to the marrow as the most probable primary seat of disease.

It has been suggested that there was a general hyperplasia of the bone-marrow cells, and that the terminal drain, perhaps of the operation and subsequent oozing, drew out the erythroblasts, platelets and polynuclears into the blood-stream but left the giant cells *in situ* because of some mechanical obstruction. It is undoubtedly true that rapid changes took place in the blood just before death and that a very large increase occurred in the number of circulating polynuclears and erythroblasts, but there is no evidence in the smears of marrow of increased formation of those cells, while there is to be seen a great excess of giant cells. In fact, the giant cells may be considered to have interfered with erythrocyte and perhaps with polynuclear leucocyte generation in some such way as the lymphocytes do in acute lymphatic leukemia. In other words, this case may be considered to be a "leukemia" of the megakaryocytes. It is to be said, however, that this is only one way of explaining a somewhat obscure and, in the absence of autopsy, an incomplete clinical and histological picture. Just where the case is to be placed in any system of classifying diseases of the bone marrow is a matter for discussion. If the term "aleukemic leukemia" is admissible, if "leukemia" is taken to mean merely a primary hyperplasia of one or more of the cellular elements of marrow without regard to leucocythemia, why is not "pernicious anemia" a "leukemia"? "That way madness lies." It is likely that in future a system of nomenclature to include all the myelogenous diseases may be adopted, with divisions allotted to erythroblastic, leucoblastic and platelet-forming cells.

In conclusion the writer wishes to acknowledge his indebtedness to Drs. Cutler, Vickery, Porter and Jones for permission to report the cases, and to Dr. J. H. Wright for kindly and enlightening aid in their study.

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ON THE TOXIN OF ECLAMPSIA.

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I. EARLIER RESEARCHES ON ECLAMPSIA.

PUERPERAL eclampsia has been for many years the subject of careful clinical and pathological study. The literature on eclampsia is very extensive and goes back to the early forties. But in spite of this fact very little real progress has

been made in determining the cause of the disease. It is to-day considered a decided pathological entity associated with lesions so characteristic that after death, at least, there can be no doubt about the diagnosis. As to the diagnosis during life, it is a very different matter; personally I have no doubt that many cases of uremia and epilepsy are diagnosticated as eclampsia, and when the patients recover there is no means of knowing that the diagnosis was not correct.

Based on the presence of albumin in the urine, the theory of Frerichs, who pronounced eclampsia a condition secondary to uremia, was for many years the accepted teaching. Frerichs attributed the symptoms to a poisoning of the blood through the retention of urea and the conversion of the latter into ammonium carbonate. We know to-day, however, that cases of eclampsia without albuminuria or signs of kidney disease occur not infrequently. Rosenstein attributed the convulsions to anemia of the brain; others (Cohnheim, Spiegelberg, Schroeder) sought the cause of eclampsia in a spasm of the arteries in the kidneys and brain, produced reflexly by the pregnant uterus. Halbertsma assumed that the convulsions were due to disease of the kidneys secondary to pressure upon the ureters. But many cases of eclampsia have come to notice where each and all of these conditions were absent.

Ludwig and Savor revived a modification of Frerichs' original uremia theory by their assumption that eclampsia was an auto-intoxication due to the retention of poisonous products of metabolism which the kidneys were functionally unable to eliminate from the system. These authors chose carbamic acid and ammonium carbamate as the *materia peccans* in place of the ammonium carbonate of Frerichs. Vollhard succeeded in completely disproving this theory. Numerous theories, some of which have sought to account for eclampsia as due to anomalies of structure and function of the blood vessels and nerve centers (von Herff, Kundrat, etc.), and others which sought the cause in a bacterial invasion or bacteriotoxic poisoning of the system (Albert, H. Mueller, Levinowitch, Stroganoff) have shared the fate of the earlier theories.

Of course, physico-chemical methods of investigation have also had their day. The freezing point of the blood and urine of normal and eclamptic pregnancies was studied by many authors (Koranyi, Kroenig, Fueth, Szili, Dienst, J. Veit, Schroeder, Caspar and Richter). They found that in three fourths of all cases of pregnancy the urine shows an insufficient molecular concentration and consequent insufficient diuresis, which reaches its lowest level at childbirth. Immediately after childbirth the molecular concentration of the urine begins to rise rapidly. The molecular concentration of eclamptic urines was found by most authors to be practically that found in normal cases; Schroeder alone found values in eclampsia which are far below those given as the lowest for the molecular concentration of the urine in normal pregnancies. It is a remarkable fact that with such decidedly insuffi-

cient diuresis the urine may be absolutely free from albumin.

