6. The Research

THE GHETTO DOCTORS WERE DRIVEN BY THEIR SCIENTIFIC zeal to utilize the great wealth of material available to them because at no other time in modern history had there existed such localized mass starvation in the presence of comparatively good medical facilities. True, studies on starvation had been done in World War I, but they had been limited in scope and many questions had been left unanswered: the riddle of the swellings found in hunger, the nature of the bony deformities, the problem of hemorrhages, the role of vitamins and the endocrine glands. The doctors set as their goal the answering of these questions.

In order to establish a norm in which non-essential deviations would be held to a minimum, infants, pubescent children, and the aged were excluded as subjects for the study, as were all cases of starvation complicated by tuberculosis and other diseases. Only "pure" starvation cases were examined. If the terminal autopsy showed that the "pure" cases were really "impure" because of superimposed infections, the findings were discarded.[1] No similar study had ever gone to such lengths to make a base line for its determinations.

CLINICAL FEATURES OF STARVATION IN ADULTS

Nothing was too obscure or too trivial; nothing was overlooked in the investigators' searching descriptions of the course of starvation from the beginning to the final moment of death.

The first symptom of starvation was a dry mouth, accompanied by increased urination; it was not unusual to have patients with a daily excretion of more than four litres of urine. Then came a rapid loss of fat and a desire to chew constantly, even on inedible objects. These symptoms subsided as starvation progressed; even weight loss slowed down. The next group of symptoms were somatopsychic: Patients complained of general weakness, of being unable to carry out the simplest tasks; they became sluggish, lay down frequently, slept fitfully, and wanted to be covered up to combat the abnormal feeling of coldness. They lay in the characteristic fetal position, legs drawn up and shoulders hunched over, so that contractures of the flexor muscles often developed. They became apathetic and depressed. Even the sensation of hunger became dulled; yet when any type of food was seen, many grabbed at it and wolfed it down without chewing.[2]

Weight was from 20 to 50 percent lower than the prewar weight; it varied between 65 and 90 pounds. The lowest weight observed was in a 30-year-old woman—53 pounds.[3]

Bowel movements increased, often leading to a bloody dysentery, causing further weakness. Swelling appeared first in the face, then in the feet and legs; it later spread over the entire body; fluid often accumulated in the chest and abdominal cavities.

Very few patients complained of bone or joint pains. As a matter of fact, very few complained of anything other

than weakness. An even smaller number said they "felt nervous," a surprising finding in view of the surroundings. Gross mental breakdowns, the classic psychoses, were rare.

The skin was pale and curiously pigmented over areas traumatized by the scratching that went with a variety of skin afflictions (scabies, pustulation, boils and louse bites). A very thorough study was made by Dr. Raszkes of the gross and microscopic features of the skin in starvation; he was put to death at Treblinka and his detailed papers were lost. Some were recovered, however; they demonstrated a definite tendency to pigment accumulation under the influence of minor injury. Even early and even in the presence of swelling, the skin was dry and scaly. It became thinned out, lost its elasticity and came to look like cigarette paper and feel like parchment, so that mere youths took on the appearance of old men. The sebaceous glands atrophied; acne and dandruff disappeared. In the summer, sunburn to the point of blistering occurred rapidly; in cold weather, chilblains were common. In the final stages of starvation, the face became dirty-gray in color.

The nails became opaque, striated, often clawed. Infections of the nail-beds came on easily.

Body temperatures were uniformly lowered, the mean being 96.8° F. Sometimes it dropped to 95.4° F. In infectious diseases such as typhus, where fever is usually very high, there was only a slight rise. In tuberculosis the rise was insignificant, thus often presenting a diagnostic problem.

Muscular weakness was so pronounced that it resulted in slowness of movement even under conditions of stress. To give one instance, a patient snatched a piece of bread from a doctor and tried to run away with it, but he fell to the ground, crying out, "My legs won't carry me!"[4] Creatine,

which appears in the urine as a rule only after great muscular effort, destruction of muscular tissue, or some endocrine disorders, was found in the urine of 17 out of 20 starving patients.

