SPHINCTERS OF THE PULMONARY VEINS IN MAN, AND THEIR SIGNIFICANCE

V. S. Gurfinkel', L. L. Kapuller, and M. L. Shik

Institute of Experimental Biology and Medicine of the Siberian Section of the AN SSSR (Director, Prof. E. N. Meshalkin), and 52nd Urban Clinical Hospital (Chief Surgeon, P. S. Petrushko)
(Presented by Active Member AMN SSSR V. V. Parin)
Translated from Byulleten' Éksperimental'noi Bioligii i Meditsiny, Vol. 51, No. 6, pp. 14-17, June, 1961
Original article submitted June 10, 1960

At present a great deal of intensive work is being carried out on the reflex regulation of the pulmonary circulation.

The immediate importance of such researches has increased on account of its application to the problem of pulmonary hypertension in thoracic surgery. The physiological significance of reflexes originating in the baroreceptors of the lesser circulation has been demonstrated several times [1, 3, 5]. An explanation of the significance of reflex regulation in pathology requires a knowledge of the hemodynamic conditions accompanying the pathological condition. Insufficient information is available, because until recently there was no method of studying this critically important problem.

A number of hemodynamic factors determine the condition of the vessels of the small circulation, and they include the contractile power of the right ventricle, the viscosity of the blood, the pressure in the left auricle, the functional condition of the valves of the pulmonary artery and of the sphincters of the pulmonary veins, which last factor plays a significant part. We developed this approach from studying results obtained by various authors and described by Halmagyi [4]. It has been shown that in patients with stenosis of the left atrioventricular valve, the resistance of the pulmonary vessels remains normal until the mean pulmonary capillary pressure exceeds 20 mm mercury. Not until higher pressures occur in the left auricle can changes in the pulmonary vessel occur which lead to an increase of their resistance to blood flow, which may be extremely important in some patients, and only moderate in others. Our results obtained by catheterization (23 patients), or by puncturing the vessels during operation (26 patients), completely support the results of previous investigators.

To determine whether the sphincters of the pulmonary veins play an essential part in initiating vascular changes in the lesser circulation, it was necessary to find whether they facilitate the unidirectional flow of blood from the left auricle while the pressure does not exceed 20 mm mercury, and whether they remain ineffectual at higher left auricular pressures. For this purpose it was necessary to compare curves showing the pressure in the left vein and in the left auricle in the same subjects for different values of left auricular pressure. The measurements could be made during operation, when they are necessary in order to assess the effectiveness of the mitral valvotomy.

METHOD

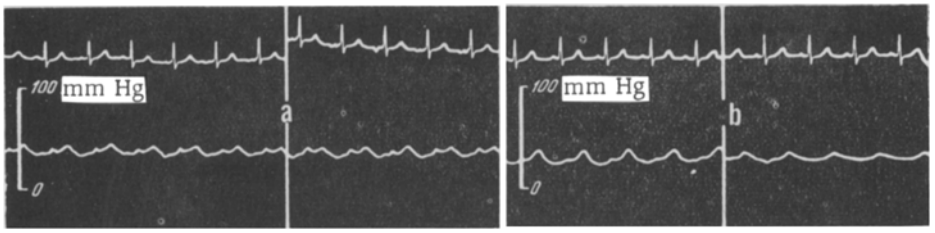
The needle introduced into the heart and vessels was connected to a polyethylene tube filled with physiological saline which in turn was connected to a rigid manometer. The recording system comprised an amplifier and an ink-writing oscillograph made by the firm "Elema." The measurements were made on four patients. The parallel histological study was made of the small pulmonary arteries and veins in the lingula of the left lung, which was removed during operation.

RESULTS

Figure 1 shows pressure curves recorded in the patient B. When the pressure in the left auricle was 45/33 mm of mercury, the curve for the pulmonary vein corresponded precisely to that for the left auricle, thus indicating failure of the sphincters of the pulmonary vein. The pressure in the pulmonary artery at this time was 63/42. After mitral valvotomy, the pressure in the auricle was reduced to 37/24 mm, and the oscillations of potential in the pulmonary vein became weaker than those in the left auricle. The pressure in the left ventricle remained unchanged at 76/8 mm. The resistance of the expanded left venous aperture became less than the resistance to the

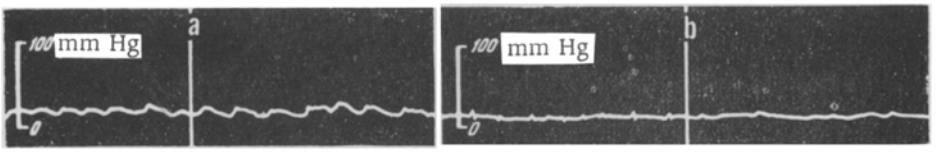
reverse flow of blood from the auricle offered by the sphincters of the pulmonary veins. The mean pressure in the left auricle had therefore to be reduced only to 30 mm in order for the sphincters to become effective again.

When there is fibrillation of the auricle, the sphincters may fail even at a mean left auricular of about 20 mm (Fig. 2, patient T).



Left auricle Pulmonary vein Left auricle Pulmonary vein

Fig. 1. Curves of the pressure in the left auricle and the pulmonary vein in the patient B during operation. a) Before mitral valvotomy; b) after the operation. Curves, from top down: electrocardiogram, pressure.



