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SURVEY OF TEN YEARS' RESEARCH ON MAJOR ENDEMIC DISEASES, SHOCK, AND ARTERIAL BLOOD AND FLUID TRANSFUSIONS

-COMMUNIST CHINA-

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SURVEY OF TEN YEARS' RESEARCH ON MAJOR ENDEMIC DISEASES, SHOCK, AND ARTERIAL BLOOD AND FLUID TRANSFUSIONS

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/Following are translations of three articles appearing in Chung-hua Ping-li-hsueh Tsa-chih (Chinese Journal of Pathology), Vol. 5, No 4, 1959. The figures are appended to the end of each article./

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RESEARCH ON THE PATHO-ANATOMY AND EXPERIMENTAL PATHOLOGY OF LARGE JOINT DISEASE IN CHINA DURING THE PAST DECADE

By Li Pei-ling (Mukden Medical School) and Wang Heng-wen, China Medical Scientific Institution.

"Large Joint Disease", which is also called "Liu-ling-tse Disease", occurs mostly in the northeastern and northwestern parts of our country. Its symptoms and pathology indicate that this disease is similar to /Kaschin-Beck Disease/ or "Wu-lo-fu Disease" which occurs in the USSR. (1) The etiology and onset of the disease have not been determined definitely thus far. Research on this particular subject, observation, patho-anatomy, and animal experiments to discover the typical causes of this disease are, indeed, very important.

However, even though many people are afflicted with this disease, before liberation our country had not done any preventive or research studies on this subject. After liberation, the Peoples' Government, having a close interest in the public health problem, set up disease prevention headquarters locally. These were established initially in the northeastern parts (Kirin Province Garrison and Heilungkiang Province Pei-an), and subsequently in other affected areas. A large part of the work dealt with prevention of the disease and examination of patients. Since large joint disease does not lead to immediate death of the patients, and also since the results of research on its patholologic anatomy have not been conclusive, we have had, up until now, very little basic information to go on.

In 1956 Li Pei-ling (2) of Mukden Medical School collected material on the "abnormal physiology and histology" of the disease. He recognized that the areas of the body which are mainly affected are the extremities of the cartilages, in which the early stages of degeneration begin. The extremities of the cartilages become irregular and uneven, especially in the case of long, compact bones. The tissue becomes red, stained, softened, bubble-like or slitted /with/ the appearance of fibers and non-vital cells. (Figs. 1,8) In the cartilages, the cells become uneven and irregular, with loss of normal shape. The normal characteristics of cells may disappear altogether. And around this areas bone cells may gather themselves into

clumps. At the extremities of the cartilages small, normal bone spicules sometimes appear, but in cells and connective tissue, degeneration may also appear, and the extremities may arrange themselves into a horizontal bone framework, indicating that the growth of bone has stopped. But blood vessels around the horizontal network sometimes can be seen extending into the bone, thus stimulating new bone growth. Often it becomes double the normal thickness. Within the extremities and cavities of bone there are many "lumps" of individual cartilaginous organization (so-called cartilage callus or island). Near the joint side of the cartilage, Mr. Li discovered that there may be an increase of thickness, with surface appearance of ulceration and cracking. In the deep substance of the joint side of the cartilages, can often be observed disappearance of whole layers of cells. Besides this, about 20 or 30 cartilaginous cells congregated into colonies can also often be seen. The appearance at the joint side of the cartilage often changes to grain-like, wavy, bubble-like and slitted /appearance/. (Figs. 3-6) Sometimes the substance in cartilage /sic/ becomes softened, and pale in color. As far as spongy bones are concerned, Mr. Li thinks the joint disease manifests itself by the breaking down of small bone units /and/, the swelling and dying of bone marrow. He pointed out that not enough attention has been paid to such features by persons previously studying this disease. Mr. Li thinks that in the extremities, the joint side of cartilage, the spongy bone, and in the marrow, no individual variation of disease or early degeneration is especially out of the ordinary. But a combination of those variations, the incidents of the disease, the overall condition and location of occurrence of disease, become the characteristics of the disease. Among the variations, there is no invasion of inflammatory cells. It can therefore, be stated that the disease is not an inflammatory /process/, and the importance is placed on malnutrition. Besides that, Mr. Li points out that the pathological changes of this disease occur first at the extremities; then it spreads into the joint side of the cartilage.

In 1956, the Large Joint Disease Research Study team of the Health Department at Pei-an City in Heilungkiang Province observed four subjects; a seventeen-year-old with affected finger bones, and three others aged respectively nine, seventeen, and thirty-five. It was discovered that the anomaly occurs in multiple sites of the body, bilaterally, and most commonly around the joints of the wrists, knees, and shoulders. The joints are roughened and uneven,

and the cartilages become ulcerated. The ulceration is seen most at areas of friction and stress. The cartilages on the joint side are uneven in size and brownish in color; swelling occurs. They may shape themselves into small channels or split (Fig. 1). When persons are severely infected, the periphery of the cartilage becomes disk-shape and extends outward. The histological findings are as follow: The fiber-ends in the joint cavity are enlarged, connective tissue is stained red, and characteristics of fibers change. (Fig. 2) Cartilaginous cells do not line up evenly, group characteristics disappear. In addition, the cells may clump into cartilaginous colonies, and the characteristics of cells within a colony may also be changed.

Results of the team studies (3-8) of the pathology, together with case studies, confirm that the "enlarged joint disease" in the northeastern part of our country and the USSR's "Wu-lo-fu disease" are one and the same.

In 1958, the patho-anatomy staff of Hsi-an Medical School reported (9) that in Shansi province, City of Huang-Lung, four cases were studied (males ages 15, 17, and 29, and a baby). The examiners found that all four patients had in the extremities, curviness of bone, uneven thickness, and early degeneration in some places; the extremities might be enlarged to double the normal thickness. Microscopically, the main features are uneven line-up of cartilaginous cells, disappearance of whole groups of such cell colonies, abnormal shapes, and death of connective tissue cells. In two cases there was swelling and breaking down of the bone units in the marrow. The examiners concluded that because of the increased activity of cell multiplication in certain areas, recovery is quite possible, providing that the disease is treated in time, before complete degeneration takes place.

In 1959, Hunan Medical School's Chao Liu-ko reported that Hunan Province, Ling-Pao County, there were two cases of this disease. (10) The first patient was a 29 year old male who had had this disease since he was about ten. Post-mortem examination disclosed bilateral swelling of the joints, especially around the knees, wrists, fingers, and toes. The fingers and toes seemed shorter than normal. Cartilage at the margin of joints appeared convex and turned outward in a "lip shape". The rest appeared uneven in structure and color. In certain areas grayish or yellowish spots appeared. The joint side of the cartilages often appeared broken or split, or partially lost. The membrane was roughened and full of small fibers. Microscopic examination revealed that there might be loss of

cells at certain areas, the fibers enlarged, blood vessel walls thickened, hemmorrhage, and increase of tissue organization, in which a few inflammatory cells existed. As for the joint side of the cartilage, the connective tissue had become fibrous and non-vital. In some areas cartilaginous cells had disappeared, in some areas many cell colonies appeared. In the extremities small bone units were arranged irregularly, sometimes with broken or devitalized cells, but when the cartilages in joints split with exposure of bone, bone units might appear thickened in this localized area. Nearby marrow had become fibrous. The fibrous organization may invade the cartilage. In the marrow there may be emptied a membranous organization shaped like small vacules.

In the report there is another case, a 58 year old male who was afflicted with this disease when he was about 10 years old. With limited activity, the pain in his joints increased a stime want by. Within the last 10yyears he had lost almost all /ability to/ move and /carry on/ normal daily activity. In 1958 he underwent treatment by a native doctor in Hunan Province and achieved exceptional results. The patient underwent needle-puncture therapy /acupuncture/, Pa-huo-kuan therapy, and interal treatment with Ma-ch'ien-tsu tablets. Eventually the swelling lessened, pain disappeared, joints became movable, and a certain degree of activity was restored. However, after several months the patient suddenly revealed symptoms of K'o-Shan disease and subsequently died. Post mortem examination disclosed enlargement and expansion of the heart, hardening and obstructing of the coronary artery, and witish scaring of heart muscle, especially the left ventricle near the apex. Under the microscope one could see the non-vital muscle fibers with a large amount of scar tissue which are similar to the appearance of chronic K'o-Shan Disease. The joints of the patient's arms and legs were swollen and enlarged. The membrane, certilage, bone units, and marrow have the same appearance as in the first case; as also cartilaginous islands were present. However, in the joint disease there was no invasion of inflammatory cells.

