

ON THE SIGNIFICANCE OF THE FUNCTIONAL STATE
OF THE LEFT AURICLE MYOCARDIUM IN THE PATHOGENESIS
OF PULMONARY HYPERTENSION IN MAN

A. A. Semenov

From the Institute of Experimental Biology and Medicine
(Director — Prof. E. N. Meshalkin) of the Siberian Division of the
Akademii Nauk SSSR, Novosibirsk

(Presented by Active Member of the Akad. Med. Nauk SSSR V. V. Parin)

Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 56, No. 7,
pp. 39-43, July, 1963

Original article submitted October 17, 1962

At the present time, in studying the pathogenesis of pulmonary hypertension associated with pathology of the mitral valve, sufficiently complete investigation has been carried out on the role of the contractile capacity of the right ventricle and the muscular apparatus of the arterioles and fine branchings of the pulmonary artery. Due to methodological difficulties, considerably less investigation has been carried out on the role of the muscular apparatus of the venules, the sphincters at the orifices of the pulmonary veins, and especially the contractile capacity of the left auricle myocardium [1, 2, 5, 7-12]. The importance of studying the rule of the latter is obvious. In the first stages of existing mitral stenosis, maintenance of normal hemodynamic indices, clinical compensation, and ultimately the work capacity of the patient, is guaranteed not by the entire heart, but basically by compensatory hyperfunction of the left auricle.

In this work we present data on the significance of the functional state of the left auricle myocardium in the pathogenesis of pulmonary hypertension.

METHOD OF THE INVESTIGATION

Pressure was measured by puncturing the great vessels and the cardiac cavity in the process of operating, immediately prior to a mitral commissurotomy. The recording system consisted of a thin needle, polyethylene tube filled with physiological saline containing heparin, an amplifier and an ink-writing oscillograph made by the firm of "Élem". The zero level corresponded to the position of the left auricle.

RESULTS OF THE INVESTIGATION

In analyzing the data obtained, it was rather important to consider the effect of the narcotic preparation, the depth of narcosis, and other factors related to the operative procedure, on the pressure level in the pulmonary circulation. Coworkers of our institute [3] have shown that during an operation for mitral stenosis the pressure in the pulmonary artery, according to a comparison of experimental data, is decreased by a rather constant figure of 20%. Such a regular, and essentially not so significant, reduction is explained, in the first place, by the stereotyped performance of this operative procedure, and in the second, by properly executed, controlled respiration associated with superficial narcosis.

We studied a total of more than 300 patients. Only 78 blood pressure curves were analyzed, obtained from patients with marked constriction of the mitral orifice, not exceeding 0.8-0.9 cm² (an average of 0.37-0.38 cm² for all three groups of patients analyzed below), and representing only cases of "pure" stenosis or stenosis with negligible mitral insufficiency.

According to the opinion of K. Uiggers [16], the energy of contraction of the left ventricle (E_{LV}) can be inferred from its blood pressure curve. We used the planimetric method of curve analysis proposed by this author, applying it to the curve for the pressure in the left auricle, which allowed us to obtain a series of valuable hemodynamic indices, described earlier [4], as well as such measurements as A_{1a} and $E_{exp \cdot 1a}$. The A_{1a} represents that portion of the area