Eye examinations, done with great difficulty, gave surprising results. The patients, according to Dr. Fajgenblat, objected to the investigations and manipulations; most of the studies, those on visual acuity and fields of vision, are subjective and require good will as well as strict attention from the patients. Nevertheless, the visual apparatus of 20 female patients, none older than 30, was examined in great detail. Unfortunately, all the exact figures and the field of vision graphs were lost during the July 1942 expulsions. Enough data remained, however, to demonstrate that what was expected to be found was not there; *i.e.*, the signs of avitaminosis-A: keratomalacia, night-blindness, and Bitot's spots. No visual malfunction was found nor complained of, allowance being made for the presence of cataracts. The most striking finding was a bluish discoloration of the sclera, such as is usually found only in rare diseases associated with fragility of the bones, low blood calcium levels, and high excretion of calcium in the urine and feces. All these occur during starvation, other studies showed; they indicate a parathyroid dysfunction, avitaminosis-D being finally ruled out by still other studies. The pupillary reactions were sluggish, interpretable as a slowing up of the neuromuscular reaction or as weakness of the iris muscles. Almost without exception, the patients showed lens opacities resembling those in early senile cataracts, with the opacity increasing proportionately to the age of the patient. The intra-ocular pressure in all the patients was lowered, even down to 12 mm. Hg. (normal is 20-28 mm.). The starving patient has low blood pressure; if the intra-ocular pressure remained normal, there would be

great resistance to blood entering through the retinal artery, thus causing retinal disturbances; Dr. Fajgenblat felt that the lowered intra-ocular pressure was a defense mechanism against such changes, although he could not rule out hypo-adrenalism. The visual fields for light and the basic colors showed no departure from the normal. "Further investigations on adaptation to dim light could not be pursued because of technical difficulties."[5]

The auditory apparatus showed no changes. Hearing and equilibrium were undisturbed.

The tongue was often coated; the base was smooth, with atrophic papillae. Patients often complained of burning of the tongue. Tonsillar tissue was atrophied.

The teeth quickly became carious. Young people (20 to 25 years old) often had such advanced dental caries that they could barely chew their food.

As starvation progressed, the voice became hoarse. The cause was undetermined; it may have been organic, relating to weakness of the laryngeal muscles, or functional, due to the drying out of the mucous membranes of the larynx. In adolescent young men, the voice did not deepen.

Particular attention was paid to the lungs. It was found that hypotonia was constant; this finding was confirmed by X-ray and fluoroscopy in a few cases and by autopsy in most. Vital capacity was lowered greatly (to 1.5 to 3 litres) in studies made on 20 cases. The frequency of respiration was lowered to 11 or 12 a minute (normal is 18 to 20), and thus the volume of the respired air greatly diminished. The impaired elasticity of the lungs contributed greatly to the decrease in air exchange. Bronchitis and bronchopneumonia were common, as was tuberculosis (see below). Often, however, what was diagnosed clinically as pneumonia turned out to be, on autopsy, pneumonomalacia ending up in cavernous destruction of pulmonary tissue. Bronchial asthma was rare, as were all diseases of allergic origin.

The heart sounds were faint, with an occasional functional systolic murmur. The heart beat slowly and regularly, the mean being 40 to 50 beats per minute, the extremes 36 and 80. The arteries felt barely filled; pulse taking was consequently difficult. The peripheral veins were almost invisible. The blood pressure was greatly lowered: the systolic ranged from 60 to 100 mm. Hg. and the diastolic from 40 to 60 mm. Hg.

Since every part of the body is affected by starvation, it was not surprising that endocrine malfunction appeared. The changes in children will be described in the next section. Menstruation ceased in adult women; men became impotent. These obvious disturbances of the genital function were related also to the hair distribution, depending on the age of the patient. Starvation coincident with puberty caused increased hairiness of the head; hair rarely appeared around the sexual organs, and when it did, in females it took the form of an upright triangle (the normal male distribution) and in males the form of an inverted triangle (the normal female distribution). Long lashes and thick eyebrows occurred in both sexes. In girls and women a fine down appeared on the face, to the point of actual mustaches and sideburns; this down sometimes came out even on the eyelids. Young men were relatively beardless. In patients 20 to 50 years of age the hair was lost rapidly from the head, the axillae, and the genital areas. There was no evidence of the castration syndrome, however, in the eunuchoid form of abnormal growth of the lower extremities nor as abnormal deposition of fat.

Hypothyroid features (dry skin, low metabolism, etc.) were the norm in starvation. The parathyroid functioned

on a greatly lowered level, shown by tests on neuromuscular activity and trophic changes in the teeth, skin and nails. Likewise, other examinations showed hypofunctioning of the adrenals and pituitary gland. Studies on sugar metabolism (see page 65) showed, however, normal activity of the endocrine portion of the pancreas. All in all, the doctors concluded that the energy-sparing hormones predominated over the energy-producing hormones.