The authors agreed that the disease lacks any specific symptoms. Because of the results obtained in 1958, when the Chinese native doctor, using Chinese herb medicine, treated the patient and relieved his pain and swelling, the authors are inclined to believe that the pathological process is not confined to the joint, but also /affects/ the surrounding soft tissues (nerves, muscle ligaments, adipose tissue, and membrances). Therefore, it

should be pointed out that in studying this disease special attention should be paid to those soft tissues.

The cause of this joint disease, at the present, is still not certain. In the USSR there were theories such as spirochetal food poisoning, malnutrition, or excess phosphorous and deficient calcium. (11) When dogs and white mice were fed food injected with spirochetes, a certain amount of degeneration occured. However, this food poisoning theory has not yet attained unanimous approval.(12)

The bacteriology Department of the Central Health Research Bureau (Kuo K'o-t'ai, Ts'ao Shou-li, Chang Han-hung, Kuan Ch'ung-fen and Liu Hsing-ling) and the Pathology Department (Yang Chi'en, Wang Hung-wen, Lu Yao-tseng and Shen Chung-ying) organized the Joint Disease Research Center in Chiling Garrison and collected over a hundred species of spirochetes from water and food samples. (13) According to their shapes and growth characteristics, the bacteria were classified into 68 types, then the similar types were combined into six groups. The bacteria in each group were incubated separately in millet culture media. When the growth reached its peak, heat was applied to attenuate the bacteria. The culture was mixed with feed. The feeds were fed to rabbits, hamsters, and large and small white mice. The result was that from the fifth group, three types of the bacteria succeeded in infecting the animals; their bone development was hindered at a constant degree.

Between 1955 and 1956, these three types of bacteria were fed to 37 young, large white mice and 11 young dogs, using as controls animals of similar age and weight or from the same litters. The animals and their controls were killed and bones were examined. Among the white mice, the experimental animals revealed certain definite indications of degeneration of cartilage: the over-all long bones were shorter than in the controls, bone units were smaller and more sparse, bone extremities were curvy with uneven thickness and, in general, thinner. In the extremities, the rate of multiplication and number of well-formed cells were decreased, and there were more vacuoles and abnormal fiber cells. However, none of the experimental mice revealed any changes in the joint side of the cartilage. (Fig. 13-16)

Among the dogs that had been fed group-5 bacteria, one dog, fed the bacteria continuously for 5½ months, had thickened ankles of the 2 fore limbs and the plantar side of the toe-joints (Fig. 9). Excepting for one, all eleven dogs showed a definite degree of bone degeneration and

developmental hinderance, which included thinness and irregular shapes of the extremities, lack of normal rate of multiplication and large normal cells, increase of vacuoles, uneven and irregular extremities, and bone units thinner and shorter /then normal/. (Fig. 11, 12) In addition to these conditions, the cartilages at joint side showed some degeneration; the cartilaginous cells became small, sparse, died and disappeared (Fig. 10). Three of the experimental dogs, including the animal with thickened joints, had increased numbers of non-vital patches of cells and cartilaginous cell colonies. Two of the experimental dogs had joints with abnormal fibers and fibers invading the cartilages. None of the control dogs manifested any of the above changes.

The authors are of the opinion, as a result of these two experiments, especially the dog experiments, that there is a definite similarity of the disease in dogs and humans, though the degree of infection is less in dogs. But from 1956 on, the feeding of Group-5 bacteria to animals has not produced any clear-cut case of this joint disease. It is possible that the virulence of the bacteria has decreased under laboratory conditions.

During 1956, Hsi-An Medical School (14, 15) had isolated eight types of virulent spirochetes from food and water samples. In 1957, after cultures had been obtained on millet culture media, they were fed in pure form and mixed with feed to 13 young dogs, and at the same time the nutritional value of the feed was decreased. Five other puppies were used as control. After four to 14 months the bones were examined; a slight change in the extremities was noted, but the real sumptoms of the joint disease were not duplicated. An experiment with 19 pairs of white mice gave the same result as with the dogs.

Because we are unable to produce the disease in animal, great further effort must be put into research.

Summary:

During the last ten years, the pathologic anatomy and other aspects of this joint disease have not been /studied/ enough. From the material available, this disease appears similar to the USSR's Wu-lo-Fu Disease. There is no single specific symptom that a diagnosis can be made from. But the many incidents /sic/ of the disease, the bilateral characteristics, and the various degenerative phenomena, together with the absence of inflammatory reaction, are the main points of the disease. According to the results of recent treatment by a Chinese native doctor, special attention should be paid to the surrounding soft

tissues; this point has generally been overlooked by previous researchers. From the result of experimental work on the spirochetal food poisoning theory, a condition similar to joint disease has been produced in white mice and dogs, though not to such a severe degree as in humans. These experiments should be repeated and revised in order to obtain conclusive results.

Fig. 1. 29 year old male, left knee joint; severe damage inside the joint, with irregular slits and ulceration (Chao Ling-ko, Reference 9).

Fig. 2. Membrane around the knee joint of a 58 year old male: fibers increased and thickened with increased blood vessels and connective organization; walls of small vessels also thickened. (Chao Ling-ko)

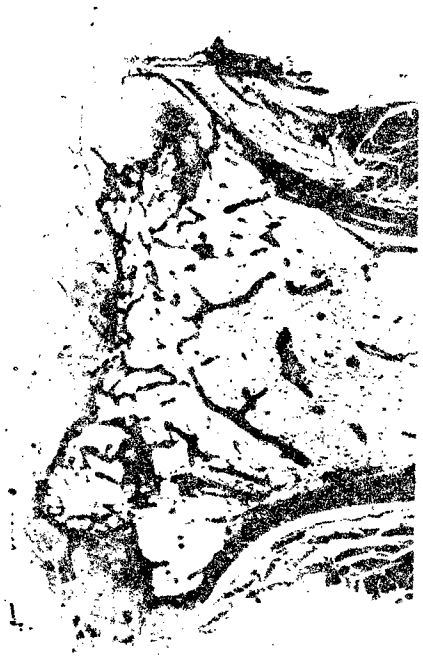

Fig. 3. Tibia of a 16 year old male: the disk shape and "lip-shape" at joint side of the cartilage, which is thickened and broken. Disappearance of bone surface with sparse bone units. (Li Pei-ling, reference 20).

Fig. 4. Ulceration of joint side of cartilage, change of characteristics of connective tissue. Loss of surface cartilaginous cells. Change from normal characteristics of such cells in deeper layers, resembling fiber cells. (Li Pei-ling)

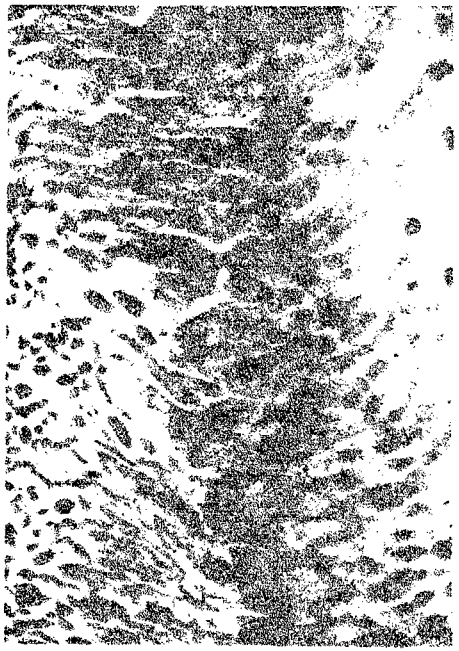

Fig. 5. Non-vital area of joint side of cartilage, with broken surface, fibrous arrangement, and small cell colonies in deeper layer. (Li Pei-ling)

