ness be established by skin tests before treatment is undertaken. This is accomplished by applying the various dilutions of the pollen or pollens to which the patient is sensitive to small scratch marks made on any of the flexor or internal surfaces of the extremities. The dilution next higher to the one that gives a reaction is the dilution with which treatment may be begun. For instance, the patient is tested with dilutions of ragweed extract of 1 : 100, 1 : 500, 1 : 1,000, 1 : 5,000, 1 : 10,000, 1 : 50,000 and 1 : 100,000. If the patient reacts to all the dilutions up to and including 1 : 10,000, the dilution of 1 : 50,000 is that which should be used with which to begin treatment. The initial injection should be 0.2 c.c. of this dilution, and the dosage should be increased by 0.2 c.c. each succeeding time until 1 c.c. of this dilution is given. Then 0.2 c.c. of the 1 : 10,000 dilution should be injected, and so on as before.

If redness, swelling and itching occur at the site of the injection, it is wise either to repeat that dose at the next treatment or even to diminish it if the action is very severe. When the treatment is begun, as is usually the case, about six weeks before the known onset of the attack, the interval of treatment should not be less than four days. When it is possible, we are now giving weekly injections throughout the year, and our results have been far better than when the prophylactic treatment has been of short duration, just preceding the hay-fever season. This plan is being carried out in the hay-fever and asthma clinic at New York University and Bellevue Medical College. When the stronger dilutions of pollen extract are used, such as 1 : 500 and 1 : 100, a great deal of caution must be exercised in increasing the dose, because even a slight increase may precipitate an attack of anaphylaxis; and I would warn those who arrive at these dilutions that the increase in dosage should not at any time be greater than 0.02 c.c.

Drug.—During an attack of bronchial asthma, certain drugs may be used to alleviate the patient's suffering. The solution of epinephrin (adrenalin) chlorid, 1 : 1,000, when given hypodermically, has the effect of overcoming the spasmodic constriction of the bronchial tubes, and thus stops the paroxysm or ameliorates the symptoms. The effects of this drug begin to make themselves manifest within fifteen minutes, and last anywhere from one half to two hours. For this reason it is necessary to administer repeated doses while the attack, which may last for weeks, is in progress. The patient gradually becomes accustomed to repeated doses of epinephrin, so that the amount given has to be increased from time to time, and finally, the patient develops a complete tolerance to the drug and no more relief is obtained from its administration. The constant exhibition of this drug is not unattended by baneful after-effects; its immediate action of precipitately raising the blood pressure must necessarily have a damaging influence on a heart even in the healthiest condition. Besides this, its constant use is known to produce a sclerosis of the larger arteries. Epinephrin should never be given intravenously, or while the patient is in the physician's office, as I have seen very harmful effects from such practice. Recently Dr. Hugo R. Miller has been using the active epinephrin extract in oil and, when given prepared in this way, he finds that the effect of the drug comes on more gradually and lasts much longer. The adrenalin inhalant, from our experience, is inactive and does not produce the results which Dr. Miller claims for his preparation.

Atropin given subcutaneously or intramuscularly in gradually increasing doses, up to the point of tolerance, and especially when combined with small doses of morphin, will give the patient a great deal of comfort. Potassium iodid, 1 gm. every four hours, helps to render the bronchial secretions less tenacious and more liquid, and thus makes the coughing milder and of shorter duration. Preparations containing the salts of ammonia, especially liquor ammoniae anisatis, disturb the stomach less than potassium iodid, and are just as efficacious. A mixture containing tincture of belladonna, chloral hydrate and syrup of hydriodic acid has been found to make the patient comfortable in the majority of instances. It is surprising how much relief is obtained by inhaling the fumes from a smouldering powder made from nine parts of stramonium leaves and one part of potassium nitrate. This is the basis of most asthma powders on the market. In very severe cases, nothing short of large doses of morphin hypodermically will give the patient the necessary respite and mental rest from the distressing paroxysms. When the bronchitis persists between attacks, nothing is more efficacious to alleviate the cough and sustain and soothe the patient than Thompson's mixture of linseed oil [1] with codein or compound tincture of opium. Applications of cocain and epinephrin directly to the mucous membrane of the bronchial tubes through a bronchoscope during an attack certainly give respite from symptoms; but the duration of the relief is too short to warrant this trying procedure.

NUTRITIONAL EDEMA AND "WAR DROPSY" *

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Before the recent war, medical literature contained frequent references to the type of edema now recognized as "war edema." With the clinical picture in mind presented by meager reports that have appeared in the American [1] and British scientific journals of recent publication, a somewhat extended study of wars, famines and epidemics of the past has proved fruitful in bringing to light evidence of the prevalence of edema of this type under varying conditions of insufficient and inadequate food. This edema resembles that of renal disease. In mild cases it may be confined to the lower limbs, but in the severe type the edema may extend to all parts of the body. There is no albuminuria. Accompanying this edema there are emaciation, muscular weakness, depression, anemia, and very frequently gastro-intestinal disturbances.

While the term war edema is not found in early medical literature, there is much evidence that the condition known by this term has been of frequent occurrence in the past. In giving a name to this disease, the authors usually express the chief etiologic factor with the most pronounced clinical symptom, so that war edema, prison dropsy, hunger swelling, epidemic dropsy, edema from inadequate food, deficiency edema, edema without albuminuria, and many similar

1. Mixture of linseed oil (Thompson) consists of: dilute hydrocyanic acid, 10 c.c.; oil of wintergreen and oil of cinnamon, of each, 8 c.c.; glycerin, 20 c.c.; simple syrup, 300 c.c.; Irish moss, 15 gm.; linseed oil, 450 c.c., and water, 780 c.c.

* From the Otho S. A. Sprague Memorial Institute.

1. War Edema, Current Comment, J. A. M. A. 70: 627 (March 2) 1918. Warthin, A. S.: War Edema, Int. A. M. Museums Bull. 7: 196 (May) 1918.

terms have been used. In civil practice previous to the war, essential idiopathic or primary edema, salt edema in children, alimentary dropsy, anemic dropsy and edema following gastro-enteritis were some of the more common terms employed.