Investigations on the toxicity of the blood and urine by the so-called Bouchard method have not yielded results of any value. In fact, Schuhmacher found that the urine of non-pregnant women was equally as toxic as that of normal pregnancy or eclampsia. This he found also to be true of the blood sera of normal and eclamptic women when they were injected intravenously into rabbits.

The bio-chemical method of investigation was used in cases of eclampsia by Halban and Langsteiner. Applying the methods of Ehrlich and his pupils they studied the action of parental and fetal blood on normal human blood, on the blood of rabbits and on bacteria (hemolysis, agglutination, bactericidal action), as also the influence of the two kinds of blood on fermentative (trypsin) and toxic processes (agglutination with abrin and ricin). Finally they attempted to determine the quantity of agglutinable and precipitable substances in the parental and fetal blood. As a result of their work Halban and Langsteiner, as also Veit, found slight but distinct bio-chemical differences between the mother's blood and that of the fetus; and Halban was able to show that a reciprocal agglutination and even hemolysis is possible between parental and fetal blood.

The careful studies of Stumpf and Winkel, and after them of Jürgens, Schmorl and Lubarsch, have thrown much light on the nature of the eclamptic lesions. Stumpf and Winkel were the first to call attention to the fact that the fetus usually shows kidney and liver lesions similar to those of the mother. Schmorl discovered the fact that eclamptic blood contained from five to eight times as much fibrin as the blood of normal pregnancy. He also laid great stress on the peculiar nature of the liver lesions as well as on the almost universal thrombosis present in cases of death from eclampsia. To quote from Williams: "In eclampsia, as was first pointed out by Jürgens, Schmorl and others, the lesions consist in hemorrhagic necroses occurring in the portal spaces, dependent upon thrombotic processes in the smaller portal branches. At first they are sharply limited to the portal spaces, but as they enlarge, they invade the lobules from the periphery toward the center; and are so characteristic that their mere presence justifies the diagnosis of eclampsia without any clinical history of the case."

J. H. Wright does not agree with Schmorl in attributing the focal liver necroses to thromboses. While he concedes the fact that the necrosis seems to proceed from the periphery of the lobule toward the center, he maintains that the lesions are not necessarily associated with thrombosis. To Wright the most striking feature of the eclamptic liver section is to be found in the character of the necrosis, which is always associated with the formation in large quantity of a peculiar reticulated fibrinoid substance. This substance, according to Wright, is never seen in such remarkable distribution and quantity in any other pathological condition of the liver.

The occurrence of an abnormally large quantity of fibrin in the eclamptic blood has led Dienst to base a method of diagnosing eclampsia on the determination of fibrin in the blood of pregnant women. If the fibrin test is positive Dienst immediately begins a treatment for eclampsia which consists in the introduction of the largest possible quantities of alkaline solutions into the system, together with hot-air baths, catharsis, etc. He claims in this manner to be able to prevent in practically every case the occurrence of convulsions and coma.

Schroeder, who has made a special study of the blood pressure in cases of eclampsia, comes to the conclusion that in many cases of this disease the blood pressure is practically normal and that on this account the determination of the blood pressure is of no value as a means of diagnosis.

Weichardt, a pupil of Schmorl, and Pilz have recently obtained most interesting results. They argued that since both the mother's tissues and those of the fetus showed the lesions of eclampsia, the toxic element was probably produced in and disseminated from the placenta. In order to eliminate from the placental tissues any antitoxin which the blood might contain, they removed the blood as completely as possible from the fresh placenta (perfusion with physiological salt solution at body temperature through the umbilical arteries) and extracted the finely divided organ with physiological salt solution. This extract they found to contain a most powerful toxin (syncytiotoxin) which was very virulent to rabbits. One to three cubic centimeters of a total extract of 30 ccm. (obtained from 50 gm. of crushed placenta) given intravenously to rabbits, caused death to occur very rapidly due to paralysis of respiration. The heart continued in each case to beat for some time after the cessation of respiration. By injecting very small, non-lethal doses of the active extract into the mesenteric vein of rabbits, Weichardt and Pilz succeeded in producing an immunity to the poison. Their active solution was, however, very thermolabile, since heating it at 60° on the water-bath for a short time was sufficient to render it absolutely innocuous. It is to be noted that Weichardt and Pilz obtained their active solution from normal placentas.