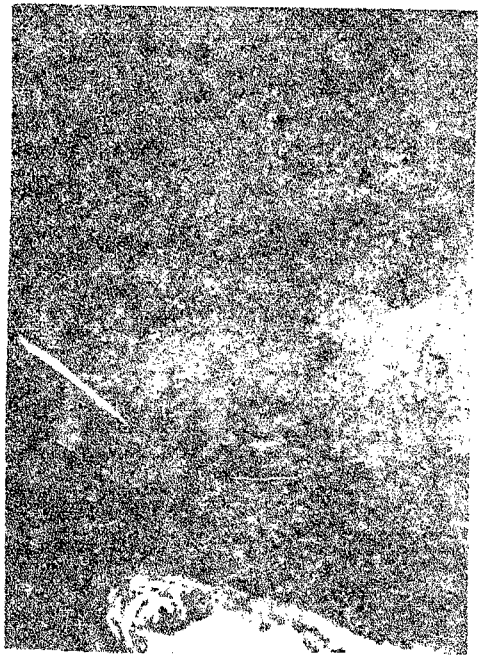

Fig. 6. Finger bone of a 17 year old male: cartilage at joint side has various areas of cell-less vacuoles. Cartilaginous cells clumped together. (Pei-an County, Heilungkiang Province Research Disease Center).

Fig. 7. Tibia bone of 14 year old male: uneven extremities, cartilaginous island on one side and horizontal bone units on the other. Sparse bone units. (Li Pei-ling)

Fig. 8. Lower extremities of a 15 year old male: irregular extremities, with early degeneration in three places. (Li Pei-ling)

Fig. 9. Left Figure: 7⅛ month old bitch that was fed Group-5 bacteria continuously for 5½ months: markedly increased thickness of the ankle and toe joints of a fore limb. Right Figure: control animal of the same experiment. (Wang Heng-fung, Reference 12)

Fig. 10. Tarsal bone of the animal in Fig. 9: large non-vital area at the joint side, with congregation of surrounding cartilaginous cells. (Wang Hung-fung)

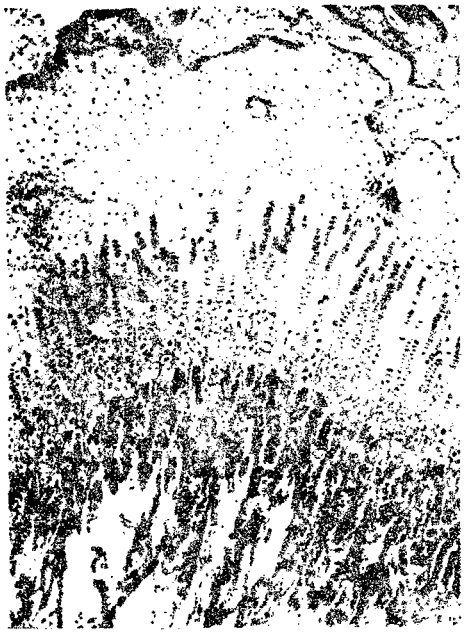

Fig. 11. Upper extremity of /elbow bone?/ of a 3 month old male dog who was fed Group-5 bacteria for two months. x 60. The extremities are thinner with a slight curve. Irregularity of cartilaginous cells. Thickening of most of inner layer and thinning of two outer layers. More area without cells and abnormal fibers. Sparse bone units near extremities. (Wang Heng-fung)

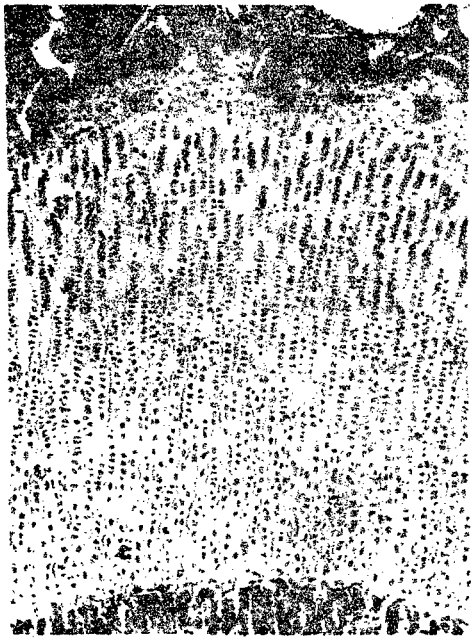

Fig. 12. Control animal in the same experiment as in Fig. 11. (Wang Heng-fung)

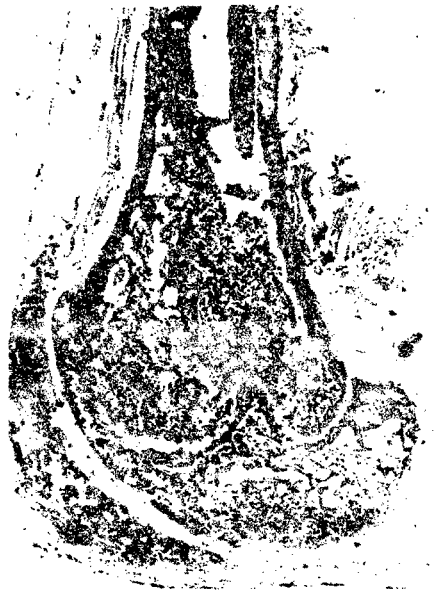

Fig. 13. Lower extremity of the hip bone of a 17 week old large white mouse which was fed Group-5 bacteria for 13 weeks. x 10. The joint side of the cartilage protrudes outward. Outer covering thin and curved. Small bone units decreased, shortened, and irregular. (Wang Heng-fung)

Fig. 14. Control mouse from the same litter as the one in Fig. 13. (Wang Heng-fung)

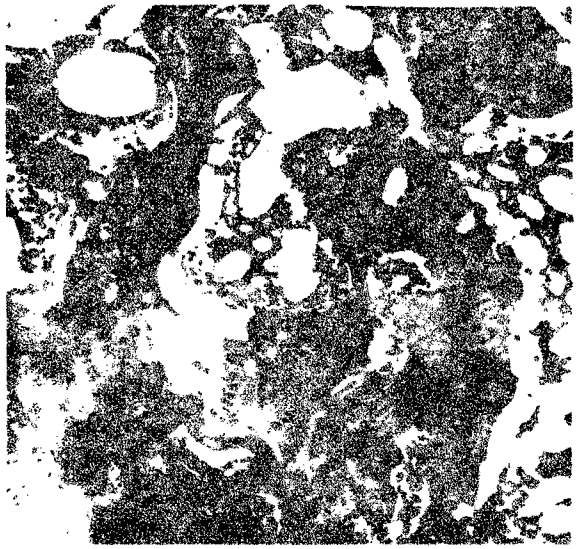

Fig. 15. Cartilagenous area of lower extremity of the hip bone in Fig. 13. x 120. Cartilaginous cells shrunk and decreased in number. Early appearance of fat. (Wang Heng-fung)

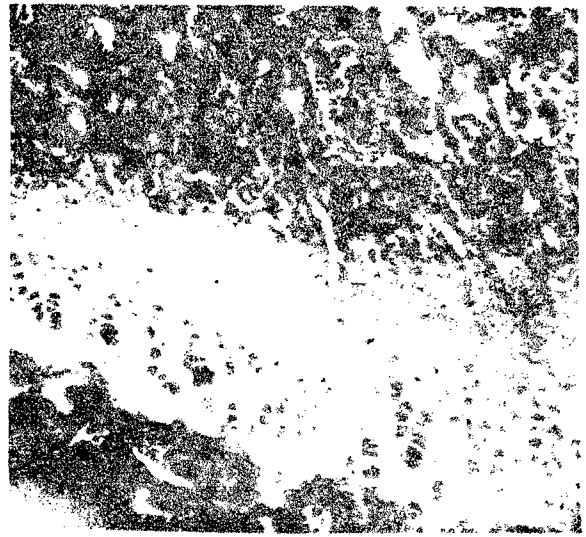

Fig. 16. The same mouse as in Fig. 14. (Wang Heng-fung)

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RESEARCH ON THE PATHOLOGY OF K'O-SHAN DISEASE IN CHINA DURING THE PAST DECADE

By Lu Yen-jung, Pathologic-Anatomy Faculty, Peiping Medical School.