Prinzing [2] makes no mention of war edema from the time of the Peloponnesian Wars, from 430 to 425 B.C., to the siege of Port Arthur in 1904. In a large number of these etiologically related conditions, edema appears as a symptom rather than as a specific disease. In the destruction of the French army before Naples in 1528,[3] "those soldiers who were not confined to bed in their tents were seen with pallid visages, swollen legs, and bloated bellies, scarcely able to crawl."

Vacher,[4] writing of the conditions of childhood during the siege of Paris in 1870-1871, finds that the effect of insufficient nourishment showed itself in progressive emaciation, edema of the integument, anemia, and uncontrolled diarrhea, which were characteristic symptoms of the hunger fever which decimated the infant population. Between 1877 and 1880, there broke out in Calcutta a peculiar disease to which the term "epidemic dropsy" was applied.[5] This disease followed an extensive famine in southern India.[6] It persisted in epidemic form until 1880. The number of cases increases in each cold season and falls off in each hot season. It has continued to appear in Calcutta sporadically. Neighborhood and family groups continued to be reported until 1915.

Edema of war occurred during the Napoleonic campaigns, the siege of Paris, and in the concentration camps during the Boer War, when it was known as epidemic edema.[7] Falta [8] of Vienna mentions that the disease was known in Russia during famines before the war, and that the expression "swollen from hunger" was current in the affected districts. Landa [9] related that in August, 1915, when the City of Mexico had been the seat of military operations for two or three months, the appearance of numerous cases of edema began to be noted. Starvation edema was reported by Dr. Patterson [10] in 1899 after a season of famine in China. Holst,[11] in connection with his researches in ship beriberi and scurvy, furnishes this interesting group of historical data:

> Many dropsical cases were observed during the Crimean War when scurvy was prevalent. Dropsy without sore gums occurs every year on board the French fishing vessels off the coast of Newfoundland. During the first part of the nineteenth century dropsy was common in European and American prisons. This prison dropsy is stated in 1847 to have been, besides typhoid fever and consumption, the most prevalent cause of death in forty-one prisons in England, France and North America. In 1857, it caused one-half the deaths in a prison in Breslau.

RECENT REPORTS OF EDEMA IN EUROPE

The first record of war edema in the recent war was made in 1915 by Strauss, who described the "hunger disease" in Russian Poland and Galicia, where the poor had an insufficient and monotonous dietary, and were exposed to war epidemics. About the same time, in July, 1915, Budzynski and Chelchowski [12] described a series of 110 cases of a peculiar affection occurring among the inhabitants of certain towns in Poland as a result of insufficient and inadequate food caused by the German occupation. The name "hunger swelling" was applied to this disease because its most characteristic feature was the presence of marked edema, recalling that encountered in dropsical beriberi. All the patients suffering from the disease were in a state of semistarvation.

In Germany, war edema first made its appearance in prison camps in July, 1915. Rumper at first considered relapsing fever responsible, but in 1916, when many cases with dysenteric symptoms occurred in prison camps free from relapsing fever Rumper and Knack [13] regarded dysentery as a predisposing cause. At this time, cases were reported among Russian soldiers at the front. Early in 1917, there were many cases in the civil population and labor battalions in Germany, and in the spring, cases appeared in Austria among civilians, especially workmen, but rarely among the troops. The disease appeared in Vienna with great suddenness. In 1917, marked attention was given in German medical journals to this peculiar disease, which seemed to have become widespread throughout Germany. The first cases noted in Berlin were in January, 1917.

Guillermin and Guyot [14] made personal observations and reported in March, 1919, that insufficient food in Russia, Germany and Austria had caused a specific disease of hunger which was known as hunger edema. These authors described the disease as it occurred among French prisoners of war.

In Poland, the patients were in a state of semistarvation, none having eaten meat for several months and some not since the beginning of the war. The cases occurred almost exclusively among the poorest of the people and the unemployed factory hands, who were without money to buy food at the famine prices. The staple diet of the people consisted of potatoes supplemented by small quantities of soup and bad bread on certain days. The amount of potatoes eaten averaged 5 pounds for each person daily, a dietary which caused diarrhea and eventually led to most of the food's being passed through the gastro-intestinal tract undigested. The causes of the disease, according to the Polish authors Budzynski and Chelchowski, were lack of proper food, especially the absence of fats, and the large amount of bad potatoes consumed. Maase and Zondek,[15] in agreement with other authors, consider the cause of the disease to be underfeeding, resulting especially from the diminished quantity of fats. Another factor suggested was the amount of fluid ingested. Owing to the changed conditions, most of the sufferers had been taking a more watery diet than normally, in the form of soups, turnips, etc., and the occurrence of diarrhea was fairly common. Schiff [16] suggests that this purely war disease has obvious similarities to beriberi and other diseases resulting from a lack of vitamins. Falta says that all of the patients

2. Prinzing, F.: Epidemics Resulting from Wars, New York, Oxford University Press, 1916.
3. Hecker, J. F. C.: Epidemics of the Middle Ages, London, 1846, p. 231.
4. Vacher: La mortalité à Paris en 1870, Gaz. méd. de Paris, 1871, p. 9, cited by Prinzing (Footnote 2).
5. Green: Epidemic Dropsy in Encyclopedia of Medicine and Surgery, Edinburgh and London **2**: 422.
6. Leys, J. F.: Epidemic Dropsy, Reference Handbook of Medical Sciences, Ed. 3, New York, William Wood & Co., **3**: 696, 1914.
7. Maliwa, E.: Wien. klin. Wchnschr. **30**: 1477, 1917.
8. Falta, W.: Wien. klin. Wchnschr. **30**: 1736, 1917.
9. Landa, E.: Deficiency Edema, Gaceta méd., Mexico **11**: 67 (Jan.-June) 1917; abstr. J. A. M. A. **78**: 424 (Feb. 9) 1918.
10. Patterson, A. H.: Starvation Edema, Med. Rec., November, 1899, p. 715.
11. Holst, A.: Experimental Studies Relating to Ship Beriberi and Scurvy, J. Hyg. **7**: 621, 1907. Holst, A., and Frölich, T.: J. Hyg. **7**: 670, 1907.
12. Budzynski, B., and Chelchowski, J. M. H.: Hunger Swelling in Poland, J. Trop. M. **19**: 141 (June 15) 1916.
13. Rumper and Knack: Brit. M. J. **2**: 560 (Oct. 27) 1917.
14. Guillermin, R., and Guyot, F.: Undernourishment and Famine Edema, Rev. méd. de la Suisse Rom. **39**: 115 (March) 1919.
15. Maase, C., and Zondek, H.: Berl. klin. Wchnschr. No. 36, Sept. 3, 1917; abstr. Brit. M. J. **2**: 560 (Oct 27) 1917.
16. Schiff: München. med. Wchnschr. No. 22, 1917.

