

OTS: 61-31,600

JPRS: 4942

1 September 1961

RECENT NEWS FROM SOVIET THERAPEUTICS

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19981211 079

FOREWORD

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JPRS: 4942

CSO: 1914-S

RECENT NEWS FROM SOVIET THERAPEUTICS

Following is the translation of two articles in Terapevticheskiy arkhiv (Therapeutic Archive), Vol XXXIII, No 5, Moscow, 1961. Additional bibliographical information is given at the beginning of each article.7

TABLE OF CONTENTS

Page

Concerning the Efficacy of Bilateral Ligation of the Internal Mammary Artery in Chronic Coronary Insufficiency . .	1
More on "Malignant Hypertension" (An Answer to the Opponent) . . .	6

CONCERNING THE EFFICACY OF BILATERAL LIGATION OF THE
INTERNAL MAMMARY ARTERY IN CHRONIC CORONARY INSUFFICIENCY

[Following is the translation of an article by L. B. Shimeliovich, pp. 7-11.]

The efficacy of the bilateral ligation of the internal mammary artery because of insufficient coronary circulation, as suggested by Fiesci, has attracted attention for the simplicity of the technique, minimum trauma and very little counterindication (A. N. Bakulev, V. I. Kolesov, I. I. Neymark, B. K. Osipov, B. V. Petrovskiy).

Anatomically the operation was based upon the presence of anastomoses between proximal branches of the internal mammary artery and the myocardial vessels. The existence of anastomoses was confirmed by the experiment of introducing dye, fluorescein, and radioactive iodine into the internal mammary artery.

The ligation of the internal mammary artery in the second-third intercostal space caused a rise in the pressure and an increase of blood accumulation in the artery above the space of ligation and assisted in the uncovering and more effective filling of the anastomoses with blood, and this leads to improvement of the myocardial blood supply.

A. N. Bakulev states that the ligation of the internal mammary arteries under the conditions of an acute experiment lead to an increase of the retrograde coronary blood flow, but Hardin, Shumacker, Hurley, Eckstuy and Mozel do not agree. Blair observed the increase of the blood outflow from the coronary system only in chronic ligations of the internal mammary arteries (about 6 months previous to the experiment).

Griffin repudiates the possibility of a significant flow of the blood from the internal mammary artery to the myocardium. Battezzati, Glover, V. I. Kolesov acknowledge the protective role of the ligation of the mammary artery in dogs with the ligated descending frontal branch of the left coronary artery. Sabiston, Blalock, Vansant, Hurley, Hardin and Shumacker did not agree.

Hence the experimental data on the physiological justification of the operation were contradictory.

The clinical estimation of the efficacy as a rule is favorable. In 626 (94.7%) of the patients -- immediately after the operation -- stenocardia disappeared or became less pronounced; positive later results (observation from 2 months to several years) reached 440 (85.9%) out of 512 patients (A. N. Bakulev, V. I. Kolesov, I. I. Neymark, B. V. Petrovskiy, Battezzati, Glover, and others).

Other results were obtained by Fish, Crymes, Lovell, Ferrari, Fremont and Adams. In all, they operated upon 47 patients. An immediate pain relieving effect was noted in 85% of the cases; it lasted only a few days to three or four months. The authors doubted the effectiveness of the operation as a method of improving the supply of blood to the myocardium, and any connection between symptomatic post-operative improvement with the ligation of the internal mammary arteries.

The majority of the authors (Glover, B. V. Petrovskiy, I. I. Neymark and others) register dissociation between cessation of post-operative attacks of pain and the absence of the positive advances in the electrocardiogram. Cobb and Dimant operated upon 35 patients; in 21, internal mammary arteries were ligated, and in 14, only skin incisions were made. The results of the operations were estimated by a method of "double blind control," in which neither the patient nor the attending physician knew about the character of the operation. The authors came to the conclusion that the skin incision alone is as effective as the ligation. The anesthetic effect of the operation is considered psychological.

We analyzed the therapeutic results in 35 patients operated on in the Second Surgical Clinic of the Central Institute for Advanced Training for Physicians led by Professor B. K. Osipov.

The operation was conducted under a local anesthesia (0.25% solution of novocain) after preliminary injection of morphine. The incision of the skin was made 1 cm. outwards from the edge of the sternum and almost parallel to it on the level of the first-fourth costal interspace on both sides. The internal mammary artery was ligated in the second costal interspace with two ligatures and the artery was cut between.