K'o-shan disease is one of the most important diseases in the northern part of our country. Before liberation, little was done about anatomical research. But after liberation, research centers were set up in our medical schools in the northeastern and northwestern parts of our country under the leadership of the Party.

In the northeastern part of the country in 1950 Ying Tai-k'ai (1) and his associates studied the hearts of five persons afflicted with this disease. In the heart muscle there was evidence of drying up of muscle fibers with increased grain-like appearance.

In 1952, Chen Tung-ch'i (2) dissected one cadaver and made a complete examination of all vital organs. In the cardiac muscle he found scar tissue with broken muscle fibers. The brain tissue seemed normal.

In 1956 the Central Health Bureau organized headquarters specifically to study this disease. The headquarters are located in Heilungkiang Province, Harbin Medical School and Peiping Medical School. In 1957, these institutions dissected 24 cadavers to study the pathological changes in the heart (3). Important findings included enlargement and rounding of the heart, also the simultaneous existence of newly devitalized tissues and old scar tissues. Destruction of heart muscle was of two types, dissolution and disintegration. Congregtion of cytoplasm is the early symptom of dissolving muscle. The conclusion drawn is that there is non-cellular hardening to form scars with no evidence of chronic inflammatory infection. Scar tissues are of four types, classified according to the distribution of pathological changes in the smooth muscle and the distribution of capillaries in the muscle. It is thus deducted that the etiologic agent may be carried from the blood into the muscle.

In 1958, the Pathology Department compiled and analyzed the results of 103 dissections within the last five years (1953-1958) (4) and confirmed the conclusions of the 1957 studies. At the same time, further research was done on the pattern of infection and its relation with the rest of the body: injury of skeletal muscles, stimula-

tion of heart and carvical nerve junctions, brain, spinal nerve changes. Also, 14 fetuses from patients who had succumbed were examined. It was discovered that the hearts of the fetuses gave evidence of K'o-shan disease symptoms.

In the northeastern part of our country in 1958, /researchers at the/ Hsi-an Medical School concluded that in the Huang-lung area, 36 cases of "Yellow Water Vomiting Disease" (5) /a descriptive translation/ were the same as K'o-shan disease in the northeastern areas. In one of the cases mentioned above, the heart of a fetus displayed the same symptoms as in K'o-shan disease. In addition to the above-mentioned research units, the medical school of Yen-pien College and Lan-chou Medical School also set up research centers for this disease. Both Li Pei-ling (6) and Hung Pao-yuan (7) published reports on this disease.

Under the Party's leadership and with the support of all personnel doing research on the pathology, a great deal of dissection information have been collected to provide /a basis for/ further study and give understanding to the whole facet of pathology and furthering the understanding of the heart muscale changes, thus giving the study of cause and pattern of disease a firm foundation. This report is based mainly on the results of 103 autopsies performed in the northwest, with /?special/ reference to the research work done in the northwest. The report of K'o-shan disease is as follows:

The Pathologic-anatomy of K'o-shan Disease:

The 103 patients in northeast part of the country all died of K'o-shan disease. The youngest patient was four years old and the oldest 60 years old; 68 were females and 35, males. Of the 36 cadavers from the northwest, there were 24 males and 12 females. Because of the definite seasonal incidence of this disease, most of the specimens were collected during the winter.

A. Pathology of heart:

Gross Examination:

The degree of enlargement of the heart varies, with the largest two or three times /larger/ than normal. The most prominent change of the outer surface was the increase of horizontal ligaments; often there were as many or even more than longitudinal ligaments, thus giving the heart a somewhat spherical shape. Two-thirds of the hearts examined were heavier than normal. The increase in weight was

ad much as 560 grams; at least one-third of the hearts had an increase of 100 grams or more. The chambers of all hearts were markedly expanded, especially the left heart ventricles with papillary muscles and "ju-tou" muscle appearing flat (Fig. 1). The muscle became slack because of the original expansive characteristics of the muscle. The heart walls had not thickened but in cases in which there was a high degree of expansion of the heart chambers the walls became thin; the closer to the apex the thinner they became. In abot one-quarter of the cases there was adherence of blood "ch'uan" in the chambers. Often it could be seen in both left and right auricles or in the papillary muscle of the left ventricle.

The main pathological changes seemed to be in the heart walls; while the membranes did not display any significant changes, neither did the coronary arteries. In the ventricles, underneath the membrane, were grayish-white or brownish-yellow pathological changes especially through the papillary muscle and "ju-tou" muscle, having the pattern of tiger skin. On the cut surface of the heart wall there was often a "spreading" distribution on the external surface with an irregular number of grayish-white or yellowish pathological changes, and the muscle could be stained into a peculiar flower-like shape (Fig.2). The pathological tissue might approximate the size of a chestnut, with the largest 0.2 to 0.3 "li-mi" in diameter. The tissue might be arranged in star-like lines or connected in serrated lines (a zig-zag line). When an area had an abundance of scar tissue, it might be arranged in irregular patches, often mixed with brownish muscle organization. The brownish pathological tissue did not have any clear demarcation, but did have a slight convexity at the cut surface. Microscopic examination showed that the muscle fibers had either died or dissolved. However, in the grayish-white area, the demarcation was more definite; the surface often showed concavity with a slight degree of transparency which were part of the scar tissues. Even though this pathological change occured in both ventricles, its distribution was not even. There were more pathological changes in the "ju-tou" muscle, left heart ventricle, and the area between the ventricles than in the right ventricle. However, they were difficult to detect with the naked eye. From observation of the heart wall, it appeared that papillary muscle in the inner surface of the heart muscle was more involved than the outer surface of heart muscle (Fig. 3).

Among the different cases of the disease, the

severity of involvement of the heart muscle varied. The more severely the muscle was involved, the greater the expansion of the heart cavity and of the heart as a whole.

Microscopic examination:

The /pathological/ involvement seemed to be in the muscle with uneven distribution of non-vital tissue, scars with large "grainy" malnutrition patches and also congregation of cytoplasm. Inflammatory cell invasion and multiplication of cells were not prominent. On occasion both pericardium and endocardium could be seen invaded by lymph-like cells. The heart valve membrane did not display any obvious pathological changes.

1. Non-vital cardiac muscle

In the non-vital zone, smaller areas involved only three or four muscle fibers, larger area could be seen with the naked eye. Sometimes dead muscle could be seen in large patches, but invariably some normal healthy fibers were mixed in those areas. It is quite possible that those large patches were the result of coalescence of adjacent smaller patches. Most of the nin-vital muscle were of the dissolving type (Figs. 7, 8).

In dissolution of muscle fibers, the fibers became enlarged. First the outer cytoplasm dissolved, followed by the thinning of inner cytoplasm with the appearance of vacuoles and many small vacuoles which combined into larger vacuoles, /until/ only the framework of muscle membrane remained (Fig. 6). Nuclei and their surrounding cytoplasm remained longer than the fibers. The capillaries in the connective tissue were still preserved, therefore after the dissolution capillaries, small veins, and small blood drains were still present in the connective tissue. There was also evidence of hemmorrhages. Staining of the fibrous network has proved that the reticular structure was still intact.

In the disintegration of muscle fibers, the cell membrane was destroyed, the outline of fibers obliterated, and cytoplasm broken into pieces. In the connective tissue there were often isolated brownish fat granules and leucocytes. From the staining pattern it could be seen that the fibrous /reticular/ network had been broken and destroyed. Therefore it is obvious that this type of disintegration is far more severe than dissolution.