I began my work on eclampsia in 1896, when I was assistant in pharmacology at the University of Strassburg. In attempting a solution of the problem I approached the subject from the point of view of the pharmacologist, that is to say, my aim was to determine whether or not the blood of eclamptic women, or that of the eclamptic placenta, contained an active, non-proteid body, which might perhaps be isolated and recognized to belong to one of the established pharmacological groups of poisons, or to form a representative of a new group.

With this end in view I proceeded in a manner radically different from that of the serologists of the Ehrlich school. It was my object to obtain proteid-free solutions; this necessitated the application of heat with consequent inactivation or

precipitation of serum or tissue-juice constituents like the body of Weichardt and Pilz. To test the activity of my solutions I made use of intravenous injections into rabbits. In each instance but one I have given the chief clinical data of the case from which the material was derived. It will be seen from the histories that the clinical picture of the case gives no indication of the presence or absence of the toxin in the blood and tissues.

II. PERSONAL OBSERVATIONS.

A. — My first material was obtained from the Frauenklinik in Strassburg, and consisted of 250 ccm. of blood drawn by venesection from a patient who, according to Professor Fehling, had a typical case of eclampsia.

The blood was clotted when I received it; I was, therefore, obliged, after removing the serum, to triturate the clot with a little water and pure washed sand. The finely divided clot was transferred to a filter and thoroughly extracted, first with cold, then with hot distilled water. The filtrate was united with the serum, the whole barely acidulated with acetic acid and then heated in a large evaporating dish on the water-bath for several hours. When the red color of the fluid had changed to a light straw color the finely divided coagulum was collected on a filter and thoroughly washed with hot water. The clear filtrate and wash water were united and again heated on the water-bath. After a certain degree of concentration had been reached, a second finely divided coagulum was formed; this was again removed and the same process of fractional coagulation was continued until the resulting fluid amounted to about 10 ccm. of a reddish-brown liquid which did not yield the faintest biuret reaction, and which was therefore free from proteid.

Feeling that the fluid if active at all would be only slightly so, I slowly injected nearly the entire quantity into the large vein of a rabbit's leg. As I was completing the injection the rabbit had one violent convulsion and died. Only about 1 ccm. of the solution remained; this I injected into the ear vein of another rabbit. The second animal did not seem in the least disturbed by the injection; a few moments after it he ate food in a normal manner. This was at about 3 o'clock in the afternoon; at 6 P.M. I examined the animal and found it apparently normal. The next morning I learned from the attendant that at 9 P.M. he had found the animal dead, but still warm. The rabbit had probably died very shortly before 9 P.M., death having occurred in about six hours.

Post-mortem section of the two rabbits showed no macroscopic changes; microscopic sections of the organs were not made.

B. — On March 14, 1907, I received a placenta from the Boston Lying-in Hospital. The history of the case is as follows:

E. S., Russian Jewess, twenty-seven, Ipara. Brought to hospital in state of coma on the evening of March 13. Indefinite history of convulsions, headaches for past two to three days. Catheter specimen of urine showed slightest possible trace albumin, no sugar, no renal elements. Left hemiplegia, left pupil widely dilated. Reflexes normal, no Babinsky, no Kernig's. Temperature 101°, pulse 112.

Patient was delivered under ether with forceps at 12.55 A.M., March 14. After delivery patient continued comatose and died at 10 P.M. A post-mortem section, which was performed at the Massachusetts General Hospital by Dr. O. Richardson, showed:

Disseminated areas of hemorrhage in the brain. Thrombosis of right lateral sinus. An area of soften-

ing on the floor of the third ventricle. Acute degeneration of the kidneys. Acute degeneration of the liver with hemorrhages. Hemorrhagic edema of the lungs. The liver findings are not those typical of eclampsia. (J. H. Wright.)

The placenta was finely divided in a meat chopper and extracted with large quantities of distilled water until the strained extracts were practically colorless. The blood and the tissue extracts were united with the total wash waters, barely acidified with acetic acid and heated in a large porcelain dish on the water-bath. The proteids were removed by fractional coagulation and the solution concentrated as described above. The final reddish-brown fluid obtained showed a faint acid reaction and possessed a peculiar, not agreeable, odor.