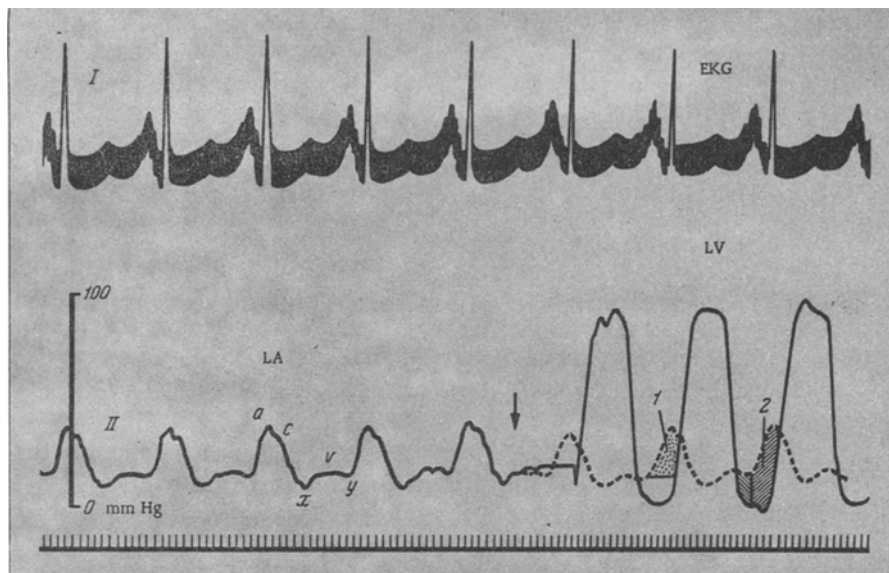


Fig. 1. First type of blood pressure curve in the left auricle, characteristic for "pure" and marked stenosis with a good, functional state of the left auricle myocardium. Patient B. Operation performed on January 1, 1961. Mitral orifice 6×6 mm. LV) pressure in the left ventricle; LA) pressure in the left auricle; \downarrow) moment at which record was changed. The dotted line represents the curve for the pressure in the left auricle superimposed on the curve of the left ventricle.

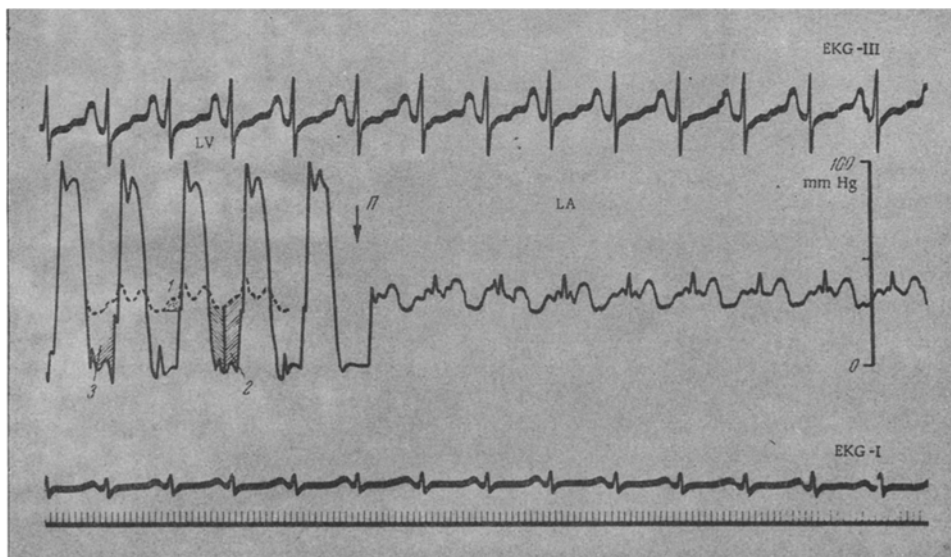


Fig. 2. Second type of blood pressure curve in the left auricle, characteristic for "pure" and marked stenosis. Patient S., 26 years old. Operation performed on November 24, 1960. Mitral orifice 10×5 mm. Symbols are the same as in Fig. 1. 3) Planimetric diastolic gradient (DGP).

of the planimetric diastolic gradient (DGP) in the auricular phase of ventricular filling (area 2 in Fig. 2). To a certain degree, this index offers a quantitative idea of when principal expulsion of the blood occurs — in the phase of rapid filling, or in the phase of auricular contraction. The $E_{exp.1a}$ represents the energy of expulsion developing in the left auricle (area 1 in Fig. 2). It should be noted that in the planimetry of this area we show the lower line horizontally, knowingly permitting a certain inaccuracy, since, actually, the pressure level in the left auricle caused by the flow of blood from the pulmonary veins is lowered, which can be clearly seen in auricular flutter (see Fig. 3).