In the non-vital areas of smooth muscle there was occasionally evidence of regeneration, /such/ as the

splitting of mother cells and their nuclei. This regeneration was more obvious in children. When tissue became non-vital through disintegration, there was evidence of increased number of fibrous mother cells. As distinguished from acute or long standing cases of the disease, there was an increased number in the smooth muscle of capillaries and the membrane of small blood vessels, and an invasion of lymph-like cells. Inflammatory cells were not prominent, in the non-vital area, but small numbers of lymph cells, acidophilic white blood cells, and mono and multi-nucleated white blood cells could be seen around the blood vessels, and the cytoplasm had spilled through.

Distribution of the non-vital area

There seems to be no significant relationship between the distribution of non-vital area and of the coronary artery with its larger branches. However, there seems to be a definite similarity between the distribution of non-vital areas and of the netlike distribution of small blood vessels (Fig. 11).

The dissolution type of non-vital area appeared only in "ju-tou" muscle and in the left ventricle and its valve. The degree of involvement was less in the right ventricle, with even less, if any, in the auricles.

2. The "grainy" appearance of cardiac muscle and the clumping of cytoplasm

Under the low power microscope, grain-like particles that stained red were present in the cytoplasm. Under the high power, these particles were shown to be composed of the broken muscle fibers. Of special interest is the fact that these pazrticles often congregated with cytoplasm and lined up perpendicular to the muscle fibers. They might be darkly and evenly stained, with uneven cross striation, or grouped into cotton-like patches. In cytoplasm between these areas they were lightly stained. Under those conditions the fibers often enlarged, with uneven diameters along the whole length of the fibers (Figs. 9, 10).

3. The effect of fat deficiency on cardiac muscle

The muscle fibers often manifested a diffused type of fat malnutrition. There seemed to be no relationship between this pathological process and the blood extravasated in muscle. Sometimes, when the course of the disease was short and acute, pathological changes in muscle also reached an acute degree, whereas when the tubercular type

of chronic condition occured, the pathological changes were negligable. This indicates that the malnutrition of fat is not due to the environmental obstruction, but is rather a manifestation of the result of the disease.

4. The scar tissue of cardiac muscle

In most cases of heart involvement a certain amount of scar tissue is present. According to the staining methods of "van Gieson, Mallory and Pollack" scar tissue can be classified into 4 types: (1) Isolated, small, star-like scars along the sides of small blood vessels; (2) branch-like scars distributed along the small blood vessels between the muscle bundles (Fig.12); (3) Net-like scars at broken surface areas, large size gelatinized fiber bundles, lined up in the same direction as smooth muscle fibers often appear; at certain intervals they connect with each other like a net by piercing across the smooth muscle bundles (Fig. 14), and (4) Large scar tissue; in the centers of these irregular patches, there are often the remenants of muscle fibers. At the same time, their margins retain the net- or branch-like scars (Fig. 15). Perhaps these large patches result from the combination of smaller scars.

Sometimes both old and fresh scars are present. The fresh scars are usually spread out thinner than the old scars, which appear glazed. This indicates that the scars are not formed simultaneously.

Either with same or different diseases, one can see the transitional stages of muscles just before and after the formation of scars. The cytoplasm has already disappeared and the fiber membrane has collapsed and become dense. Staining shows that the net-like fibers have become gelatinized; the little cells remain and the gelatinized fibers become more dense. This is followed by a repairative process. There is no chronic inflammatory reaction during the change from non-vital to scar forming.

5. Changes in connective /tissue/ in smooth muscle

The longer preservation of connective tissue, even after the muscler substance has been severely damaged, is quite obvious. There are exposed many broken surfaces of blood vessels, not only in those large non-vital patches, but also in the large old scar tissue, which presents the same picture. In the non-vital area, there was some invasion of inflammatory cells and increase in the number of spindle-shaped cells. There were also lymph cells and

acidophile white blood cells, mono-nucleated and multi-nucleated white blood cells invading the blood vessels. Cells of capillaries were often swollen.

6. Pathology on stimulation of heart conduction system

Using the Maurice method for collecting samples, four pieces of tissue from between the ventricles were obtained. These were made into histological slides. Seventeen samples were examined. There were no pathologic changes in either ventricles or auricles. Three of the samples showed small scars in the bundle of His. In the left side bundles, four samples revealed lumping of cytoplasm. Three samples had small scar tissue. In the right bundles, there appeared some non-vital area and scars, as /?in/ the nearby smooth muscles. At the lower part of the sample tissues and wall, especially in the convex area of papillary muscles, one could often see the lumping of cytoplasm, dissolving of muscle, and scaring of Purkinje fibers in ventricle.

7. Examination of smooth muscle for toxic "Pau-han" bodies

There were no "Pau-han" bodies.

B. Examination of striated muscle

Examination of diaphram muscle, rib muscle, and tongue muscle revealed some degree of insufficience of fat. In some cases there were scars and non-vital spots, which presented the same histological picture as smooth muscle.

C. Examination of the Central Nervous System

Detailed examination of brains and spinal cords of 70 victims gave no evidence of softening of brain tissue, gelatinous degeneration of nerves, or increas of diffused gummy type of cells. All cases showed malnutrition of nerve cells (disappearance of Nissl bodies, flattening of nucleus, and hemorrhage of nucleus). Eleven specimens showed slight hemorrhages around the brain substance.

D. Examination of cervical nerves

Fourteen cases from the northeastern part of the country were examined. All showed malnutrition of nerve cells (swollen, wrinkled, shrunken vacuoles and breakdown of Nissl bodies). Eight specimens showed increased pigments.

The 12 cases from the northwestern areas displayed the same picture.

E. Examination of other organs

Pituitary gland: Of the 66 cases examined, 35 cases had mild chronic inflammation. Of the 18 cases from the northwestern area, three showed chronic pituitary inflammation.

Lung: All cases seemed to have extravasated blood with edema; wix had hemorrhagic obstruction. There was no relationship between fat insufficiency and extravasated blood.

Kidney: All cases showed fat insufficiency and extravasated blood. In all cases there was a parallel relationship of fat insufficiency between heart, liver, and kidney. This can be explained by the etiology arising from the malfunctioning of different organs.

Adrenal gland: Rich deposits of fat.

Stomach: Mild hemorrhage with no evidence of an infectious process.

Intestine: Adhesion of membrane or hemorrhage, mostly in duodenum. Hemorrhage occured in 21 of the cases [examined].

"Chia-chuang" Gland [possibly thyroad gland]: Local swelling in 41 cases.

Examination of fetal hearts in 14 cases of fatal K'o-shan disease:

In the northeastern part, the 14 fetuses were all from mothers who died of this disease. Except in two cases, all bodies underwent patho-anatomic examination. Of the 14 fetal hearts examined, seven showed sumptoms typical of K'o-shan disease. All of these fetuses were more than 7 months old; fetuses younger than six months showed no symptoms. Both non-vital areas and scars were present in the fetuses, though to a lesser degree than in their mothers (Fig. 16). The right ventricle was affected more than the left; the expansion was also more obvious in the right ventricle. There were no pathological changes in the placentas.

Discussion:

A. The characteristics of K'o-shan disease

Japanese pathologists have published a few

case histories. From these reports it appears that this disease is a disorder of smooth muscle with enlargement of the heart, expansion of the centricle, excess of fat in the heart structure, breakdown of fibers, invasion of inflammatory cells in connective tissue and formation of scars. As to the pattern of disease, characteristics of the disease, distribution of blood vessels and stimulation conduction relationship [these factors] have not yet been examined and analyzed in detail. There are also differences in opinion (8) regarding the presense of connecting fibers, the repairative process, or the increased muscle organization process. However, we are quite certain of the pattern of the disease outlined by our postmortem examinations. First, the smooth muscle dissolved and became non-vital, later on the remaining membrane collapsed, with net-like fibers gelatinizing and forming a scar which ultimately repaired the "space". The shape and the distribution of non-vital area and scars correspon to each other (Figs. 11, 12). This is enough to establish that scar formation is the result of repairative process of smooth muscle.

In five long term patients, non-vital areas had been repaired with no further evidence of devitalization. In some cases, after a one-month rest in the hospital, there is manifested fresh scars in the muscles. Therefore, it is thought that the muscle membrane framework remains longer and complete conversion to scar tissue seem to be a slow process.