had been improperly fed for a long time, especially as regards proteins, and the liability to edema, always present in malnutrition, was aggravated by the large quantity of sodium chlorid in the food. He states that persons showing war dropsy had usually been getting from 1,200 to 1,400 calories a day, including only 30 to 50 gm. of protein. But as such degrees of edema do not ordinarily occur in simple starvation, he believes that another factor must be present in these cases, namely, the ingestion of large amounts of fluid and salt in the attempt to sustain life on the thin vegetable soups common in prison camps and in famine districts. Cold, hard work and infectious diseases increase the tendency to edema simply because they increase the deficiency in food calories.

The Swiss authors found that in some regions the conditions of underfeeding were extreme, resembling the great famines of history. The disease was also very frequently found in men who were subject to hard work on a diet of from 800 to 1,200 calories, consisting of 15 per cent. and more of indigestible cellulose, bread containing 97 per cent. potatoes, very little fat, and a ration of 50 gm. of albumin, daily, at the highest. Exposure to cold and hard physical labor were contributing factors in the development of this disease.

Maase and Zondek [15] suggest that the toxic products of protein metabolism may cause damage to the endothelial lining of the blood vessels and so lead to edema. The high residual nitrogen and ammonia values found by them in the urine, blood and edema fluids were considered to be evidence of this hypothesis. Franke and Gottesmann,[17] in their study of the functional efficiency of the kidney in seventeen cases of war edema, found delay in excretion of urea and sodium chlorid in ten patients, and of potassium iodid and lactose in seven. They therefore call war edema a nephritis without albuminuria.

Maliwa,[7] from investigations of four cases, correlates the stage of polyuria with an excess of sodium chlorid in the blood, and finds that after the polyuria has passed off the blood is deficient in the sodium chlorid content. The change in the osmotic relations of the tissue is the essential factor in the disease, the polyuria and edema, though prominent clinical features, being secondary in importance. To determine the cause of the edema, Knack and Neumann [18] sought to secure its return in convalescents by restricting the diet principally to turnips, and by giving large quantities of water internally. Neither measure separately ever produced the result, but with the restriction of the diet, plus water drinking, the edema rapidly returned in convalescent patients. Lange [19] discusses the causation of a group of cases observed by him in West Prussia, and concludes that an altered permeability of the blood vessels was present, owing to a qualitative alteration in the food, perhaps to the extreme deficiency of calcium. Hulse [20] reaches similar conclusions, except that the edema was more frequently found as a sequel to relapsing fever, and in men who had been previously exposed to extreme cold. Recent writers discuss the subject of vitamins and the rôle they play in deficiency diseases, especially those belonging to the beriberic type, but generally recognize that it is not a well-defined deficiency disease, and in the majority of cases is the result of an inadequacy of diet to supply the nutritional requirements of the body.

The characteristic symptoms found among the inhabitants of certain towns in Poland were edema, debility, muscular weakness, intestinal disorders, mental depression, dimness of vision, disappearance of sexual impulses, and alterations in the blood and urine. With the disappearance of the edema the wasting was evident, the patients sometimes being reduced to mere skin and bones. Maase and Zondek [15] state that there were no noteworthy premonitory symptoms, but suddenly marked edema developed, especially in the lower limbs, with anemia and frequent diarrhea. Among French prisoners, there was great emaciation, the loss of weight being frequently 40 per cent. of the initial weight, anemia, general edema, muscular weakness and nervous exhaustion: and apathy and depression were associated with a "facies pestica." Falta states that prostration, apathy and weakness were almost constant; a feeling of heaviness in the legs, and diminished reflexes were found, but no typical polyneuritic symptoms occurred. The nutritional value of the food was still further diminished by diarrhea and dysentery, which were frequently associated early in the development of the disease.

In cases among the civil population, the edema was generally located in the feet and legs, occasionally in the thighs and trunk. In more than one-half the cases there was some degree of facial swelling; in one-sixth, the hands were swollen, and in one-ninth ascites was present. The face and scrotum were often affected, and in a small number of cases, ascites and hydrothorax occurred. Its features were remarkably uniform: the edema resembled that of renal disease and in mild cases was confined to the lower limbs; but in severe cases it was universal and caused considerable limitation of movement, sometimes interfering with the opening of the eyes. The edematous tissue was soft and elastic; the skin and puncture fluids were pale. In some cases the edema came on gradually, but after severe physical exertion, more rapidly. Following the disappearance of the dropsy, relapses were prone to occur, especially if there was any return to hard work or unsuitable food. The edema sometimes led to bursting of the skin with serous exudation, or so stretched the skin that pink scars like striae gravidarum resulted from it. The swollen extremities felt cold, and were painful when touched. Beyermann [21] states that twelve cases among the insane suggested scurvy or purpura except for the remarkably slow pulse and the absence of changes in the gums. On the addition of fresh vegetables to the ordinary diet, conditions returned to normal.

The urine was usually pale like water, alkaline, and contained neither sugar nor albumin. The amount of urine passed varied greatly in different cases, but on the whole was increased, sometimes reaching 60 ounces and over when the swelling was disappearing. As soon as the patient was put to bed, a marked diuresis began, and during the stage of recovery the amount of urine passed daily was from 3 to 4 liters. High residual nitrogen and ammonium values were found in the urine, blood and body fluids. Falta found polyuria and frequency of micturition; the urine being clear, of low

17. Franke, M., and Gottesmann, A.: Wien. klin. Wchnschr. **30**: 1004, 1917.
18. Knack, A. V., and Neumann, J.: Outbreaks of Edema in Germany, Deutsch. med. Wchnschr., July 19, 1917, p. 901; abstr. Lancet **2**: 248 (Aug. 18) 1917.
19. Lange, F.: Deutsch. med. Wchnschr., July 12, 1917, p. 876; abstr. Lancet **2**: 248 (Aug. 18) 1917.
20. Hulse W.: München. med. Wchnschr., July 10, 1917, p. 921; abstr. Lancet **2**: 248 (Aug. 18) 1917.
21. Beyermann, W.: Edema Disease in the Netherlands, Nederlandsch Tijdschr. v. Geneesk. **1**: 2265 (June 28) 1919; abstr. J. A. M. A. **73**: 1172 (Oct. 11) 1919.

specific gravity, and free from albumin and formed elements, except a few hyaline casts. Tonin [22] comments on the odd fact that polyuria constantly accompanied the hunger edema in ex-prisoners of war seen at the hospital. As noted by others, this polyuria usually began when the patients were at rest in bed.