Prolonged starvation changed youths into prematurely old men. "My strength fades away like a candle flickering out," one of them said. The apathy became more pronounced, sleep more prolonged, arousal less possible. The passage between life and death was very gradual, sometimes imperceptible. Death resembled the fading out of an old, old man. Pulse and respiration slowed up more and more. Finally all vital functions ceased. Death came silently.[6]

STARVATION IN CHILDREN

Apathy was the first symptom of starvation in children. They lost interest, stopped playing, became sluggish, cranky and whiny. Then they became very quiet; their intellectual development seemed to stop, even to retrogress. In more advanced stages they lay on their sides, hunched up, with bent knees, with blankets over their heads because they felt chilly, even in summer. They lay quietly, almost motionless, but they did not sleep. Most suffered from insomnia. In the very advanced stage they no longer were able to sit, much less walk.

Weight averaged about 50 per cent of the normal, taking into consideration the arrest of growth as well as the height. Although growth arrest was not so obvious as weight loss, it occurred in almost every child. Children

from 2 to 5 and from 7 to 9 showed the greatest growth deficit; 3 and 4-year-olds were often no taller than infants and 9-year-olds no bigger than the usual 6-year-old. Among the older children starved during puberty the growth differential was not so great; they even appeared taller than they actually were because of their emaciation.

Body temperature was subnormal, rising only slightly in infectious diseases, in contrast to the usual high fevers seen in normal children with the same diseases. In tuberculosis, fever seldom appeared; very severe cases were observed often, with extensive destructive lesions, in which the temperature throughout was often normal or subnormal.

Skin changes varied. Sometimes the skin was pale and pasty looking; sometimes, cold and cyanotic. Most of the children had brown spots, usually at the site of old scars, or on the back, neck, abdomen, or in the skin creases or where clothing rubbed on the body. Sometimes there was pigmentation of the entire body. Pigmentary changes occurred mostly in those over five and those without edema. The skin became dry and scaly and lost its elasticity. Hairy growth was increased on the neck and on the face, appearing on the latter as sideburns.

Edema varied in the frequency of its appearance and its localization depending on the age of the children, being rare in those up to 2 years of age. Those from 2 to 5 showed the greatest tendency to edema; in older children dry cachexia was commoner. Swelling appeared mainly in the face and neck, becoming more quickly generalized the younger the child; it was always symmetric, its location varying with the position of the body, being greater in the dependent areas. Severe edemas led to hydrothorax, ascites, hydropericardium and swelling of the scrotum and labia.

The peripheral lymph nodes were felt as hard, painless, movable bean-sized masses; the atrophy of the subcutaneous tissues made them even more evident.

Parotid and submaxillary gland enlargement was a very characteristic sign often found in children with advanced cachexia; the parotids, especially, were greatly enlarged, although the ducts were unchanged. In all these cases the sublingual glands were not enlarged. Salivary secretion being very poor, it was felt that the enlargement was due to edema of the salivary glands but that the atrophy of the surrounding fatty tissue made them appear even larger.

The muscles were so weak and atrophied that the skin seemed to adhere directly to the skeletal structures. Children without swelling looked like skin-covered skeletons. The epiphyses of the long bones looked swollen, as in rickets; however, exact bony measurements and X-ray studies showed that these were apparent changes which had nothing in common with rickets. Muscle contractures were found in cases of extreme debility and included the flexor muscles of the thighs and legs and, to a lesser degree, the flexors of the forearms. Contractures were caused by the constantly bent extremities, the extreme passivity, and the muscular atrophy and the biochemical changes attributed to that atrophy. Sometimes there was limited motion at the hip, knee, and elbow joints without actual contracture. The contractures disappeared very slowly with a fuller diet; they occasionally lasted a long time after the child had seemingly recovered, thus making walking impossible. Some convalescents waddled in their gait despite a normal angle between the neck and the body of the femur, as shown by X-ray; the peculiar gait was probably due to over-relaxation of the muscles and ligaments.