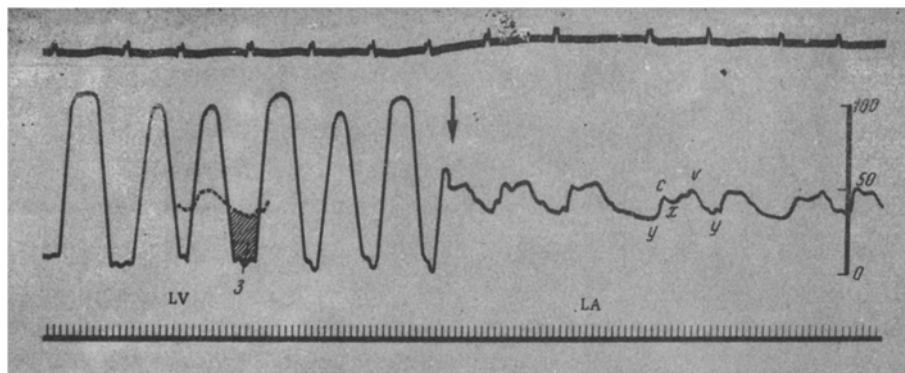


Fig. 3. Third type of blood pressure curve in the left auricle, characteristic for "pure" and marked stenosis. Patient Shch., 29 years old, with auricular flutter. Mitral orifice 8 x 6 mm. Symbols are the same as in Fig. 1.

The Level of Pulmonary Hypertension as Dependent Upon the Functional State of the Left Auricle Myocardium (Mean Data for Each Group)

Index	Group of patients		
	first	second	third
Number of patients	19	45	14
Rhythm	Sinus	Sinus	Flutter
Greatest diameter of the mitral orifice (in mm)	7.5 (from 4 to 10)	7.3 (from 5 to 10)	7.8 (from 4 to 10)
Approximate area of the mitral orifice (in cm ²)	0.38	0.37	0.38
Systolic pressure in the left ventricle (in mm Hg)	92.5 (from 74 to 105)	95.5 (from 79 to 120)	102 (from 82 to 124)
Blood pressure in the pulmonary vein (in mm Hg)	15* (from 7 to 22)	29 (from 17 to 46)	32.3 (from 24 to 40)
Blood pressure in the pulmonary artery (in mm Hg)	22.7* (from 13 to 30)	58.3 (from 33 to 96)	59.5 (from 33 to 97)
Pulmonary arterio-venous gradient (in mm Hg)	7.7* (from 4 to 16)	29.3 (from 8 to 59)	27.2 (from 9 to 40)
E _{exp.la} (in % of E _{lv})	6.6*	3.6	0
A _{la} (in % of DGP)	76* (from 50 to 100)	54.5 (from 22 to 83)	0

* Without patients N. and K.

According to the functional state of the left auricle myocardium, all patients were divided into three groups. The functional state was judged mainly from the clarity of the a wave, which, according to the generally accepted opinion, reflects systole in the left auricle [6, 14, 15]. The first group included patients with myocardium in a good functional state; the a wave was large, occasionally could be described as gigantic, and significantly exceeded the y wave (Fig. 1). Blood expulsion occurred essentially as a result of contraction of the auricle itself. The second group consisted of patients in which the contractile capacity of the left auricle myocardium was lowered; the a wave was equal to or smaller than the y wave (Fig. 2). In the third group were patients with complete functional incompetence of the left auricle (auricular fibrillation). Naturally, there was no a wave or auricular phase of ventricular filling (Fig. 3).

The left auricle was no longer a source of supplementary energy in propelling the blood, and, in essence, represented a simple continuation of the pulmonary veins.

