyond all question of dispute that the plague bacilli taken in contaminated food multiply while the food is in the intestines, enter through the lymph channels or lacteals of the intestines, and invade the blood in swarms. The only qualification is that the microbes shall be so protected as not to be affected by the gastric juice.—*The Lancet*, 13th July, 1907.

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