The most interesting observation was the absence of

rickets. The only cases seen, and they were rare, were in 1942 in infants and 2-year-olds from a new group of deportees. During the entire period no case of late rickets was found. The X-ray pictures of the long bones were normal. In a few cases there was mild general decalcification, but the normal contours and growth lines were preserved. These observations contradict those made during World War I when rickets was very frequent and often appeared as rhachitis tarda, the late form, in older children and youths. The Ghetto infants all got prophylactic vitamin D, which was not used during World War I, but the doctors felt that such prophylaxis was not the only factor in the non-appearance of rickets. Scurvy, a sign of avitaminosis-C, was also absent, and there was no substitute for the vitamin given.

Dentition was faulty. The tonsils were small. The tongue was smooth.

The respiratory system showed pulmonary emphysema, especially in advanced cachexia, with a lessening of respiratory mobility and weak respirations. Fluid accumulations in the chest cavity were common, usually unilateral because the children lay constantly on one side.

The slow pulse, unusual in children, was typical of starvation, being as low as 55 to 60 per minute. It was often imperceptible, and faded out a long time before death actually occurred. Vascular collapse was indicated by the cyanotic coldness of the extremities, the barely visible superficial veins, and the tendency to thrombosis. It was not unusual to find faint heart sounds and irregular contractions. The highest blood pressure recorded was 60-65 mm. (systolic), much lower than the norm.

The blood usually showed only a mild anemia and a leukopenia, with a relative lymphocytosis. Noteworthy was the absence of eosinophiles even when intestinal parasites

were present. And equally noteworthy was the absence of the hemorrhagic diathesis, which would ordinarily be expected with a diet so low in proteins and vitamins.

Diarrhea was one of the most common symptoms, coming on very early in the course of starvation. In some cases it lasted throughout the entire period of observation; such children usually died. The stools were frequent, semiliquid, and curdy; sometimes they resembled those of a bloody colitis.

The gastric contents were analyzed in 15 children, fasting and after the giving of an alcohol-test-meal. Hypoacidity was found in every case.

No urinary disturbances were found, other than diuresis. No casts, hyaline or other types, were found in the urine. Concentration and dilution tests were done in 25 cases. The amount of water eliminated exceeded the amount taken in, a finding normal for well children, but the elimination of water was generally slowed up; the children passed urine as long as two hours after taking the water, showing the slow passage of water through the body tissues.

No neurologic changes were found, sensory or motor, not even the expected peripheral neuritis.

In pubescent children sexual maturation was markedly impaired, evidenced by the absence of pubic hair, menstruation, and breast development in all but a tiny minority of cases.

Tuberculosis took a virulent form in the starving children. It attacked not only the lungs, but also the peritoneum and the intestinal tract, even the uterus in two girls (proved by autopsy). Its course was violent and acute, demonstrating the complete lack of reaction of the body.

The situation was totally different in children with acute infectious diseases. Despite the great crowding in the hospital, epidemics were rare and their intensity more and

more benign. At the beginning of 1940 an epidemic of chickenpox lasted about six months; almost all the children caught it, but in 1941, when a case of chickenpox appeared, no epidemic broke out and only two children got the disease out of 250 to 280. Only older children, in a wretched state of nutrition, were in the hospital at this time, but in other groups of children, relatively better nourished, chickenpox spread rapidly. The same thing was seen in measles, confirming the clinical impression previously held that measles attacks the well-nourished first of all. Measles occurred rarely and in non-epidemic form amongst the most poorly nourished groups; an extensive epidemic appeared in rather well-fed deportees from Germany. With the exception of one epidemic early in 1940, meningitis was also uncommon. The body reacted weakly to the contagious diseases, with insignificant eruptions and low fever. Thus, despite the barely marked symptoms, such diseases in these very enfeebled children ended fatally.

The weak body reaction was shown in another field. Allergic phenomena (serum reactions, bronchial asthma, rheumatic fever) were rarely found. Before the war, the end of the winter and the beginning of spring were marked by a very high percentage of cases of rheumatic fever in the Children's Hospital. In the first three years of the Nazi occupation there were only five cases, two of them recurrences of earlier attacks.

The course and the prognosis in starvation disease depended on the duration of starvation, the type of diet, the individual constitution, and the age of the child. The younger the child, the more poorly he withstood starvation; the greatest mortality was in infants, reaching close to 100 per cent. Edema was a bad prognostic sign, indicating almost certain death in most of the children under 5 or 6 years of age.[7]

PHYSIOLOGIC REACTIONS AND EXPERIMENTS

The foregoing clinical descriptions led to the conclusion that in starvation energy saving and parsimony was concurrent with the insufficiency of energy. The sparing of energy, however, is limited by the physiochemical laws by which life is governed. The basal metabolic level cannot be indefinitely depressed; an end-point is eventually reached. A healthy active person uses up about 3000 calories per day; the clinical material in the Warsaw studies was made up of individuals who lived on 600 or 800 calories a day, often much less. The caloric deficiency could only be made up by endogenous feedings; that is, by eating up their own tissues. It can be readily calculated that these patients should have lived only 60 to 75 days, and yet they survived for months and years longer.