In some literature the reported progressive smooth muscle failure (Fiedler's myocarditis) is also one of the more important heart diseases, with eakening of heart as its main symptom. The pattern of the disease can be acute (malignant) or chronic (benign). But the reports of this disease are all isolated cases of different types (9). The etiology of the disease has not been agreed upon (infectious, food poisoning, antibody reaction and toxemia). It is possible that the same disease may be caused by different agents, whereas in K'o-shan disease, it is a local disease, with a definite pattern, and has as its main symptom the non-vital areas. Therefore the disease is thought to be caused by one common agent.

B. The pattern and types of disease

In the past, the Japanese divided this disease into 3 types: (1) Acute type; the onset is sudden, terminating in death within a few days, or in acute weakening of heart; (2) Chronic type; a slow process that leads to hemorrhage

and weakening of the heart, and (3) Suppressed or recessive type; enlargement of heart, abnormal heart beat with change of impulse rate not noticed by the patient. Among the 103 cases in the northeastern area, 83 cases belong to Type 1 and 18 cases to Type 2, with two cases undetermined. In Type 1, death occured from two hours to one week after onset. In those cases, in addition to the non-vital area, there were large amounts of scar tissue. Some of these patients had been diagnosed as having Type 3 a year or so ago. Perhaps some tissue had already been damaged, or the damage had been mild, or the tissues had good repairative ability; thus the symptoms were not revealed earlier. In 18 cases of Type 2, some patients survived more than three years. The development of t e disease may be divided into two types: (1) Without the patient's knowledge, the "supressed" type with gradual loss of function and compensatory ability, finally chronic hemorrhage, weakening of the heart, and death. (2) The patient may survive one or more seizures, but eventually decompensation and death occur. The pathological changes in the acute and chronic types are the same, except that more scar tissue in present in the chronic type. Also, the heart becomes enlarged, with increased weight, and edema in the legs or abdominal cavity. In both acute or chronic types, the degree of pathology changes in the heart muscle varies greatly. Also, the manifestation of symptoms and the extent of pathological changes are not strictly parallel, indicating that the onset and development of the disease are related not only to the extent of cardiac damage, but also to the mechanical aspect of compensatory ability. Therefore, it is difficult to divide this disease into types according to the degree of cardiac involvement. It should be kep in mind that the three types of K'o-shan disease mentioned above are not three separate diseases, but rather different degrees of manifestation of symptoms.

C. The Etiology of K'o-shan Disease

As early as 1936, the Japanese suggested the theory of chronic carbon monoxide poisoning (10-12). But in reports of acute and chronic carbon monoxide poisoning, central nervous system involvement is quite prominent, with the appearance of pale white cells and bilateral softening mass (13,14) while in K'o-shan disease there are no such changes. From the biological point of view, the brain cells are far more sensitive to lack of oxygen than heart muscle, and in severe K'o-shan disease there is no evidence

that the brain cells are affected at all. Therefore it is difficult to explain this disease in terms of carbon monoxide poisoning.

Some literature has advanced the theory that the toxic products of streptococcus bacteria cause the destruction of smooth and striated muscle. (15-17) We have examined the pituitary gland, nasal membrane, salivary gland, bile sac and various parts of the digestive tract, and we have never found any evidence of this. Only a few cases had a mild pituitary infection while the stomachs did not have any symptoms of infection. Blood cultures from diseased hearts and /other/ organs have not disclosed any conclusive evidence of bacteria. Therefore there is no proof for this streptococcal infection theory either.

Some reports have also stated that toxemia may cause the idiosyncrasy of heart muscle infection. (18) Even though we have not encountered this phenomenon in our studies, we should not rule out the possibility of such a theory.

According to the process of non-vital areas changing to scar tissue, there was no evidence of chronic infection. Patients in the hospital had not undergone any antibiotic therapy, yet no new devitalized areas appeared. This indivates that the focus of infection may not originate in the heart. Because of the presence of both old and new pathological changes, it may be deducted that the cause of the disease can be recurrent. As far as the pattern of distribution of the changes in the heart is concerned, it is possible that the etiologic agent follows the blood into the smooth muscle. Since it has been found that even the fetal heart can be affected, although there is no direct interchange of blood between mother and fetus, it is also possible to deduce that the etiologic agent may seep out from the capillaries and into the muscle. As to the toxic agent, either from vital or non-vital sources, or whether there is any selectivity in invasion of albumen of muscle, or whether the heart damage is related to the metabolism of cardiac muscle, further studies must be done before any conclusions can be drawn.

Summary:

A. The Characteristics of K'o-shan Disease Based on Findings from 103 Cases in the Northeast and 36 Cases in the Northwest.

1. K'o-shan disease is a severe and spreading type of heart disease which affects the whole body. Its onset

can be acute or chronic, with weakening of the heart and termination in death. Besides the involvement of heart muscles, striated muscles may also be affected to a lesser degree. The nervous tissue of the heart is less affected than the muscles. Malnutrition is evident in cells of the central nervous system and the cervical nerves.

2. Certain common characteristics of the muscles: There are widely distributed areas of disappearing muscle cells, preceded by clumping of cytoplasm. At the same time there are both old and new scars.

The distribution of pathological changes is heaviest in "ju-tou" muscle, left heart ventricle and its valve, milder in the right ventricle and the auricles. There are four types of scar tissue, the net-like and branch-like scars being the most prominant. The breakdown of muscle is caused directly by the hardening of non-cellular substances into scars, without any stage of chronic infection. There is a definite correlation between the distribution of pathological changes and small blood vessels. The hearts of fetuses seven months or more old are involved but to a lesser degree than their mothers', and the pathological changes occur mostly in the right ventricles.

B. Analysis from the Point of view of Pathologic-anatomy

Because of the simulatneous presence of both old and fresh pathologic evidence, we know the disease can be recurrent. From the relationship between muscle pathology, small blood vessel distribution, and fetal heart involvement, it can be said that the disease is spread through the blood stream. From the phenomenon of changing from non-vital area to scar as a repairative process, we see no evidence of a live etiologic agent. Though there is a large possibility that the disease originates inside the body, the possibility of infection by outside agents should not be completely ruled out.

Fig. 1. Highly expanded left ventricle, flattened papillary muscle and "ju-tou" muscle. (A93, acute K'o-shan disease, 12 year old female)

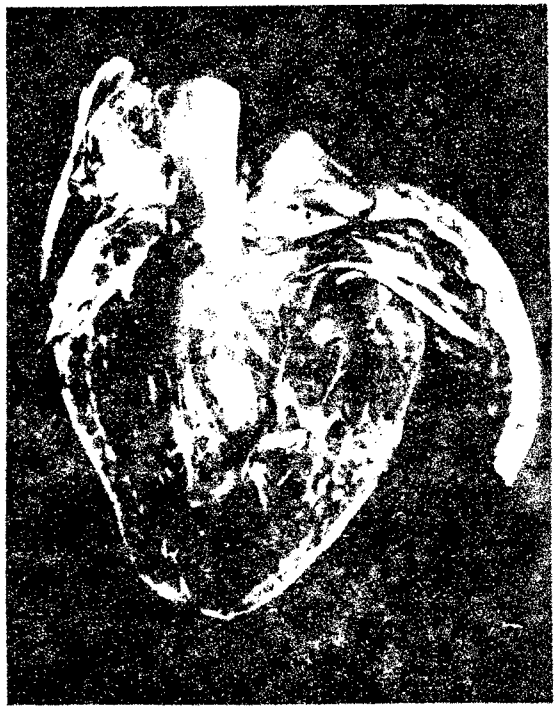

Fig. 2. Cut surface of heart with flower-like stain. Blood on papillary muscle. (108, chronic K'o-shan disease, 24 year old female)

Fig. 3. Left Ventricle showing inner layer; pathological changes of muscle and "ju-tou" muscle are more severe in inner layer. (A92, suppressed type of acute disease, 39 year old female)

Fig. 4. Posterior view of left ventricle; smooth muscle has become brownish with loss of normal texture. Under the microscope most of the muscle fibers are seen to have dissolved. (A94, acute K'oshan disease, 41 year old female)