EXPERIMENT 1. March 16. Rabbit (2 kg.) given, 3 P.M., 4 ccm. placental fluid into vein of ear. No immediate symptoms. 5 P.M., animal refuses food, crouches in somnolent condition in a corner of the cage and does not react to stimuli. Respiration 140. Pulse too rapid to count. Temperature 105°.

March 17. 8 A.M., rabbit found dead in cage, still warm; death had therefore occurred in about eleven hours. Sections of the liver show necrotic areas with diffuse hemorrhages.

EXPERIMENT 2. March 17, rabbit (1.5 kg.) given, 5 P.M., 1.5 ccm. subcutaneously. No action.

March 18. Animal well.

March 19. Animal well. 3 ccm. solution given subcutaneously. No action.

March 20. Animal well.

EXPERIMENT 3. March 20. Guinea pig given, 5 P.M., 1 ccm. solution subcutaneously. No immediate symptoms.

March 21. Animal refuses all food; crouches in corner of cage with fur bristling.

March 22. Animal normal.

March 23. Animal normal. 2 ccm. solution subcutaneously. No action.

March 24. Animal normal.

EXPERIMENT 4. March 21. Rabbit (2 kg.) received, 11 A.M., 1.5 ccm. dilute solution intravenously; 5 P.M., animal seems affected, refuses all food and does not react to stimuli.

March 22. Animal decidedly ill, refuses all food; labored, rapid respiration.

March 23. Animal still ill, but not so prostrated as day before; 1 ccm. fluid injected into vein of ear.

March 25. Animal very ill; somnolent; hind legs appear to be somewhat paralyzed.

March 26. Animal a little brighter; makes weak effort to eat. Intravenous injection of 3 ccm. fluid.

March 27. Animal shows decided dyspnea; lies in unnatural position with outstretched hind legs.

March 29. Animal died during night.

Post-mortem sections of liver show multiple necrosis, but no typical hemorrhages.

C. — On Sept. 20, 1907, I received from the Boston Lying-in Hospital a placenta with a clinical history as follows:

L. C., Italian, eighth pregnancy. No eclamptic history with any of earlier confinements. Nine months pregnant. No symptoms until night before. No headaches, no edema or eye symptoms. 10.30 P.M., Sept. 18, epigastric pain. 5.30 P.M., Sept. 19, first convulsion followed at intervals by nine others, the last 3 of which occurred in the hospital. Convulsions called "typical eclamptic." Very little edema of legs, none of face and hands. Unconscious; respirations good, not stertorous.

Urine: bloody, albumin 1%. Sediment: many hyaline and fine granular casts; much blood. Case delivered. Recovery.

The placenta was treated as described under *B* and a proteid-free fluid was obtained. With this the following tests were made:

EXPERIMENT 1. Sept. 22. Rabbit (1.5 kg.) receives, 11.30 A.M., 2 ccm. fluid intravenously. 2 P.M., animal appears stupid; is easily hypnotized to retain any position and is roused with difficulty only from the hypnotic sleep. Respiration rapid and labored. 5 P.M., hypnotic condition still more pronounced. Respirations labored, 184. Pulse not obtainable on account of rapidity. Temperature 104.5°.

Sept. 23. Animal found dead.

Sections of liver show necrotic areas with diffuse hemorrhages.

EXPERIMENT 2. Sept. 25. Rabbit (2 kg.) receives, 3 P.M., 1.5 ccm. concentrated solution intravenously. 3.30 P.M., animal appears normal. 4.30 P.M., animal decidedly affected; appears to fall asleep spontaneously; does not react to stimuli. Respirations very rapid. Pulse too rapid to count. Temperature 105.2°. 5.30 P.M., animal dead.

Sections of liver show considerably cloudy swelling, but no typical hemorrhages.

D. — On July 22 I received from the State Hospital at Tewksbury, a placenta with the following history:

Ipara, nineteen. For several weeks under treatment for albuminuria, which has somewhat improved. False pains July 17. July 20, 3 P.M., convulsions of five minutes' duration (number not given). 3.30 P.M., delivered under ether with forceps; no more convulsions. Urine at birth, clear, considerable in quantity, trace of albumin.