Jensen's [23] study of the blood showed from 1.5 to 4 million red corpuscles with a color index greater than one, usually with a leukopenia, in 60 per cent. of the cases there being less than 5,000, with a relative lymphocytosis (from 30 to 55 per cent.). The coagulation time was usually shortened, and the blood proteins were nearly always decreased, generally being from 4 to 6.4 per cent. (normal is from 6.5 to 8.5 per cent.), that is, there was a hydremia with hypo-albuminosis. The freezing point was normal, the residual nitrogen normal or low, uric acid normal, and sugar and calcium usually low, chlorin usually approaching the upper normal figures, although it was occasionally low. Chemical examination of the blood and urine (Knack and Neumann) revealed a diminution in lipoids and in the organic phosphorus content of the blood. The depletion of the tissues in nutritive reserve in war dropsy is shown by Falta's statement that when absolute fasting is studied in these cases there are only from 2 to 3 gm. of nitrogen eliminated per day, as against 10 to 12 gm. of nitrogen excretion during the fasting of normal persons.

There were no cardiac symptoms reported by Maase and Zondek, but other observers found a condition suggestive of a cardiac lesion with failing compensation. Falta states that the slow pulse, from 35 to 40 a minute, characteristic of war edema, is best marked in males. Schiff reports a somewhat higher pulse rate of from 42 to 56. The edema was frequently observed with cardiac symptoms and infections in children, but in adults without these complications.

Hemeralopia frequently preceded the development of the edema. In severe cases, corneal ulcer and xerosis of the conjunctivae were troublesome. Ophthalmologists describe these eye changes as the result of debility and poor nourishment. Nyctalopia, or night blindness, is common in the spring and fall as a symptom of debility. Night blindness seldom occurs as a functional disorder except in cases of general debility, starvation or scurvy. The development of xerophthalmia is now recognized as due to a specific deficiency in fat-soluble vitamins. Maynard [24] discussed twenty cases of increased intra-ocular tension found in the course of epidemic dropsy. There was dimness of vision, the cornea was a little steamy, and the pupils were small or moderately dilated. The tension of the eyeball was distinctly increased. Halos, generally rainbow-like, were complained of at one time or another during the attack of dropsy.

Vandervelde and Cantineau [25] made observations on 200 patients treated by them in the St. Pierre Hospital at Brussels. Most of these cases were among deported Flemish civilians. There was marked edema of the lower limbs, frequently associated with "grave phlegmons." The general condition was brought about by lack of food and by deplorable hygiene. There were weakness and profound anemia; and dyspnea resulted from the slightest effort. Those deported were recruited without any medical examination and were forced to do hard physical work. Minor symptoms and complications were common. Among these were: ringing in the ears and dry, painful skin with frequent secondary pyogenic infections; and in one or two instances dark pigmented patches were observed on the face, similar to the pigmentation in Addison's disease. (Noted by the Polish authors.)

In mild cases under the influence of a more generous dietary, recovery took place. The regulation treatment for the condition consisted in a better diet as far as possible and rest in bed until all swelling had disappeared. Knack and Neumann found that recovery always followed rest in bed on ordinary hospital diet and that the restriction of fluids was rarely necessary. Maase and Zondek, by giving three patients 100 gm. of fat daily for a week, were able to cure the disease completely without rest in bed or other remedial measures. The diet should be ample, especially in regard to protein. The lack of resistance to cold is striking, death following relatively slight chilling, so that warmth is an important part of the treatment. The prognosis is good if the patients are kept in bed on a proper diet, but severe cases frequently prove fatal. Postmortem findings are seldom reported. Chronic marasmus with atrophy of the viscera, especially the heart and spleen, fatty degeneration of the liver and kidneys, and in some instances, dysenteric ulcers were found. In three cases Budzynski and Chelchowski found a diminution in the amount of blood, and a reduction in the size of the liver.

REPORTS FROM INDIA, CHINA AND MEXICO

Leaving the recent reports of edema in Europe and turning to the literature of other countries, we find that in many lands similar epidemics of dropsy have resulted from famine. Until the appearance of "epidemic dropsy" in India following the famine in 1876-1877, "swellings" were regarded as a minor symptom, when arising in the course of famine diseases. During this famine the mortality was high, and in eight famine districts nine tenths of the total recorded deaths were caused by famine diseases—dysentery, dropsy, diarrhea and debility.[26] Government works and a system of rationing were established for men, women and children unable to earn the daily ration. To test the value of this ration, a system of weighing the people was undertaken. In these tests great caution was found necessary for, it was reported, many of the people who came into the camps appeared to be filling out and fattening, when in reality they were getting dropsical and in a fair way to die. In the nursery of the famine relief camp near Madras, many children were found to be in a dropsical condition, and most of the old people were in the same state. Old men and old women were bloated with dropsy, and others again, many of them in the prime of life, were mere skeletons, the bodies of full grown men weighing only from 58 to 85 pounds at necropsy.

To supply the vast population of southern India with the necessary amount of food for health was the "Hoover problem" of the famine relief agencies. Practically all the grain had to be imported, and transportation facilities were inadequate. It seemed necessary to keep the grain ration, principally rice, as low as possible. Animal foods were scarce. Dr. Cornish, adviser of the government of India on public health

22. Tonin, R.: Gazz. d. osp. **40**: 636, 1919.
23. Jensen: München. med. Wchnschr. **65**: 925, 1918.
24. Maynard, F. P.: Preliminary Note on Increased Intra-Ocular Tension Met with in Cases of Epidemic Dropsy, Indian. M. Gaz. **44**: 373, 1909.
25. Vandervelde, M., and Cantineau, M.: Edema Among the Deported, abstr. J. A. M. A. **73**: 1229 (Oct. 18) 1919.
26. Digby, W.: The Famine Campaign in Southern India, 1876-1877.

questions, pointed out that effects of insufficient nourishment might not be immediately apparent, and throughout the famine constantly emphasized the importance of the nitrogenous value of the ration, and advocated a ration consistent with age and work, sufficient to replace tissue waste. After this famine, reports began to appear in the *Indian Medical Gazette* of acute dropsy and acute anemic dropsy. In 1881, McLeod [27] termed the disease "epidemic dropsy." The "new disease" continued to be the subject of many reports and extensive bacteriologic investigations until 1909-1910. According to bacteriologic phraseology, it appeared endemically and epidemically, and much study was given to a specific organism, with no constant results.