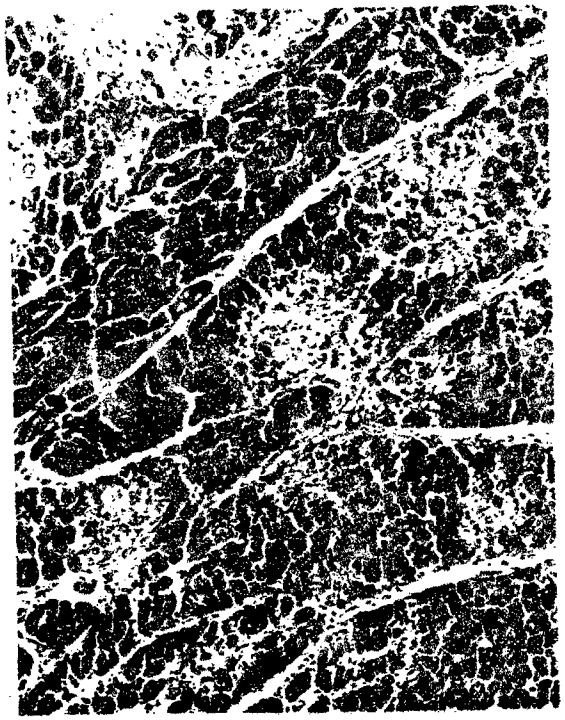

Fig. 5. Distribution of cardiac muscle with large area of dissolving fibers. (A93, acute, Su-mu-su-i stain, x 19.6)

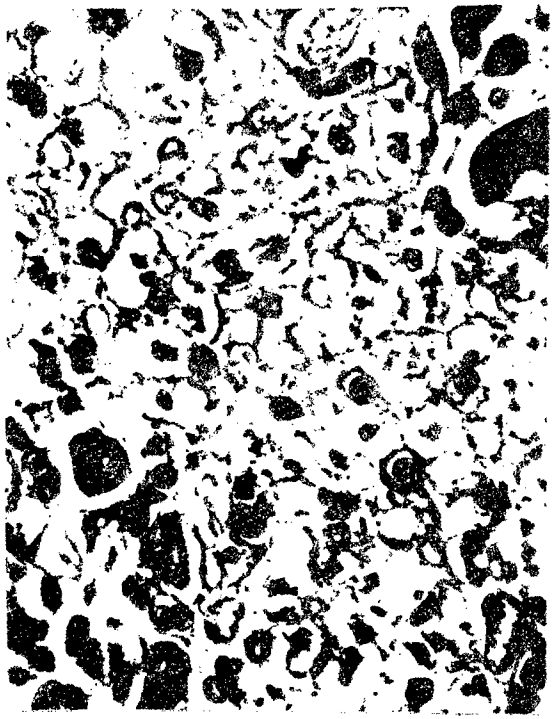

Fig. 6. Collapsing muscle membrane (C23, chronic, Su-mu-su-i stain, x 631)

Fig. 7. Dissolving muscle fiber, the remnants of muscle fiber frame-work after cytoplasm has dissolved. (A101, acute, Su-mu-su-1 stain, x 1282)

Fig. 8. Disintegration of muscle fibers, cytoplasm broke, membrane obliterated. (A94, Su-mu-su-1 stain, x 1282, acute.)

Fig. 9. Malnutrition of smooth muscle. (A87, acute, Su-mu-su-i stain, high power)

Fig. 10. Clumping of cytoplasm. (A88, acute, Su-mu-su-i stain, x 367)

Fig. 11. Distribution of small blood vessels along muscle bundles in "dissolving" type of disease. (C60, acute, Mallory's stain, low power)

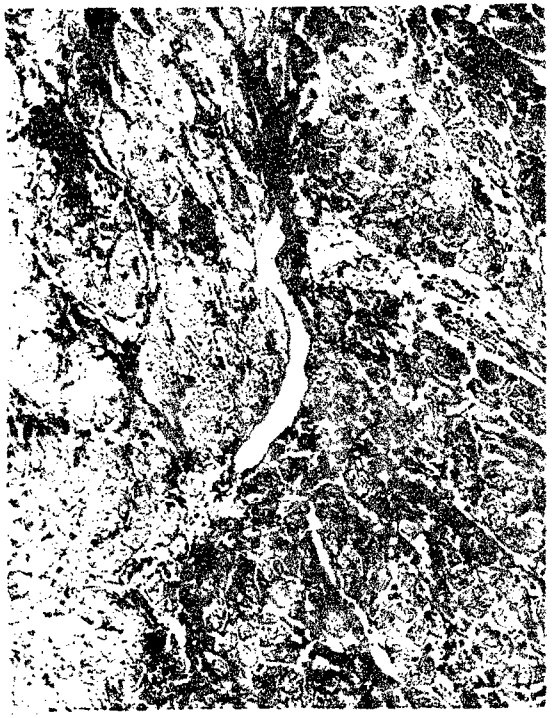

Fig. 12. Distribution of small blood vessels along scars. (A103, acute, van Gieson's stain, x 135)

Fig. 13. Dense scarring of smooth muscle. (A38, acute, Su-mu-su-1 stain, x 481).

Fig. 14. Net-like scarring. (A111, chronic, van Gieson's stain, x 114).

Fig. 15. Three large scar areas in "ju-tou" muscle with some normal fiber mixed in. (A86, chronic, van Gieson's stain, x 19.6.)

Fig. 16. Dissolving muscle of right ventricle of a 7 month old fetus. (Su-mu-su-i stain, x 510).

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CHINESE RESEARCH ON SHOCK AND ARTERIAL BLOOD AND FLUID TRANSFUSIONS DURING THE PAST DECADE

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The etiology, pattern, and treatment of shock, whether in animal experiments or case study of patients, have been the subjects of considerable discussion.

Shock is a common and recurrent type of condition. Its main manifestations are in the nervous system, especially the function and activity of the central nervous system. The blood circulation mechanism is changed. The capillaries of all organs and structures of the body dilated, with increased osmosis, and at the same time there is irregularity of the whole process of metabolism. Persons such as Shen Ko-fei (1), Tu Chuan-shu (6), Cheng Fu-min, (2) Yu Pei-li (4) have all discussed the pathology of shock.

Many causes contribute to the onset of shock (2). Such causes are large wounds, excessive bleeding, extreme temperatures (either hot or cold), excessive pain during operations, severe infection, or severe emotional stress. Sensitivity to certain drugs may also cause shock. Mr. Mai Kai (8,9), Ch'en Yi-hsin (10), K'ang Tsu-cheng (7), and Chai Yu-chung (22) have reported that shock results from intake of ch'ing-mei-so, lien-mei-so /some kind of molds/, sulfur /?/, novocain, etc. Other causes are intake of albumin (Ting Ting-wu (17)), repeated injection of the serum of its substitute.

The etiology of shock has been discussed at length. According to ShenKo-fei (2), Cheng Fu-min (1), Tu Chuan-shu (16), Yu Pei-li (4) and Ch'en Hua-ts'ui (5), there are the following viewpoints:

1. Toxemia: This theory was suggested by Dale and Cannon, who contend that shock is caused by toxic wastes of body structures. They based their theory on the observations that (1) when wounded lower limb was ligated, shock did not take place, but when the ligature was released, shock appeared, and (2) when the nerve distribution of lower limb were cut and /the limb/ wounded, shock still occurred. This happens because the break down of cells change into toxic wastes and get into the blood stream. They think that the wastes may cause paralytic dilation of the blood vessels, with increase of osmosis resulting in loss of fluid and consequently shock.

But the differing opinion of Mr. Cheng Fu-min (2) is as follows: (1) During shock, there is no increase of wastes in blood. (2) When a large quantity of wastes is injected into veins and lymph vessels at a localized obstructive area in a lower limb, there is no shock. But even after the obstruction is removed, shock is not produced immediately. (3) If one massages the wounded limbs, a temporary lowering of blood pressure occurs. This same phenomenon happens with massaging of the limbs before the wound is made. After the limb is wounded, this wounded area does not present the same picture as when the capillaries are over-filled and dilated. Therefore this theory is incomplete. Mr. Liu Chai-chu (3) suggested that application and subsequent removal of bandages might cause stimulation of the nervous system. This stimulation impulse relays itself into the central nervous system and causes irregular mechanism, especially the regulation of blood vessels causing obstruction and therefore shock.