What prolonged their lives? Only an energy economy different from that in the normal human being, an economy that resembled somewhat that in animals during a state of hibernation. The laboratory studies and experiments attempted to determine what new mechanism came into play to bring about such a dramatic sparing of energy. Repeated observations were made, checked and rechecked, and the findings summarized in charts and tables on a scale never before done in a study of this nature.

The most extensive investigations were done on the circulatory system.[8] Its function, to carry oxygen and nutritive elements to the tissues and remove the waste products of metabolism, was thoroughly studied. It was found that the arterial blood pressure was very low; the venous pressure was equally low. The velocity of the blood in the circulation was half of the norm. The capillary circulation was slowed. The minute-volume of the heart was

1.5 liters in contrast to the normal 8-10. There was an asthenia of the entire circulatory system, a peculiar weakness that showed itself in a failure to respond to stimulation, thus saving energy vitally needed to sustain life. For example, contrary to what takes place in a state of health or when the body is attacked by other diseases, there was no compensatory change in the circulation after change of body position, nor after physical effort, nor after drugs (adrenalin, caffeine, atropine), not even after increased protein intake (although two eggs more than the usual starvation diet could not exactly be considered a great increase). No functional cardiac neuroses were discovered, despite the environment of terror; pre-existing paroxysmal tachycardia disappeared.

Electrocardiograms showed a very slow regular rhythm, low voltage in all leads, no changes in the ventricular complex, flattening of the T wave in all leads, increased conduction time in the S-T segment, and no change after exercise. The last finding indicated the heart's rigidity in adapting, which, even at rest, was functionally insufficient and hypo-energetic.

And yet cardiac failure, in the usual sense, did not occur. Why? The Warsaw investigators, after a long and complex series of laboratory studies on capillary circulation, arterial and venous pressures and blood volume, concluded that the lessening of tissue combustion in the body lessened the work of the already malfunctioning heart. A balance was struck. The heart conserved its energy to make up for its inadequate nutrition. The blood still circulated, sluggishly, at a lower speed than previously, and yet sufficiently fast to prevent tissue death.

The blood itself was the subject of a number of studies, morphologic and functional, including examination of the bone marrow in a number of cases.[9] Here, too, the rigidity

of response was noted: there was no increase in white cells such as usually occurs after a protein meal or in infectious processes nor was there any change in the sedimentation rate in superimposed acute or chronic infections. Anemia of varying degree was constant, with a reduction in the number of white cells, also. There was no evidence that the anemia was caused by increased blood destruction or by the dilution of the blood. It was found that the bone marrow was rich in cellular elements but that migration to circulating blood was impaired. Hemorrhages did not appear despite a low thrombocyte count.

Attempts were made to correct the anemia by various means: administration of iron, transfusions, liver injections, feeding of yeast, and increase in diet. The last alone had effect; more food was not available for the patients; the anemia persisted and became progressively worse. As the cachexia of starvation deepened, the bone marrow began to fail and eventually a state indistinguishable from aplastic anemia developed.

Laboratory studies were made on stomach functions. It was noted early in the research that gastric and duodenal ulcers, classic psychosomatic disorders, did not occur in spite of the anxiety and tension of life in the Ghetto. The reason for their non-appearence was disclosed by the studies on gastric acidity: hyperacidity was never found; instead, very low values of both free and combined acid were encountered, sometimes to the point of total achylia.[10]

Bile was very thin and was poorly excreted. Liver function tests showed a latent hepatic insufficiency. The digestive enzymes of the pancreas were found to be within normal limits.

Urine analysis was, surprisingly, normal. There was no change in the diluting or concentrating powers of the kidney. The edema was proven to be primarily extra-renal

in origin even though the kidney, like all the other organs, carried on its functions at a greatly diminished rate.