Dr. Greig,[28] in his report on epidemic dropsy, states that there is evidence to show that epidemic dropsy is a nutritional disease which is brought about by a one-sided dietary, and that the two severe outbreaks of epidemic dropsy in Calcutta and Bengal, namely, from 1877 to 1879, and from 1907 to 1909, have been correlated with a sustained high price of food grains during this period, and the cessation of these epidemics has synchronized with the fall in prices of food grains. The study of the parasitic origin of disease has somewhat overshadowed the question of the relation of defects of dietary to the causation of disease in the tropics. In one locality, Greig found in 321 houses, with 4,637 inhabitants, 1,581 persons who were dropsical. The persons attacked consumed polished rice, and this was their staple diet. The amount of rice consumed daily varied from 2 to 16 ounces (from 1 to 8 chittaks). When the price of grains rose, the capacity for purchasing additional suitable articles of diet diminished and the diet became dangerously onesided.

The peculiar qualities of rice as a diet were pointed out by McCay [29] in his investigations of jail dietaries. Rice is the poorest of all cereals in protein, and when cooked it swells up and absorbs three and one-half times its weight in water. The percentage of starch in rice is high—up to 80 per cent. Rice is deficient in fat. Rice is a bulky diet, 26 ounces of dry rice when cooked measuring about 2,800 c.c. A large carbohydrate diet, by its mere presence in the intestinal canal, hinders the absorption of protein. On account of the fermentation processes that are quickly set up, there is increased peristalsis, and the food is hurried through the small intestine past the area most favorable for absorption. The amount of rice present in the diet influenced in a marked degree the quantity of urine excreted. The rice may have a diuretic action on the kidneys, or water may be formed in the tissues from the constituents of the rice, in addition to the large water intake with the boiled rice itself.

Dr. Patterson [10] of Chinkiang, China, described a group of cases of dropsy occurring in dispensary patients after a famine season. The food of these people consisted largely of weeds and wild plant greens. As no literature could be found on the subject, the disease was called "greens dropsy." The only symptom complained of was the swelling. With some medical treatment and money to buy grain, the patients recovered rapidly.

When the City of Mexico had been the seat of military operations for two or three months, Landa [9] related that many cases of edema in men, women and children began to be noted. Hundreds of cases were found with no albuminuria. As in other famine epidemics, many persons died of actual starvation, while others developed edema cachexia from defective nourishment. The mortality was high, the patients dying in marasmus with heart failure. There was hydremic anemia, hypothermia, slow pulse, reduction of the reflexes, and pain in the muscles. The only food obtainable had been vegetables of the families Chenopodiaceae and Amaranthaceae, such as beets and spinach.

RELATION OF WAR EDEMA TO DEFICIENCY DISEASES

Frequent reference is made to the similarity between the clinical symptoms found in war edema and those associated with diseases of the beriberi type. Falta states that the wet form of beriberi is the only other deficiency disease in any way resembling war edema. In this group of edematous diseases, as discussed by various authors, are tropical beriberi, ship beriberi and epidemic edema. The polyneuritic symptoms in tropical beriberi have been so constantly emphasized that they have obscured the equally important edematous conditions which form the chief feature in the wet type of the disease. In epidemic edema and ship beriberi, nervous phenomena are rarely present, but edemas of various degrees constitute the major symptom. Infants nursed by beriberic mothers suffer from edema, dyspnea and cyanosis. Authorities agree that this is an infantile beriberi due to some deficiency in the mother's milk. Almost all cases of infantile beriberi are edematous. The pathologic findings observed at necropsy in 219 infants under 1 year of age showed a percentage of 56.6 of infantile beriberi. Vedder and Williams [30] regard this edema in infantile beriberi as due to a specific avitaminosis. Vedder [31] furnished a list of food deficiencies found by the various investigators in beriberi: (1) deficiency in fat (Bremaud and Laurent); (2) nitrogen starvation (Takaki); (3) deficient vegetables combined with an infection (Fales); (4) deficiency in organic phosphorus (Schauman) and (5) deficiency of some unknown substance, not phosphorus (Fraser and Stanton, Chamberlain and Vedder, Shiga and Funk).

It is interesting to contrast with this group the findings by the various authors in war edema. The lack of calcium, fat, phosphorus in the blood, fresh vegetables, proteins and vitamins have each been emphasized in war edema. In addition there was general underfeeding; the diet as a whole was low in caloric value. The food was quantitatively as well as qualitatively deficient. There was semistarvation.

Lind,[32] in his early account of scurvies, found dropsy a constantly recurring symptom. Scorbutic persons were found to have edematous swellings at first about the ankles, later extending to the legs and other parts. The face, especially, became pale, swelled and bloated. Long want, improper diet, melancholy and cold are given among the causes. Dr. Cook, in a letter to Lind at this time, finds the term "nervous disorders" universally applied to most chronic and cachetic ailments. The lower people "who live continually on

27. McLeod, K.: Epidemic Dropsy in Calcutta, Indian M. Gaz. **16**: 148, 1881.
28. Greig, E. D. W.: The Scientific Memoirs of the Government of India, No. 49, 1911-1912.
29. McCay, D.: The Scientific Memoirs of the Government of India, 1909-1911.
30. Vedder, E. B., and Williams, B.: Concerning the Beriberi-Preventing Substances of Vitamines Contained in Rice Polishings, Philippine J. Sc., Sec. B, **8**: 175, 1913.
31. Vedder, E. B.: Beriberi, New York, William Wood & Co., 1913.
32. Lind, J.: A Treatise of the Scurvy, Edinburgh, 1753, p. 319.

farines and a gross diet,"and among whom these complaints are found, had a universal lassitude, pains which they termed rheumatic, and a breathlessness on exercise. The legs were sometimes swollen and the abdomen almost always tender and tumefied. Professor d'Espine observed these edemas during the siege of Paris as a first stage of scurvy; and Guillermin and Guyot, commenting on similar scorbutic complications, ask if scurvy may not be simply a state more advanced in the evolution of this disease, of which edema is an initial symptom. But the number of deaths occurring without scorbutic symptoms seems to plead for war edema as "une entité morbide." Dropsical patients without sore gums were frequently observed in epidemics of scurvy in Russia during the Crimean War when scurvy was very prevalent.