The patients were from 46 to 70 years of age; the length of illness varies from 1 to 21 years. All the patients had a distinct pain syndrome of insufficient coronary circulation, which in one case was caused by syphilitic mesaortitis and in 34 cases, arteriosclerosis of the heart coronary arteries. All of the patients except eight had angina pectoris. In the past, 15 patients had myocardial infarct. Eighteen were hospitalized because of chronic coronary insufficiency (in 12 of them disease ran as a type of prodromic period of myocardial infarct, and they were operated on after the process was stabilized). In 11 patients coronary insufficiency ran in the background of hypertonic disease. In 30 patients insufficient blood supply to the myocardium was confirmed by the changes of the electrocardiogram at rest. Extrasystole was recorded in five patients; auricular flutter in three (paroxysmal in one); in one patient was a block in the left part in the His bundle. The operation was performed due to non-effective therapeutic treatment.

The same medical therapy was applied after the operation as was used before. In order to prevent the development of the postoperative pneumonia, penicillin was prescribed and respiratory exercise administered. To spare the patient's peace of mind in case of failure or unsuccessful operation, we explained to them the technique of the operation, its safety and effectiveness in about 70% of the cases. We tried

to put the prepared patients in contact with those who had undergone it easily.

In the predominant majority of the patients there were not gross changes of the electrocardiogram in standard deviations. During the operation in three standard deviations no prominent changes were revealed, except tachycardia. By registration CR₄-deviation in four of five patients found considerable growth of depression of the S-T interval and a lowering of the T prong. In 15 patients in the first days after the operation the pain syndrome of the coronary insufficiency was intensified. One patient developed myocardial infarct, and another paroxysmal flutter of the auricles. In one patient appeared a constant galloping rhythm and repeated acute circulatory insufficiency. In ten patients, according to the cardiogram, there was increased ischemia of the myocardium. Similar changes in the electrocardiogram was marked again in four patients, inspite of ceased stenocardiac attacks. Electrocardiographic signs of worsening myocardial supply of blood, as a rule, were not constant and levelled out in a few days. Therefore, in the first postoperative days in 19 out of 35 patients the blood supply of the myocardium grew worse, and in 14 it was confirmed by electrocardiogram.

Apparently such changes were not caused by the character of the operation, but by the consequences of combined emotional stress (coronary spasm, tachycardia and considerable fluctuation of arterial pressure). Tachycardia and the rise of arterial pressure immediately before or during the operation were noted in the majority of the patients; often they reached 150-200% in comparison with the preliminary condition.

After being discharged from the hospital, the patients were given examinations monthly during the first three months and afterwards every three months following the operation. Fourteen patients were observed over one-two years; 16 from one-half year to one, and three not less than three months. In 15 patients, after the operation, the attacks of pain ceased or became rare.

Male patient K., 50 years old. Prior to the operation for three years he suffered from an infarct of the posterior wall of the left ventricle. Afterwards he developed angina pectoris. He was hospitalized in the therapeutic department after prolonged attacks of posterior sternal pain. In the hospital the attacks of stenocardia were brought on by the slightest motion in bed, after food, and during defecation at night. Nitroglycerine action became considerably worse; often it was necessary to resort to the injection of narcotics. Attacks of pain lasted longer. The six weeks treatment with the use of anticoagulants did not bring any improvement.

After the operation pain ceased. Only on the third day while walking along the section of the hospital a paroxysm of pain arose in the wrist, which soon passed. In the following six weeks momentary pain developed under the right scapula and in the wrists while walking rapidly once or twice a week. The improvement which was given by the operation lasted only two months.

Slight improvement of the electrocardiogram was noted for six patients, but the pain syndrome subsided only in two. In five people the pain renewed after 14 days after the operation, in three after one or two months, in seven longer (inspite of the negative dynamics of the electrocardiogram in two of them).

In estimating the patients' condition in the longer period after the operation, it was shown that in those who were operated on when there was no aggravation, attacks of stenocardia remained upon a return to the preoperative load; and no sort of definite regular pattern of a change in the future condition was not shown. It reflected a natural wavering with change in acuteness and stabilization in the process of gradually progressing course of the disease.

The patients who were operated on during the acute stage of the disease (especially in the stabilized prodromal period of myocardial infarct), required subsequent continued treatment in the therapeutic hospital; long-range results were more favorable (in four patients over 1½ years separate attacks of angina pectoris appeared rarely). Such a result evidently is caused by the peculiarity of the progress of the disease, and not by the effect of the operation.

We did not find a cardiac insufficiency after the operation, as it was stated by Battezzati.