Many studies were made on various metabolic processes, within the bounds of experimental capabilities under the circumstances. The basal metabolism was decreased 30 to 40 per cent below normal, far lower than in myxedema; in two cases of extreme starvation cachexia it was minus 60 per cent. The specific dynamic action of protein was lowered or totally absent. The amount of carbon dioxide excreted by the lungs was greatly reduced, a concomitant of the disturbed pulmonary ventilation.[11]

Carbohydrate metabolism was intensively studied. Capillary and venous blood sugar levels were measured; glucose tolerance curves were taken. Measurements were made of the response to adrenalin and to insulin. The speed of the utilization of carbohydrates, using the respiratory quotient as a guide, was determined.[12] Prior to these studies, no extensive research into carbohydrate metabolism during starvation had been undertaken by modern methods; studies that had been made yielded equivocal results. The Warsaw studies established the reasons for the previously observed "self-healing" of diabetes. The doctors found that the highest fasting blood-sugar level was 80 mg. per cent; the mean was 60 mg. per cent; in several cases it was as low as 24 mg. per cent, a figure never previously reported in the absence of insulin adenomas or insulin shock. In spite of the low blood sugars, clinical hypoglycemic reactions were absent; the doctors interpreted this phenomenon as an indication of the paralysis of the anti-insulin mechanisms. Sugar tolerance was increased; there was no rise in the curve after stimulation by food or adrenalin. Ingestion of sugar was followed by its extremely rapid disappearance from the blood stream, indicating its quick utilization by the starving tissues. A surprising find-

ing, in view of the theory current at the time, was that the muscles gave up their glycogen, decomposed into sugar, into the blood. Since the liver glycogen stores had already been depleted, this indicates the mobilization of the only available peripheral reserve of glycogen, that in the muscles.

The swelling occurring during famines had been noted for a long time, but the Warsaw doctors were the first to study it by modern methods. Precise measurements were taken of water intake and output, water balance, renal and extra-renal ratios of water loss, water in the circulating blood, and water in the tissues. Mineral ash studies gave information on how water was divided between the plasma and the blood cells. Vascular wall permeability and the hydrophilic action of the subcutaneous tissues were studied by well established physiologic techniques. The doctors found: (1) a great increase in the water exchange, both intake and output going up; (2) more water was obtained from liquid foods than from that formed during the combustion process; (3) extra-renal loss of water was within normal limits, but excretion through the kidneys was increased; (4) even edematous patients excreted large amounts of water; (5) hydremia, an increased amount of water in the circulating blood, was constant in starvation; (6) the blood cells paradoxically became more desiccated and the plasma more dilute; (7) capillary permeability was unchanged; and (8) tissue hydrophilia increased, resulting in migration of fluid from the blood to the body spaces, thus bringing on edema, hydrothorax and ascites.[13]

The studies on mineral metabolism were broken off in the middle. The only detailed findings from which conclusions could be drawn were those on chlorides. Other than the expected findings, of low blood chlorides and the passage of chlorides from the plasma to the red blood cells, nothing unusual was noted.[14]

The alkalinity of the blood was studied in detail, as was the excretion of acid components in the urine, gastric juice and the expired air. The majority of cases showed a decrease in body alkalinity. Urinary ammonia excretion was increased, shown by the low titrable acidity. Many controlled experiments showed the presence of an acidosis uncompensated for by any increase in pulmonary ventilation or gastric acidity. Such an acidosis was another sign of the rigidity of the circulatory, respiratory and digestive systems. The acidosis, in turn, caused multiple disturbances in body functions.[15]

Spectrophotometric and micro-methods were used, together with other standard techniques, in the investigation of nitrogen metabolism. The results theoretically expected in a low protein, low calorie diet were obtained, plus the observation that creatine was excreted, a definitely abnormal condition.[16]

No studies on vitamin balance were made. The vitamin content of the diet was low to start with and was further lowered by the prolonged cooking of the soups dispensed by the soup kitchens and by the various processes used to preserve food. In addition, vitamin assimilation was also interfered with by the achlorhydria and by the rapid passage of food through the intestinal tract. The small amount of vitamins was balanced, however, by the very low calorie diet, the doctors said, basing their opinion on authorities who found that vitamin needs increase with the increase of calories. No clear-cut proof of any avitaminotic syndrome was found in any of the patients. There was no rickets, no scurvy, no night blindness, no keratomalacia, no pellagra. When hypo-vitaminosis-B was suspected in several cases of polyneuritis or of edema mistaken for beri-

beri and yeast given therapeutically, no improvement took place. "From our own knowledge we can state that no vitamin nor vitamin complex can eliminate the clinical or biochemical disturbances found in starvation disease."[17]

Immunologic reactions to tuberculosis were studied in great detail. The doctors were interested in finding out why starvation favors the development of tuberculosis.[18] Besides the physical and physiologic changes (pulmonary atony, poor aeration, low vital capacity, poor pulmonary circulation), a lack of immunity was also found. It was discovered that, even with gross tuberculosis, the intradermal and conjunctival tuberculin tests were negative.