It is apparent from the table that the systolic pressure in the left ventricle in patients of the first and second group was almost identical, while in patients of the third group it was somewhat higher. At the same time, the level of venous and arterial hypertension in the pulmonary circulation was different for all three groups. The pressure in the

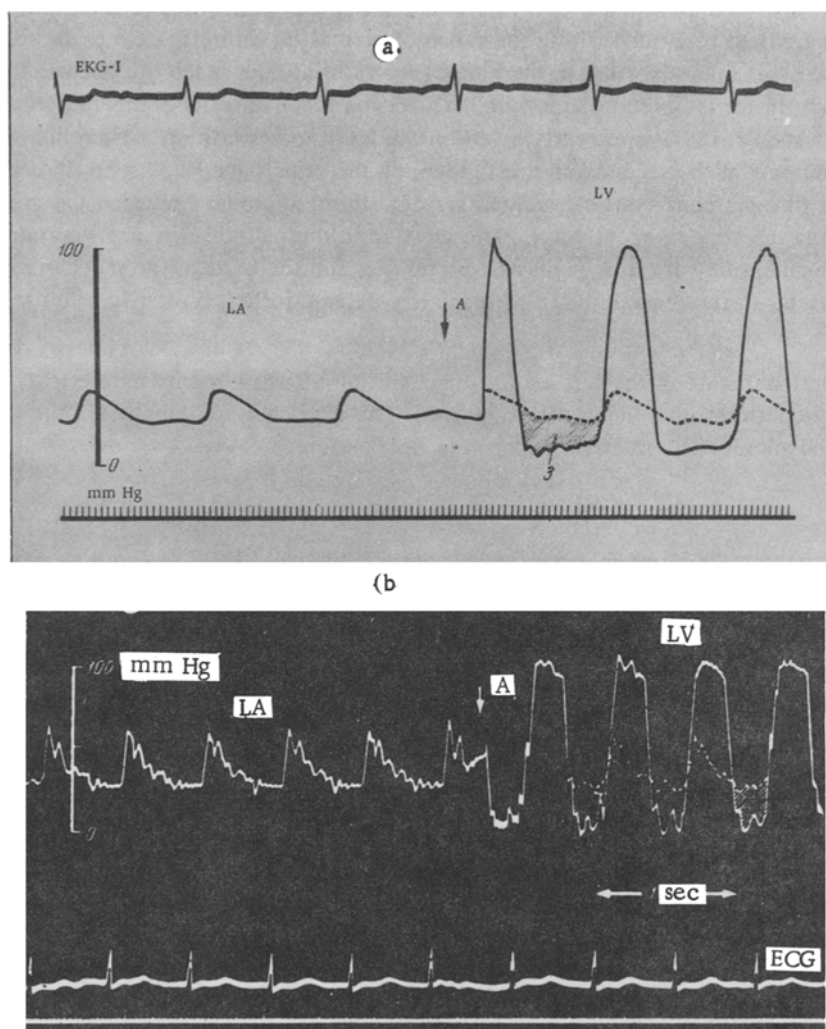


Fig. 4. Pressure curves of patient N., 22 years old, with "pure" stenosis. Operation performed on November 3, 1961. Mitral orifice 3×2 mm (a). Pressure curves of patient K., 19 years old, with "pure" stenosis. Operation performed on March 23, 1961. Mitral orifice 8×6 mm (b). Symbols are the same as in Fig. 1.

pulmonary veins of patients in the second and third groups was respectively 1.9 and 2.2 times greater than in patients in the first group, and the pressure in the pulmonary artery was respectively 2.6 and 2.7 times greater. The energy of expulsion developed in the left auricle was highest in patients of the first group, equalling 6.6%, while the level of hypertension, on the other hand, was greatest in patients of the third and second groups.

From here it follows that the level of pulmonary hypertension is closely connected, not with the degree of narrowing of the mitral orifice, but with the functional state of the left auricle myocardium. A decrease in the functional capacity of the myocardium is accompanied by an increase in the level of pulmonary hypertension. An exception was found in 2 patients, N. and K., in which, despite a good functional state of the left auricle myocardium, the pressure in the pulmonary artery was equal to 52 and 43 mm Hg respectively. This high pressure was caused, in our opinion, by the absence of hemodynamic effectiveness in the contraction of the left auricle. In both patients, contractions of the left auricle were completely superimposed on contractions of the left ventricle (Fig. 4). Thus, it would be more correct to say that the level of pulmonary hypertension is closely connected, not simply with the varying degree of the myocardial functional state in the left auricle, but with the varying hemodynamic effectiveness of its contractions.