In pernicious anemia associated with pregnancy, Williams [33] finds anemia, weakness, shortness of breath, and edema of the extremities. A general puffiness affecting the hands and face as well as the legs, without urinary findings, is common in hydremic patients. More than half of all pregnant women, according to DeLee,[34] show some edema of the feet, the hands or the face. Often this is an elastic puffiness that does not pit. The cause of this is not known. In reproductive processes throughout nature, growth occurs at the expense of the maternal tissue. The protein materials are chiefly concerned in the growth of the new cells. Miescher [35] showed that salmon, after entering the Rhine from the sea, virtually starve. Yet the genital organs of both male and female develop greatly at the expense of the liquefying muscles, which may lose 55 per cent. of their weight (protein) without destruction of the muscle cell.

In war edema and in the etiologically related edemas in deficiency diseases, hydremic anemia is a somewhat frequent symptom. Osler and McCrae,[36] in their study of the circulatory disturbances in a group of cases of chlorosis, find dyspnea in 318, palpitation in 254, and edema in 231. "Doubtless it is the occurrence of slight degrees of edema which gives chlorotic patients so plump a look." All the symptoms come on in the course of from three to twelve months. The disease is most common in ill fed and overworked girls.[37] A long continued unbalanced diet may play a large part in the process.

Sir Joseph Fayrer [38] finds that pernicious anemia in Europe resembles beriberi in the Orient. Bramwell,[39] in a table showing the most important symptoms in forty-five cases of pernicious anemia, records twenty-three cases of dropsy, associated with great prostration, weakness and loss of weight. The urine was normal in the majority of cases. This edema was considered as partly due to the watery condition of the blood, and partly to the enfeebled state of the heart. Functional derangements of the stomach and intestine are almost invariably present. A symptom [40] which is practically never wanting is edema, especially of the legs and under eyelids, though it is also seen in other places on the body. The swelling is practically never marked, but is very persistent, and is noticeable as one of the earliest symptoms of the disease. Moreover, it readily recurs in patients who show a complete remission. As in other anemias, the edema is possibly due to alterations in the blood vessel walls. A gain in body weight in pernicious anemia when unattended with increase of hemoglobin indicates dilution of the blood and escape of serum into the tissues.

Edema occurring in the course of gastro-intestinal disorders and marasmic conditions in infancy is somewhat infrequent but well recognized by pediatricians. Chapin [41] reports twenty-one cases of general and local edema in which neither the condition of the blood nor that of the urine explains satisfactorily the development of the edema. The clinical conditions in which these edemas are most frequently found are: (1) difficult digestion and malassimilation with gastro-intestinal disturbances and diarrhea; (2) exhaustive conditions, such as prematurity, marasmus, extreme secondary anemias, edema neonatorum, and in long debilitating diseases; (3) occasionally in various constitutional diseases, such as syphilis, tuberculosis, erysipelas, and pertussis, and (4) in angioneurosis of vasomotor origin.

Under the term essential, primary or idiopathic edema, Wagner,[42] in 1887, records the earliest account of this disease. An epidemic of edema in which thirteen cases occurred in thirty-five babies in which gastro-enteritis was prevalent was thought by De Wolf [43] to be of infectious origin. The cases all occurred within a short time in a children's hospital in which the food supply was modified milk alone, or modified milk with the addition of a cereal or a proprietary food.

Potter,[44] in a group of cases of diarrhea with edema following a diet of barley water with a low percentage of fat and protein, increased the fats and proteins with the disappearance of the edema in a short time. The same author later reports a large group of cases in which he considers the edema a symptom of malnutrition and marasmus. In typical cases these babies had been treated for some time with boiled water, barley water or whey. A slight gain in weight occurred as the edema developed. Potter says that it is not what the babies are being fed that causes the dropsy, but what they are not being fed; also that it is entirely owing to the fact that they are not getting enough proteins in the diet, and this notwithstanding the intestinal disturbances that practically always accompany or precede the edema. It may be that in many of the cases the continuance of the diarrhea itself is due to the deprivations of solids in the food.

Czerny and Keller [45] use the term "Mehlnährschäden" to describe a condition found in infants fed on a high carbohydrate diet, but lacking in other important foodstuffs. The tendency of the tissues to hold water is increased in carbohydrate feeding. Holt [46] finds general edema as a symptom in marasmic infants. There is often increase in weight, and the whole body may become waterlogged. The symptoms shown by some

33. Williams, J. W.: Obstetrics, New York, D. Appleton & Co., 1912, p. 509.
34. DeLee, J. B.: The Principles and Practice of Obstetrics, Ed. 2, Philadelphia, W. B Saunders Company, 1915, p. 386.
35. Miescher, quoted by Lusk, Graham: The Science of Nutrition, Ed. 2, Philadelphia, W. B. Saunders Company, 1909.
36. Osler, William, and McCrae, Thomas: Modern Medicine, Ed. 2, Philadelphia, Lea & Febiger, 1915.
37. Osler, Willam: Principles and Practice of Medicine, Ed. 8, New York, D. Appleton & Co., 1916, p. 730.
38. Fayrer, Joseph: Beriberi, in Quain's Dictionary of Medicine, London, 1888, p. 104.
39. Bramwell, Byron: Anaemia, Philadelphia, William Wood & Co., 1899.
40. Stengel, Alfred: Diseases of the Blood, Philadelphia, W. B. Saunders, 1905, p. 263.
41. Chapin, H. D.: Cases of Edema in Infants, Arch. Pediat. **31**: 5, 1914.
42. Wagner, E.: Deutsch. Arch. f. klin. Med. **41**: 509, 1887
43. DeWolf, H.: A Report of Thirteen Cases of Edema Apparently Epidemic in Character, Arch. Pediat. **19**: 895, 1902.
44. Potter, P. A.: The Relation of Protein to Edema in Marantic Children, Med. News, New York, Jan. 9, 1904; Edema in Infants, Arch. Pediat. **29**: 206, 1912.
45. Czerny and Keller: Des Kindes Ernährung, 1906.
46. Holt, L. E.: Diseases of Infancy and Childhood, New York, 1916.