2. Excessive loss of fluid: Mr. Shen Ko-fei (1) and Cheng Fu-min (2) mentioned that because of the loss of fluid, the /blood/ volume decreases and /the blood/ thickens, causing sluggish circulation with insufficient oxygen for the tissues. Because of this lack of oxygen, tissues begin to break down, producing wastes such as lactic acid and acetone bodies causing acidosis (Tu Chuan-shu (6)), dilation and osomotic capacity of blood vessels, with loss of fluid. Mr. Liu Chai-chou (3) pefformed animal experiments and found that the shock produced by simple loss of blood is milder than that produced by a combination of trauma with loss of blood or stimulation of nerve concurrent with blood loss.

3. Toxic Substance in Blood: Cheng Fu-min (2) mentioned that in the kidney, lack of oxygen may produce vaso-presso (VEM) /vasoexciter material/. Lack of oxygen in the liver /may/ produce vaso-depressor (VDM) /VDEM-vasodepressor material/. In the second stage of shock, there is a large amount of VDM, causing vessels to dilate and lowering blood pressure, with death of the patient. If there is bacterial infection, toxic substances from bacteria can dilate and paralyze the vessels.

4. Nerve Stimulation: (2) This theory is advanced by Pa-fu-lo-fu. He thinks that when inner and outer sense organs are stimulated, through the nerve conduction into the central trunk, and activate the nerves on an in "p'i-yun", they thereby increase the activity of circulation and res-

piratory system with increased secretion of hormones, as adrenal gland and kidney secreting adrenaline and vasopressors. But because of this over-stimulation, the "p'i-yun" soon produces the regulatory mechanism. Pa-fu-lo-fu called this "protective regulation".

When Mr. Yu Pei-li (4) discussed the problem of certain bone diseases and shock, he mentioned that continuous stimulation of "p'i-yun" leads to its weakening, with loss of regulatory mechanism, and causes it to become irregular. The circulatory, respiratory, autonomic nerves, digestive and excretory systems cause impedance of blood circulation, dilation of blood vessels, oxyten insufficiency and metabolic irregularity, with resulting acidosis and severe effect on the nerves and metabolism.

Secondary causes such as fear can also cause shock. (2) Mr. Ch'en Hua-ts'ui (5) proved experimentally that when alteration of nerve activity occurred, only a mild degree of shock, or no shock at all, was produced. For instance, if animals previously sensitized to shock-inducing stimulation are anesthetized or if they are under hibernation, and are given a definite amount of stimulation, no severe shock is produced.

From the data given above, one can see that the phenomenon of shock has been studied fully. The /structures/ most basically involved in shock are the nerve centers. Loss of body fluids, poisoning, and oxygen insufficiency result from the development of shock.

According to all the literature, most pathologists favor a combination of methods for preventing shock (1,2, 4,5). (1) Treat the wounded area locally, prevent pain stimulation by using novocain, and avoid excessive loss of body fluid. (2) Increased brain "p'i-yun" protective mechanism. (3) Stabilyze the blood fluid by using transfusion and heart and respiratory stimulants. (4) Mild sedation to decrease metabolism rate and thus decrease the threshold of oxygen needs. (5) Arterial transfusion of blood and fluid. Regarding arterial transfusion, we have done some exppriments on animals and observed patients.

Mr. Yang Chieh-chuan (13) reported saving two patients by arterial transfusion, as has Mr. Tan Yu (15) in his two cases. Mr. Cho Wei-chen (12) reported venous injection of a mixture alcohol concentrated /direct translation of characters/ glucose to combat shock. Of the 41 cases of shock patients, using arterial transfusion in a few cases, the result was clear. Within 10 minutes, blood pressure increased with strong heart response. Mr. Ho Lan-ch'ang (14) treated 41 shock patients (including bleeding, wounding, and surgical). Among those treated the bleeding and wound-

ing types of shock had the best result. Mr. Li Te-shou (19) reported 13 cases with severe shock, 13 of which had obvious good results. Mr. Chen Hsu-hsiao (21) reported 54 shock cases, in 41 of which the arterial transfusion method was used, and in 13 venous transfusion. In the group treated by arterial transfusion, 19 survived (46.3%), 12 with improvement (29.3%) and five with temporary improvement (12.2%). Of the venous transfusion group nine survived (69.2%), two with no improvement (15.4%) and two with temporary improvement (15.4%). Mr. Ho Kuang-yung (15) reported that all 14 patients treated with arterial transfusion had good results.

In amimal experiments, Ho Kuang-yung(15) used six dogs with hemorrhagic shock, and after arterial transfusion all had a good result. Li Hsiu-ch'in (16), Ting Ting-wu (17) and Wu Tai-chin (18) injected sodium lactic acid into dogs which were in artifically induced shock of the blood loss, trauma, and albumin loss types, Groups of eight to ten dogs were used; except in one or two instances, all had good results. Wang Yi-t'ang (20) used 50 percent glucose solution on dogs dying from loss of blood. Of the 36 dogs, all showed good results. If only "ma-huang-su" was used without the glucose, or with venous transfusion of glucose, the result was not satisfactory. Using dogs, Mr. Wu Tai-chin (23) made a comparison of arterial transfusion and venous transfusion to treat shock under low temperature, anesthetization and of the blood loss type. He proved that under these conditions, there is no difference between the types of transfusions.

Yang Chieh-ch'uan (13), Ch'en Hsu-hsial (21), Cho Wei-chen (22), Li Hsiu-ch'in (16) Ting Ting-wu (17) and Wu Tai-chin (18) discussed the different aspects of arterial transfusion:

1. Increased nutrition of heart muscle; When the blood pressure increases, the blood flow also increases. Under transfusion, the main valve closes, thus enabling blood and other fluid to flow into the coronary artery to correct the lack of blood in heart muscle. Due to the correction of this deficiency, the heart's function improves, with increased contractility. Therefore, with the pumping out of more blood, the blood pressure increases and condition of terminal blood vessels improves.
2. Increased quantity of blood in artery: The mixture of fluid from transfusion and blood pumped out by the heart activates a contracting reflex of the blood vessels, causing the volume of circulating blood to increase, blood pressure to increase, the formed elements in the blood to balance; the oxygen lack is corrected, normal metabolism established,

the tautness of blood vessel walls restored, osmosis of capillaries stopped, and loss of fluid decreased.
2. The full flow of blood in the arteries stimulates the autonomic nerves. Their reflexes cause the recovery of blood vessel tautness, with elevation of blood pressure and increase of blood flow.
4. Arterial transfusion, directly into the carotid artery, reaches the brain tissue, especially the medulla oblongata life center, vasocontrol center and respiratory center, causing their mechanisms to return quickly [? to normal], with subsequent recovery of normal blood pressure and respiration.
5. Stimulation of sense organs in blood vessel wall (pressure, chemical agents, moist heat, etc.) causes improvement in the functions of nerve centers through reflexes: in "p'i-yun" and under "p'i-yun" mechanism, especially in the medulla oblongata which made regulatory mechanisms of all systems recovered.

The above discussion is mostly concerned with arterial transfusion of blood and fluid. Other types of solutions such as glucose (20, sodium lactic acid (16,17,18), alcohol concentrated glucose solution (18), are all related to the chemical, nutritional, and detoxification reactions. When high pressure injection of the artery is done, the chemical stimulates not only the sense organs in the artery, but also other chemoreceptors. Other functions such as sodium lactic acid influence the acidbase balance to a large degree, and glucose not only stimulates the sense organ, but also is related to nutrition of this organ. Therefore, in addition to blood transfusion, fluid transfusion is also vital in treatment.

In conclusion within the last 10 years the use of transfusion in treatment of shock has increased. However, we should not stop there. Case studies and experiments must be carried out in the future.

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