Pulmonary vein Left auricle Left auricle Pulmonary vein

Fig. 2. Curves of the pressure in the pulmonary vein and in the left auricle in the patient T with fibrillating arrhythmia during operation. a) Before mitral valvotomy; b) after operation.

The curve of the pressure in the pulmonary vein reproduces almost precisely the pressure curve in the left auricle. This effect is combined with an early increase in the resistance of the pulmonary vessels (pressure in the pulmonary artery in this patient was 51/27 mm). The sphincters again became effective after the operation, when the pressure in the left auricle was reduced to 13/6 mm. The pressure in the left ventricle remained at its previous level of 54/2 mm.

It was shown histologically that in the small pulmonary veins there was a marked hypertrophy of the muscular layer and a considerable constriction of the lumen of the vein (Fig. 3).

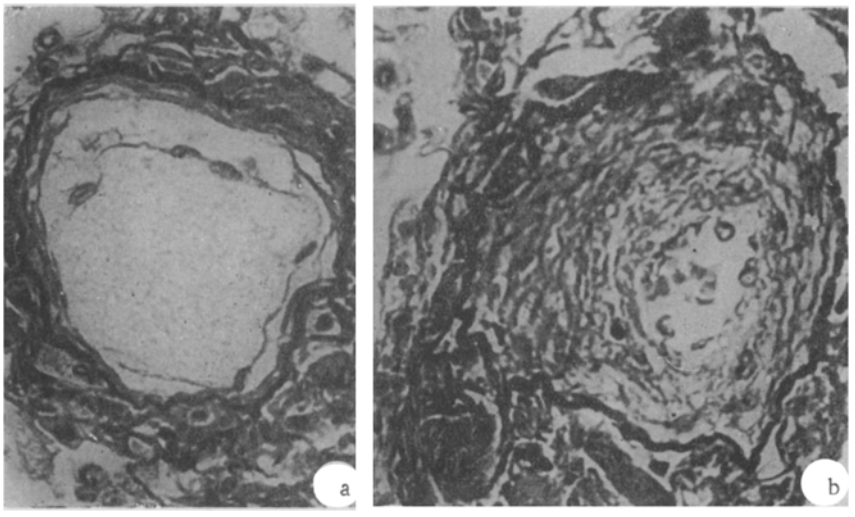


Fig. 3. Walls of the pulmonary veins: (a) a normal vein, and (b) a case of marked hypertrophy of the muscular layer and considerable constriction of the lumen. Stained in picrofuchsin and fuchselin. Magnification: objective 45 ×, ocular 6 ×.

These changes took place as a rule in cases where the mean pressure in the left auricle was above 20 mm mercury, and the sphincters of the pulmonary veins were ineffectual. In cases when the pressure in the left auricle was lower, the venous changes were weakly shown.

By preventing reverse blood flow, the sphincters of the pulmonary vein not only prevent dissipation of the work of the heart, but also protect the pulmonary capillaries from the reverse pressure wave. It would appear that failure of the sphincters, which occurs earlier in patients with auricular fibrillation, is also the factor which brings on constriction of the small intrapulmonary veins, which then take over the function of protecting the capillaries. The constriction of these veins also increases the resistance to the direct blood flow, which may lead to an increase in pressure in the pulmonary artery. The increase in pulse pressure in the latter probably leads to constriction of the pulmonary arterioles.

Auricular fibrillation, in addition to its other influences, is harmful because by putting the sphincters of the pulmonary veins out of action it brings about an early increase in the resistance of the pulmonary vessels. It is therefore very advisable to prevent auricular fibrillation during mitral valvotomy, as has been proposed by B. M. Tsukerman [2].

SUMMARY

Pressure curves in the pulmonary vein and in the left auricle were recorded in patients with stenosis of the left atrioventricular valve before and after mitral valvotomy. The curves showed that the sphincters of the pulmonary veins may fail when the mean pressure in the left auricle exceeds 20 mm Hg. Normally, by blocking the reverse flow, the sphincters protect the pulmonary vessels from the back pressure wave. With increased pressure in the left auricle, insufficiency of the pulmonary vein sphincters could provoke changes in the small pulmonary vessels, which were demonstrated histologically on biopsy material, and which had the effect of increasing the resistance to blood flow. Auricular fibrillation, by causing the sphincters to become ineffectual even when the hypertension in the left auricle is moderate, may promote a relatively early rise of pulmonary vascular resistance.

LITERATURE CITED

1. V. V. Parin, Proceedings of the Sverdlovsk Medical Institute and Sverdlovsk Scientific Research Institute of the Oblast Public Health Department [in Russian] (1941) Vol. 15, p. 3
2. B. M. Tsukerman, Abstracts of Reports on the Conference on Clinical Physiology [in Russian] (Moscow, 1959) p. 91.
3. D. M. Aviado and S. F. Schmidt, Physiol. Rev. (1955) 35, p. 247.
4. D. F. I. Halmagyi, Die klinische Physiologie des kleinen Kreislaufs (Jena, 1957).
5. H. Schwiegk, Arch. ges. Physiol. (1935) 236, 206.