infants that have been fed for a long time on an almost exclusive carbohydrate diet indicate that they suffer from "Mehlnährschäden." The carbohydrate diet is frequently given in the form of proprietary foods and cereal decoctions to overcome diarrhea. Bloch [47] applies the term carbohydrate dystrophy to a group of cases in which he found xerophthalmia associated with edema resulting from fat deficiency and a carbohydrate diet. Hume [48] observed thirteen cases in which edema appeared following gastro-enteritis and vomiting. There was no marked error in the diet to throw light on the etiology of the condition. His observations on salt retention in these infants failed to be conclusive, and as there was no evidence of kidney or heart disease, the pathologic condition was sought for in the tissues themselves. The action of toxins, developed in the gastro-intestinal tract, on the suprarenals or capillary cells is suggested as a possible cause of the condition.

Ashby [49] finds these edemas following gastro-intestinal catarrh which has persisted for weeks. The gastro-intestinal tract is so deranged that poisons absorbed from it reach the systemic circulation and in this way lower the vitality of the endothelium of the blood vessels, causing an increased permeability. Recurrences were common, and these children seemed to do better on food containing a high percentage of proteins with a low percentage of carbohydrates.

In a review of the literature on osmosis and edema in infancy and childhood, Waterman,[50] as late as 1914, finds uncertainty as to the methods of the production of this edema. In the light of present knowledge, the weight of evidence seems to be in favor of the chlorid retention theory of infantile or essential edema, although the vascular lesions theory has many points in its favor. The etiologic factors considered by this author are: (1) latent or hidden nephritis; (2) chlorid retention which leads to a hydremia and so to an edema, and (3) increased permeability of the capillary walls.

In reviewing these various etiologic factors, there is evidence that the same type of dietetic and pathologic conditions is found in these edemas in infants as those concerned with war edema and the edemas found in the deficiency diseases of the beriberi type.

A general dropsy is a common symptom in hydremic animals. Friedberger and Fröhner,[51] and Hutyra and Marek [52] describe this condition as it occurs in draft oxen and horses that work in sugar factories and in other cattle from exclusive feeding on distiller's wash. The disease is chiefly caused by feeding on beet root residue, which contains only about 5 per cent. of solid matter with 95 per cent. of water. As the proportion of proteins in the solid matter is only 1 to 10, the residue contains 0.5 per cent. proteins. Consumption of such food combined with hard work results in hydremia. All tissues are infiltrated and the body cavities filled with transudate.

A similar condition of dropsy or "cachexia aquosa" is found in sheep from insufficient pasturage and unfavorable climatic conditions, such as wet or cold weather, badly situated grazing lands, and penning the sheep on wet, cold soil.[53] Weakness, emaciation, anemia, depression and exhaustive diarrheas accompany this condition.

EXPERIMENTAL EDEMA

Denton and Kohman [54] find that dropsy occurs in a large percentage of rats fed on a carrot diet, when the proportion of nitrogen is reduced by the addition of some non-nitrogenous foodstuff, such as fat or starch. Kohman,[55] in further experimental work, produced edema in a large percentage of rats fed on a diet composed largely of carrots. The addition of fats or fat-soluble vitamin, or water-soluble vitamin, or increase in salt content of the diet had no noticeable effect on the occurrence of edemas, but there was much more marked edema when there was much water in the diet than when the animals were on a dry diet. On the addition of a sufficient amount of an adequate protein to the carrot diet without change in caloric value, no edemas occurred and the animals grew normally. Control experiments showed that the edema was not due to toxic products in the carrots, or to starvation from low carloric intake.

Harden and Zilva [56] observed edema in one of three monkeys fed on a diet complete in every respect, except that it lacked the fat-soluble "A" factor and was low in fat. Each of these animals received a daily diet of from 250 to 300 gm. of boiled, polished rice, marmite, 10 gm., and salt mixture, 2 gm. (The large amount of rice in this diet may have hindered the absorption of the protein.) [29]

Extensive experimental work was conducted by Holst and Frölich [11] in an endeavor to produce ship beriberi in animals. Abortive cases of scurvy resembling ship beriberi were repeatedly seen in guinea-pigs, but although these authors were unable to produce typical ship beriberi they frequently observed edema.

I have carried out a number of dietetic experiments with dogs, rats and guinea-pigs. These animals have been variously fed on specially prepared breads containing much cornstarch in order to reduce the protein content; also, in the case of the rats and guinea-pigs, diets of beets, turnips, cabbage and potatoes with or without the addition of starch bread or plain bread. It has not been possible to carry out this work to the extent desired to make a complete study of the subject; furthermore, the work of Miss Kohman seems to cover the ground sufficiently well. Therefore no details of this work will be published. To summarize the results it may be said that in a number of animals edema was obtained, and that these cases occurred under such conditions as to agree fully with Miss Kohman's conclusions. That is to say, edema was not observed in animals that received a dry diet even when they were allowed to take such water as wanted. Most of the instances of distinct edema were observed in animals that lived on a diet poor in protein and fats and containing much fluid. For example, no edema was observed in guinea-pigs living on potato and rye bread, or on meal bread or rye bread alone; whereas a few of the guinea-pigs living solely on beets or cabbage showed more or less edema. A few rats fed