Four patients died from the basic disease in the interval of one month to a year after the operation. Our observations gave reason to doubt that the operation improved myocardial blood supply.

For solving the question on the etiology of the cessation of painful attacks after the operation in a number of patients, we find the following observations to be of importance. In 18 out of 23 patients, with initial poststernal localization of pain after the operation the pains either weaken or disappear, while in some of them the pains remained in the previous places of irradiation, or were displaced. Among 12 patients with an initial localized pain in the region of the apex of the heart, an analogical picture was noted only in one case. Our experience shows that many patients do not connect the "new" pain with the principal illness, and consider that the operation brought them the relief.

Zones of pain sensitivity were investigated in 27 patients. Zones of hyperesthesia appeared in 23, of which 17 were in the area of the sternum and on both sides of it. Hyperesthesia on the side of the skin incision disappeared or became hyposthesia. Evidently, the absence of the attacks of pain roughly depends on the influence of the operative incision (intersection of the anterior cutaneous branches of the intercostal nerves). The cessation or weakening of pain which was obtained in a number of cases is beneficial to the patients' state of health. However, pain is a symbol of anxiety, and the removal of pain but without any improvement in the blood supply to the myocardium given the chronic character of the disease scarcely seems expedient.

Conclusions

1. Bilateral ligation internal mammary arteries in patients with marked coronary insufficiency in blood circulation does not have a positive influence on the course of the disease.
2. The operation is not absolutely safe; in certain patients it was accompanied by an increase in coronary insufficiency.
3. The cessation or weakening of attacks of stenocardia in a number of patients after the operation, apparently was caused not by an improved supply of blood to the myocardium, but by interrupting the paths of pain sensibility.
4. Until an accurate experimental confirmation of the physiological justification of the operation can be obtained, the wide application in clinical practice is undesirable.

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MORE ON "MALIGNANT HYPERTENSION"
(An Answer to the Opponent)

Following is the translation of an article by N. I. Guseva, pp. 105-107.

The fourth issue of Terapevticheskiy Arkhiv for 1960 published a discussion of Professor Ts. A. Levina and a Candidate of Medical Sciences, L. A. Serebrina regarding our article "About Malignant Hypertension" printed in No 9 of this journal for 1958.

Unfortunately, in criticizing our principal position, they did not bring forward their own material, but refer in most cases to the data in literature.

The opponents misinterpreted our individual concept. Let us scrutinize their principal objections. Ts. A. Levina and L. A. Serebrina noted, that we allegedly wrote: "...there is not accepted a terminology "Hypertonia." But the topic was not about "Hypertonia" in general, but about the malignant hypertension.

Studying the native and foreign literature on this subject, we pointed out in our article, that "up until the present time there has been no unanimity in views on the essence of the malignant hypertension, moreover, there is not even a generally accepted terminology." It is incomprehensible, why the opponents mention only the opinions of those who do not recognize the existence of different forms of hypertensive disease (G. F. Lang, A. L. Myasnikov, and M. D. Tsinamzgvashvili). They do not mention the opinions of the clinicians and pathologists who hold an opposite attitude in this question (G. Yu. Yaveyn, Kiss, Wegner and Kernogew; M. D. Konchalovskiy, P. N. Nikolayev, A. I. Germawov and others).

T. A. Levina and L. A. Serebrina accused us in erroneously attributing to Ye. M. Tareyev the opinion of "malignant hypertension" as "an independent disease with a particular etiopathogenesis." In the monograph Gipertonicheskaya bolezn' /Hypertensive Illness' in 1948 he wrote "Malignant forms of hypertensive disease or Malignant Hypertonia, set apart by us and a number of other authors as an independent form, in our opinion deserves the much attention.

Such an opinion on the nature of malignant hypertonia was advanced by Ye. M. Tareyev in his works of later years. Ye. M. Myasnikov in his monograph Gipertonicheskaya bolezn' (1954) pointed out: "among the followers keeping malignant hypertonia in a special form is Ye. M. Tapeyev."

The opponents formulated our first concept thus: "Benign hypertensive disease never transfers into malignancy, but those cases where, in the course of it malignancy forming is observed are in fact a variation of renal disease."

Our version is as follows: "By studying our patients, we did not find a transition from benign hypertensive illness into malignant hypertonia. The authors, who admitted such a transfer, were usually guided by a sudden change for the worse in the patient's condition, by a disruption in the functions of vital organs, by the appearance of pathological elements in the urine, the disturbance of the concentrating capacity of the kidneys and an increase in residual nitrogen in the blood, and characteristic changes in the eye fundus. It seems to us, that in those cases the malignancy of the process was taken often as one of the variations of the clinical course of hypertensive illness, namely the renal type, which presents itself as one of the possible sources of the natural course of the pathological process which is characteristic for the benign form of hypertensive disease. Certainly, it could be stratification of malignant hypertonicity on the hypertonic disease, similar to the way subacute septic endocarditis is deposited on arthritis."