The placenta of this case was treated as described above (*B*). With the proteid-free extract experiments were made on rabbits. *The solution was found to be absolutely inactive.*

E. — On March 5, 1908, I received a placenta from the Boston Lying-in Hospital accompanied by a history as follows:

H. A. H., twenty-four, Ipara. Last menstrual period in June, 1907. March 4, 1908, patient was seized in the afternoon with vomiting and pain in the epigastrium. Patient was given castor oil with the desired result. Pain and vomiting ceased. Early in the morning of March 5, patient was seized again with pain in epigastrium, very severe this time, and vomiting, as also blindness. She had one convulsion. She was sent to the Lying-in Hospital, and delivered at 11.30 A.M. Recovery. *The proteid-free extract obtained from this placenta was also quite inactive.*

Several other placentas which I received and which were accompanied by a clinical history strongly suggestive of eclampsia yielded an inactive extract. Extracts of numerous normal placentas were invariably found inactive.

PREPARATION OF ACTIVE BODY IN PURE STATE.

The remainder of the fluid from case *C* was completely precipitated with a large excess of alcohol. The doughy, white precipitate was collected on a filter and thoroughly washed with alcohol. The alcoholic filtrate (*A*) was evaporated on the water-bath with constant additions of water until all the alcohol had been removed.

EXPERIMENT 1. The residue, amounting to a few cubic centimeters, was given intravenously to a rabbit. No action.

The precipitate produced by the alcohol was dissolved in about 50 ccm. of water and treated on the water-bath until all traces of alcohol had disappeared. The solution was tested as follows:

EXPERIMENT 2. Oct. 6, 1907. Rabbit (2 kg.) receives, 4 P.M., 1.5 ccm. of a dilute solution intrave-

nously. At first animal appears normal. 4.30 P.M., respiration labored and rapid; animal stupid and easily hypnotized in most unnatural positions. 4.45 P.M., respiration still more labored; animal very restless, keeps turning around in a circle. 4.50 P.M., animal apparently very ill, assumes position as if about to jump, and remains in this position for hours. Respiration 104. 5.00 P.M., animal drops to sleep spontaneously, awakening just as it is about to fall over.

Oct. 7. Animal fully recovered.

Having found the solution of the alcohol precipitate to be active, I attempted to reprecipitate the active substance from solution. The solution was fractionally precipitated with alcohol, and each fraction obtained tested for pharmacological action. None of the fractions obtained showed any action. The remaining solution, to which a large excess of alcohol had now been added, showed a marked turbidity, but yielded no further precipitate, not even on several days' standing. I therefore added a large amount of ether to the solution, thereby increasing the turbidity. No precipitate was formed. The solution, well covered, was set aside. In the course of a week, beautiful, colorless needles began to form. In order to insure complete separation I added more ether and allowed the solution to stand ten days longer. At the end of this time I collected the crystals on a hardened filter, washed them carefully with alcohol-ether, dissolved them in a little water and recrystallized them from solution by the addition of alcohol and ether. The quantity obtained was minimal and not sufficient for analysis. I dissolved the crystals, therefore, in very little distilled water and tested them for pharmacological action.

EXPERIMENT 3. Nov. 1, 1907. Rabbit (2 kg.) receives, 11 A.M., 1 ccm. solution of colorless crystals intravenously. Animal at first normal except for slight restlessness. 1 P.M., animal somnolent, purging and passing of urine. 1.30 P.M., somnolence increases; animal crouches in corner with eyes closed and does not react to loud noise; slight reaction to tactile stimuli; animal very easily hypnotized in every conceivable position, and difficult to rouse from hypnosis. Respirations 136, labored. Marked tremor from time to time; pronounced trismus. The animal's snout appears peculiarly pointed on account of muscular contraction. 3.30 P.M., stupor constantly deepening. When placed flat on its back the animal remains for hours in this, for it, most uncomfortable position. Concussion of the substratum produces violent trembling, but does not rouse the animal from the hypnosis. Frequently repeated concussions cause the animal slowly to rouse from his stupor. Respiration, 192. Temperature, 105°. Veins of ears strongly injected.

Nov. 2. Animal apparently recovered.

Nov. 10. Animal found dead.