The lack of response to tuberculin led to several studies on allergic reactions in general. Bronchial asthma and hives were rare, as were other diseases attributed to allergens. The skin sensitivity was diminished in all the tests made.

AUTOPSIES

There was plenty of autopsy material. From January 1, 1940, to July 22, 1942, 3658 autopsies were done. Of these, 492 were cases of "pure" starvation, proved by the absence of any complicating disease. Obviously, it was technically impossible to do an autopsy on every patient dying in the hospital, especially in starvation cases, which ran to 20 to 30 per cent of the total deaths daily. The care with which the autopsies were done is shown by the meticulously detailed charts and summaries, as well as by the protocols.[19]

Sections were taken and examined microscopically. For the first time in medical literature, the microscopic appearance of sections of the nervous system in individuals dying of starvation was reported. Careful descriptions were made of the slides taken from the endocrine glands, the

heart, and all the other organs and tissues. Considering the condition under which the doctors labored, it is astonishing to read their reports about the microscopic appearance of the pituitary gland, about special stains for iron in liver cells, about minute changes in the layers of the skin.

The investigators found, as others had before them, total disappearance of all adipose tissue and an atrophy of the vital organs. The skeletal muscles were greatly shrunken. They pointed out that such changes are understandable because when the body does not get sufficient caloric material for its energy needs from food it first uses up all its own reserves and then is forced to burn its own tissues.

A surprising finding was the presence of a senile type of emphysema of the lungs, even in young people. Such changes were regarded as the result of atrophic changes in the lung tissues.

All the organs of the body (heart, liver, spleen, kidneys, etc.) showed atrophy, with one exception—the brain. Its weight, although less than normal, usually was far greater in proportion to that of the other organs.

One striking disagreement with other authorities who stressed the frequency of hemorrhage during starvation was noted: it appeared in only two of the cases studied in Warsaw (excluding the small number of minor intestinal bleedings and the greater number in pseudo-dysentery). About the latter, the doctors noted that the intestinal changes were characterized by mild inflammation superimposed on more or less swelling of the mucous membranes of the intestine. They felt that these swellings were symptomatic of the general tendency to edema found in starvation. They concluded that cases with intestinal changes resembling those in true dysentery should be regarded as having a complication of starvation disease because of the gross and microscopic evidence of their recent origin. Such

changes should be considered either as the result of secondary infection on the swollen intestinal mucosa or as mucosal lesions from abnormal toxic metabolic products, such as occur in uremia; they have nothing in common with true dysentery and should be properly labeled pseudo-dysentery.

"Bone material taken in the autopsies was destroyed during the expulsion from the Ghetto."[20] Note was made, however, of the marked porosity of the bones. Fracture of the neck of the femur was frequent. The surgeons often complained of the poor quality of the bony structures, which made healing difficult and operative technique sometimes impossible. In one preparation from the head of the femur, bone porosity and decalcification were present, changes halfway between osteoporosis and osteomalacia. The doctors concluded from their studies that it is likely that hunger osteopathy is not a distinct anatomico-clinical entity, but rather that, under this name, different bony changes with a common etiology have been described, occurring in various stages of prolonged malnutrition. The bony changes vary depending on the degree of malnutrition and other conditions, thus explaining why, under the name of hunger osteopathy, a series of changes have been reported, starting with common osteoporosis and going on to atrophies, decalcifications and osteomalacia. "Hunger osteopathy" is merely another sign of the general atrophy of the organs so typical of starvation disease.

The rarity with which amyloidosis appeared (6 cases in all), was commented on. Its incidence was to be expected in view of the prevalence of tuberculosis and chronic suppurative conditions; however, it was not found even in those cases of very long standing infection. The investigators felt that the essential cause of the rarity of amyloidosis

in their material was the great lack of protein in the diet, thereby agreeing with the Japanese authorities (especially Tanaki), who explain the infrequent occurrence of amyloidosis in the Japanese by the lack of sufficient protein in the Japanese diet.