What kind of arguments do T. A. Levina and L. A. Serebrina advance against our position? They refer to the observations of a number of authors who witnessed that in the malignant course of the hypertensive illness, it was not infrequent that the changes of the cerebrum, cardiovascular system became more pronounced than the changes from the renal side. But did we deny it in our article? On the contrary, we pointed out: "... in malignant hypertonicity the serious changes develop early in the renal blood vessels, but in the first instance afflictions might develop in other vessels." Analyzing the complications in our patients, we pointed out that in 11 there were paralyzes and paresis as a result of hemorrhage into the brain; in 15 the appearance of cardiac insufficiency; and in 10, urimic symptoms.

The second concept ascribed to us by T. A. Levina and L. A. Serebrina: "The patient's age in malignant hypertonia was younger than in hypertonic patients and, therefore one could not speak of the transition from one form into another." This does not correspond to the true state of the matter.

Indeed, the patients with malignant hypertonia whom we observed were younger than the patients with the benign form (chiefly 25-38). About the second half of the opponents' phrase that in our article nothing was said about what it contains. Anyway, in the cause of transition of one form into another the age of the patients could not be an obstacle. It would be wrong to build any conclusion on it.

About our third concept, "The disease begins acutely (we said "as if acute") and it lasted on an average 2-4 years (in the article is written 2, 4 years)," T. A. Levina and L. A. Serebrina did not express any principal objections. They allude to G. F. Lang, who spoke about the latent period which could be more or less long, and on his observation of patients, whose illness in the beginning was manifested by

cardinal asthma, and loss of vision due to progressive angiospastic retinal disease. It is incomprehensible what grounds the opponents have to assume in this case a hypertonic illness which have been hidden for a long time. Our deductions were built on observations of 50 patients with malignant hypertension. "An overwhelming majority of the patients (41) during the appearance of the first symptoms of the disease were practically healthy, as the illness started somewhat acutely. Some of the patients named the month and even the day of the disease's beginning.

T. A. Levina and L. A. Serebrina wrote further that the complications and the cause of death (uremia, myocardial infarct, cardiovascular insufficiency) were considered by us as characteristic for malignant hypertension. Certainly in our article was revealed only the cause of death and complications, but nothing was said about their being specific or characteristic for malignant hypertension.

A highly important moment in the present discussion is the etiology of the malignant hypertension.

On the basis of our own material we are convinced that the neuro-psychic factor is not a leading one in the rise of malignant hypertension. We do not agree with the other authors who think that trauma of the nervous system plays a leading part in the development of malignant hypertension, and the psychic traumas in such cases appears more often than in benign. This question is elucidated in detail in our article. There is no contradiction that in patients with malignant hypertension, the neurasthenic syndrome appears early (as T. A. Levina and L. A. Serebrina think) as with such defects, the arteries of the whole system including the brain (as incidentally E. L. Gerber pointed out) it could not be otherwise. The neurasthenic syndrome is not a cause of the disease but a consequence.

In our article we brought out a view of A. I. Abrikosov (1953) about the nature of malignant hypertension as a unique disease which has as its cause an allergic condition of the arteries caused by sensibility with the participation of an unknown virus. In later works, A. I. Abrikosov adheres to the same point of view. In 1950 he wrote: "There is every reason to believe that the malignant nephrocirrhosis presents itself as a particular allergic illness;" in 1954: "Possibly, the malignant course of hypertonic disease is linked with elevated reactivity of the organism as is seen in the arteriosclerotic changes which develop not only in the kidneys, but in other organs (for instance in the brain)."

Characteristic peculiarities of this disease are young age, a rapid progressive course, a unique generalized affliction of the arteries, a rapid development of the functional insufficiency of several organs at the same time (heart, brain, kidney) leading to death in the course of a few months (up to two years). All of this makes A. I. Abrikosov's definition about the nature of the malignant hypertension fully probable.

Thus, the authors of the discussion article, T. A. Levina and L. A. Serebrina, by criticising our views of malignant hypertension as

of an independent disease, could not offer, feel, any substantial data in behalf of the other point of view, that is, on the unity in etiology and pathogenesis of the rapid and slow progressive forms of hypertonic illness.