The foregoing experimental investigation would seem to show that there are certain cases of acute intoxication, due to a definite non-proteid active principle, which during life present the clinical picture of eclampsia. Much more experimental work will be necessary to enable us clinically to distinguish the cases in which the toxin is present, from cases of "eclampsia" due to other causes, such as epilepsy and uremia. The solutions I obtained do not as a rule produce the convulsions typical of eclampsia. Possibly the toxic substance undergoes certain modifications of structure or composition during its isolation which affect the quality of its action. Of recent years numerous cases of "eclampsia without convulsions" have been described; perhaps

the toxin acting in these cases is identical with the substance obtained by me. In order to produce its action the toxin and solutions containing it must be given intravenously. This is true of many other poisons. Possibly the toxin is rendered inactive by combination with a tissue-component when it is given subcutaneously.

As to the pharmacological character of the toxin little can at present be said. It seems to act as a protoplasmic poison. It paralyzes the centers of the brain and cord, especially the respiratory center.

I have at present material in hand which may make it possible for me to obtain a sufficient amount of the toxin for analysis and chemical characterization.

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THE TENT WARD AT THE MASSACHUSETTS GENERAL HOSPITAL.

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At the present time, when hospital construction is thought of as being accompanied by great expense, it may be of interest to know how much can be accomplished with a comparatively small outlay.

During the spring of 1907, two wards at the Massachusetts General Hospital were to be renovated at once. One ward was moved to the "Relief Ward," the other to the "Tent Ward." This ward was open from June 13 to Nov. 1.

The Tent Ward was equipped as follows: there were two tents, $14\frac{1}{2} \times 19\frac{1}{2}$ feet in dimensions, each of which could accommodate three patients easily, and four if necessary; one tent, $14\frac{1}{2} \times 17$ feet, used as a kitchen and fitted up with ice-box, electric-toaster, water-cooler, sink and tables; eight tents, 12×17 feet, six of which were used for patients, two being accommodated in each tent, so that a total of eighteen patients could be accommodated; one of the two remaining tents was fitted up with medicine closets, chests for linen and blankets, and a table and chairs for nurses' use; and in the other there were a bath-tub, hop-

per, toilet, sink, and rack for solutions. The tent last mentioned and the kitchen tent had hot and cold water and sewer connections. All tents were provided with a fly and the walls could be rolled up for better ventilation. Ventilation was also aided by slits in the roof of tents, protected by the fly. The lighting was by electricity, and in each tent there was a Hy-lo electric lamp so attached as to be portable to all parts of the tent. The tents were arranged in a long row with the medicine and solution tents in the center of the row; the kitchen tent was opposite and at a short distance from the center of the row. In front of one of the large tents a fly was spread of the same size as the tent and used as an office; it was equipped with desk, chairs, telephone and couch. All tents had a flooring of $\frac{7}{8}$ in. spruce, laid on 2×3 joists, and there were walks of the same material connecting them. The beds were "dormitory beds" with low head, with "National wire springs" and one mattress. There were a table and chair for each bed. In the latter part of the season each bed was supplied with two mattresses, extra blankets, and when necessary hot-water bottles and soapstones were used, and even when the weather became quite cold, a tent could be warmed in fifteen minutes by means of an oil heater, so that baths could be given. Hot air baths were given with good results until the weather became quite cold. The total actual outlay for the tents, which were second hand, for lumber, labor and lighting was \$525. Everything else came from the general supplies of the hospital. All food came from the hospital diet kitchen.

Three nurses and one head nurse were assigned to the ward. The work was not more difficult than the regular ward work, the increased distances and the difficulty of watching all the patients being compensated for by the greatly diminished housekeeping necessary. There were two night nurses, for the double purpose of watching the patients sufficiently and to avoid loneliness. Canvas hats were worn by the nurses as a protection against sun and rain, and rubber coats and boots in stormy weather. The nurses, without exception, enjoyed the work, none was in any way harmed by exposure, and the general health of all seemed improved, in some cases quite markedly.

Ninety patients were admitted to the tent ward. There was no selection of cases until the last two weeks when, because of the inclement weather, it seemed wise to admit to the ward only patients whose condition called for out-of-door treatment. The mortality was seven, none in any way due to the open air treatment; two were cases of pernicious anemia who died shortly after admission and both of whom had been admitted to the hospital several times previously; two were suffering from chronic interstitial nephritis with uremia; one died on the day of admission, the other responded to treatment well at first, but died at the end of a week. One fatal case was a patient with cardiac disease who died shortly after admission; another cardiac case with