47. Bloch, C. E.: Xerophthalmia and Dystrophy in Infants, Ugesk. f. Læger **80**: 815 (May 23) 1918; abstr. J. A. M. A. **71**: 322 (July 27) 1918.
48. Hume, W. E.: General Edema Following Gastro-Enteritis in Children, Brit. M. J. **2**: 478 (Sept. 2) 1911.
49. Ashby, H. T.: Practitioner, London, May, 1914, p. 686.
50. Waterman, L.: Arch. Pediat. **31**: 135, 1914.
51. Friedberger, Franz, and Fröhner, Eugen: Veterinary Pathology, Ed. 6, Chicago, W. T. Keener Company **2**, 1908.
52. Hutyra, Francis, and Marek, Josef: Pathology and Therapeutics of the Diseases of Domestic Animals, Chicago, Alex. Eger **1**, 1916.
53. Hoare, E. W.: A System of Veterinary Medicine, Chicago, Alex. Eger, **2**: 1290, 1915.
54. Denton, M. C., and Kohman, Emma: Feeding Experiments with Raw and Boiled Carrots, J Biol. Chem. **36**: 249 (Nov.) 1918.
55. Kohman, Emma: The Experimental Producton of Edema as Related to Protein Deficiency, Am. J. Physiol., to be published.
56. Harden, A., and Zilva, S. S.: Edema Observed in a Monkey Fed on a Diet Free from Fat-Soluble "A," Accessory Food Factor and Low in Fat, Lancet **2**: 780 (Nov. 1), 1919.

solely on a carrot diet also showed edema. In one of these the visible edema disappeared when casein was added to the diet and returned when the animal was again restricted to carrots. This work adds nothing to Miss Kohman's observations, but furnishes merely a certain amount of additional corroboration.

GENERAL CONCLUSIONS

It will be seen that the final conclusions reached by those who have studied war dropsy are in extremely close accord. This condition seems not to be a typical "deficiency disease" in the sense of being the result of a deficiency in one or more specific unknown constituents (vitamins) in the diet. In a broader sense it is, however, a deficiency disease, and is the result of a protracted existence on a diet deficient in total calories, especially in protein. Undoubtedly, a high fluid intake, and possibly a high salt intake, are important accessory features. Hard work and exposure to cold are factors simply in that they increase the caloric deficiency of the food supplied.

It is gratifying to find that the experimental work agrees perfectly with the clinical evidence in establishing that a combination of low calories, low protein and excessive fluid intake will lead to a marked dropsy corresponding to war dropsy in all respects. The importance of specific vitamins seems to be excluded by these experiments.

Undoubtedly, dropsy occurring in many conditions associated with either defective food supply or absorption (as in some types of infantile dropsy) or in conditions of protracted anemia or cachexia is essentially the same as war dropsy. Hence the general term "nutritional edema" is to be recommended for this class of cases.

THE CAUSE OF ABSCESS OF THE LUNG AFTER TONSILLECTOMY

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Reports of abscess of the lung following tonsillectomy have been appearing regularly in the literature during the last six years. Such cases are occurring fairly frequently in the practice of every man who pays any special attention to chest disease, and it is within the last few years that their presence has been so particularly noticeable.

What is the cause of this complication? Why has it made its appearance only lately with such frequency?

The first report of a case that I find is by Bassim.[1] I have not, however, been able to verify the reference. Shortly afterward Scudder [2] referred to it. The most comprehensive paper is that by Manges,[3] published in 1916. Manges reports nine cases, in one of which the patient died. He discusses causation under the heads of (1) anesthesia; (2) aspiration of infected blood or of pieces of tonsillar tissue; (3) embolism, or infarction of the lung; (4) some special infective agent, and (5) some antecedent cause.

Perhaps only the last two heads need any explanation. Under the subject of some special infective agent he refers to three of his patients who were operated on in the same hospital, not far apart, and he thought that possibly the fact that they acquired their disease at the same place and the same time bespoke the exposure to an infection with special pulmonary affinity.

For an antecedent cause he warns against operating when the patient has a cough, etc.

Manges made some statements in 1916, based on his own experience, which he probably would not repeat now. He states, for instance, that "abscess of the lung should never occur if the patient has been properly treated." He goes on to say that abscess of the lung never occurs after tonsillectomy in private practice. This is, of course, not true. Richardson,[4] who published an article shortly after Manges, says that tonsillectomy is never a minor operation in an adult. He thinks these patients need more after-treatment than they get, and that every patient who is to have a tonsillectomy should have a thorough physical examination.

Coakley [5] discussed the matter with particular reference to Manges' paper. His remarks are somewhat critical of Manges. He says that all his patients are carefully examined before a tonsil operation, and asks Dr. Manges to state just what he would consider a complete examination, and specifically just how it would prevent any lung abscesses.

Manges thinks, or thought in 1916, that all these cases were due to careless treatment on the part of the operating surgeon. He is, however, very hazy as to what was done that was careless. He thinks the head should be lowered during the stage of anesthesia and that the patient should be carefully examined before the operation to see that he has no pulmonary infection.

No idea of etiology has been advanced which bears the test of close scrutiny. It is admittedly true that poor physical risks, that patients with fresh tonsillar infection, and patients with acute respiratory disease are not good subjects for tonsillectomy. But carefully examined patients, persons of all ages in the best of health other than their tonsillar disease, patients with no respiratory infection, and patients surrounded with every care, attention and operative safeguard all get abscesses occasionally when their tonsils are removed by a skilled operator. The subjoined case is cited as an example of one occurring when every care was exercised:

An unmarried woman, aged 36, had her tonsils removed, July 1, 1918, on account of frequent attacks of tonsillitis. Her general nutrition was poor, and it was thought that the removal of the tonsils would improve that condition. The operation was performed in a hospital under general anesthesia. The anesthetic was administered by Dr. H. C. Anderson, who has devoted special attention to anesthetics for twenty years, and has had his widest experience perhaps in nose and throat operations. Furthermore, he took particular pains with this patient as she was a valued friend and co-worker. The operator was Dr. J. M. Patterson, a skilful and careful nose and throat surgeon. The anesthetic was gas-ether with a suction tube, and the flow of ether was maintained by a small pump engine. Every precaution was taken against aspiration of blood or infective material. The suction tube was never out of the patient's mouth. The operation was not troublesome and there was no excessive bleeding, either during the operation or later.

The after-course was instructive. The history of lung abscess began while the patient was on the table. She began to cough immediately after the operation was completed, and continued after she was put to bed. Only after the administration of one-half grain of morphin in divided doses was it

1. Bassim: Thèse de Paris, 1913, No. 181.
2. Scudder, C. L.: Boston M. & S. J. **171**: 523 (Oct. 1) 1914.
3. Manges, M.: Am. J. Surg. **30**: 78 (March) 1916.
4. Richardson, C. W.: Laryngoscope **26**: 1001 (July) 1916.
5. Coakley: Laryngoscope **26**: 1008 (July) 1916.