In patients of the first group, despite a significant increase in the pulmonary pressure (by $1\frac{1}{2}$ times), the pulmonary arterio-venous gradient remained within the range of normal (7.7 mm Hg), i.e., the so-called second barrier was not present in these cases. The pressure in the pulmonary veins did not reach the oncotic level, and was equal to an average of 15 mm Hg for the group. Thus, the basis for the mechanism of compensatory increase in the work of the right ventricle in such cases is apparently a reflex which originates primarily from the left auricle and the pulmonary veins. In patients of the second and third group, on the other hand, the "second" barrier took place, and was clearly manifested (the pulmonary arterio-venous gradient in these groups exceeded the gradient in patients of the first group by 3.8 and 3.5 times respectively). The pressure in the pulmonary veins was quite high, and equal to the oncotic level. In connection with this, it may be postulated that the basis of the mechanism for forming the "second" barrier in patients of the second and third group is a defense reflex, primarily originating from the pulmonary capillaries.

Studying the functional state of the left auricle myocardium affords not only theoretical, but also practical, interest, particularly in constructing a functional diagnosis. The data obtained emphasize the total importance of restoring normal, hemodynamically effective work to the left auricle.

SUMMARY

An analysis was made of 78 blood pressure curves obtained by puncture of the cardiac cavities in patients with a marked constriction of the mitral orifice, not over 0.8-0.9 cm². Depending on the functional state of the myocardium of the left auricle the patients were subdivided into three groups. Data illustrating the reverse relationship between the functional state of myocardium of the left auricle and the hypertension level of the pulmonary circulation are presented.

LITERATURE CITED

1. V. S. Gurfinkel', L. L. Kapuller, and M. L. Shik, Byull. éksp. biol., No. 6, (1961) p. 14.
2. I. K. Esipova and A. D. Soboleva, in the book: The Surgical Treatment of Mitral Stenosis [in Russian]. Moscow, (1958) p. 10.
3. E. N. Meshalkin, I. N. Meshalkin, and M. L. Shik, Ter. arkh., No. 4, (1961) p. 18.
4. I. N. Meshalkin and A. A. Semenov, in the book: Data from the 4th Plenum of Pathophysiologists of Siberia and the Far East [in Russian]. Tomsk, (1962) p. 145.
5. A. A. Mikhnev and A. I. Khomazyuk, in the book: Questions in the Pathology of the Cardiovascular System [in Russian]. Kiev, (1959) p. 177.
6. Z. Belobrodek, M. Petrie, and Ya. Prokhaszka, Khirurgiya, No. 1, (1961) p. 29.
7. W. Evans and D. S. Short, Brit. Heart J., Vol. 19, (1957) p. 457.
8. B. Friedman, W. M. Daily, and R. H. Wilson, Circulat. Res., Vol. 4, (1956) p. 33.
9. J. Goodwin, Brit. J. Radiol. Vol. 31, (1958) p. 174.
10. D. F. J. Halrnagy, Die klinische Physiologie des kleinen Kreislaufs, Jena (1957).
11. D. Heath and W. Whitaker, Circulation, Vol. 14, (1956) p. 323.
12. Ch. J. McGaff, Clin. Med., Vol. 8, (1961) p. 455.
13. J. F. Martin, R. Froment, et al., Arch. Mal. Coeur, vol. 49, (1956) p. 134.
14. A. Morrow, E. Braunwald, et al., Circulation, Vol. 16, (1957) p. 399.
15. P. Mixon and O. Polis, Brit. Heart J., Vol. 24, (1962) p. 173.
16. K. Uiggers, The Dynamics of Blood Circulation [in Russian]. Moscow, (1957) p. 35.

All abbreviations of periodicals in the above bibliography are letter-by-letter transliterations of the abbreviations as given in the original Russian journal. Some or all of this periodical literature may well be available in English translation. A complete list of the cover-to-cover English translations appears at the back of this issue.
