ESTIMATION OF RESISTANCE OF THE RESPIRATORY PASSAGES, DETERMINED BY WHOLE-BODY PLETHYSMOGRAPHY

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KEY WORDS: resistance of the respiratory passages; whole-body plethysmography.

The aerodynamic resistance (R) of the respiratory passages (RP) is virtually independent of the air flow ($\dot{V}$) within the range of its changes characteristic of quiet breathing [1]. The inspiratory (R_{in}) and expiratory (R_{ex}) resistances are virtually equal. As a reflection of these facts, the loop (R_{aw}), obtained under these conditions by recording simultaneously the chamber pressure (P_w) and $\dot{V}$, is linear and symmetrical (Fig. 1; technical aspects of analysis of R_{aw} loops, connected with conversion of the gradients of the loops into units of resistance, and their discussion, are omitted as having no bearing on the topic under investigation). In lung pathology, because of nonuniformity of impairment of patency of RP, not only is the gradient of the R_{aw} loops relative to the axis P_k increased, reflecting the increase in resistance, but a wide variety of forms of nonlinearity of the loops also is found, and is connected with predominance of one mechanism or another in the genesis of the disturbances [2]. As a result of long experience of clinicopathophysiological comparisons, the shape of the R_{aw} loop characteristic of disturbances of patency of RP through loss of the elastic properties of the lungs (emphysema of the lungs) is considered to be distinguished by a flask-shaped expansion in the region of expiration [1, 2]. This shape of the loop is considered to be associated with expiratory collapse of the small intrapulmonary RP, which is facilitated by the increased collapsibility of their walls as a result of loss of elastic structures (Fig. 2a).

The actual appearance of the R_{aw} loops observed in extensive emphysema of the lungs is sufficiently varied [3].

In the course of the study of the dynamics of R_{aw} in different phases of forced inspiratory and expiratory maneuvers, we obtained a loop for a healthy individual which was qualitatively similar in shape to a pathological loop (Fig. 2b) [4]. On analysis of the experimental data, we showed that the appearance of the "loop" on the P_k versus $\dot{V}$ curve during forced expiration by a healthy subject is connected with the development of expiratory constriction in this phase of the maneuver in the extrapulmonary, intrathoracic RP. Physically, this mechanism is due to compression of the intrathoracic part of the trachea (or the large extrapulmonary bronchi) when the pleural pressure is sufficiently high. The initial region of the R_{aw} loop of forced expiration (before reaching maximal flow) is analogous in shape to the loop during forced inspiration and is determined by the resistance

TABLE 1. Aerodynamic Resistance of Respiratory Passages (R, cm water·liter^{-1}·sec) Obtained by Different Methods of Analysis of Bronchial Resistance Loops

Patient	R_{In}	R_{Ex}	R_t	R_0	b_{In}	b_{Ex}
1	3,02	5,04	4,62	2,6	0,11	0,57
2	5,34	13,51	7,82	4,2	0,27	0,50
3	8,18	10,0	8,6	5,45	0,37	0,49
4	5,4	17,9	10,4	3,4	0,20	0,67
5	6,0	9,65	7,15	3,37	0,42	0,52

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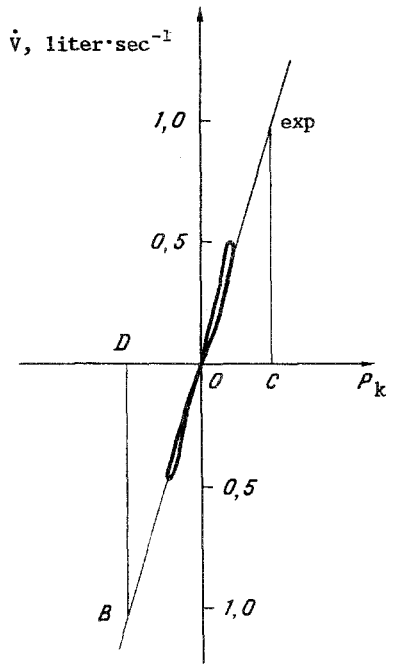

Fig. 1

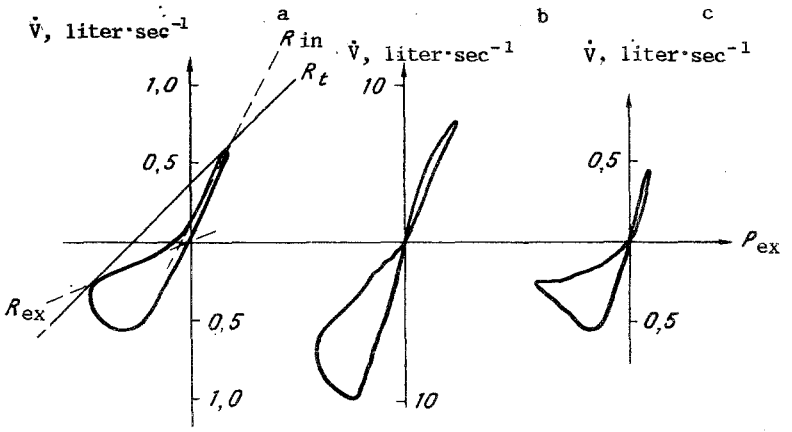

Fig. 2

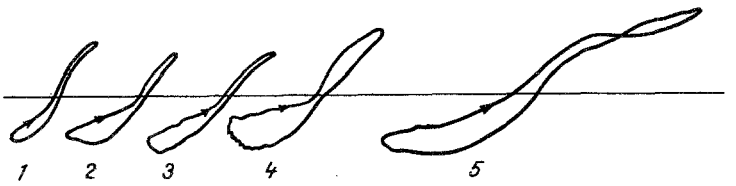

Fig. 3

Fig. 1. Aerodynamic resistance of the respiratory passages in a healthy subject.

Fig. 2. Dynamics of R_{aw} during disturbances of patency of RP.

Fig. 3. Five typical forms of R_{aw} loops recorded in patients with chronic obstructive bronchitis and emphysema of the lungs.

of all RP in the absence of expiratory stenosis. This resistance is nonlinear and depends on the alveolar pressure (the deformability of the lungs) during forced maneuvers, which give the P_k–$\dot{V}$ loop its S-shaped character for forced inspiration and the initial phase of expiration.

A R_{aw} loop similar in shape to that examined above also was found in some healthy subjects during phonation while breathing quietly, when there is a sharp decrease in width of the rima glottidis. During phonation the R_{aw} loop is suddenly widened, making it qualitatively very similar to that examined above (Fig. 2c). This is not surprising, for from the physical point of view, the processes of compression of the trachea during forced expiration and narrowing of the rima glottidis are the same.

It can be concluded from these arguments that the flask-shaped expansion of the R_{aw} loop during expiration in the course of quiet breathing by patients can be logically explained by the same mechanism of constriction of the lumen of the trachea (or the large extrapulmonary bronchi). The appearance of the shape of the loop we are considering in lung pathology may be connected either with an increase in compliance of the tracheal wall or with a redistribution of pressure within the trachea. In either case, the transmural pressure of the tracheal wall may become negative (or close to negative) at lower pleural pressures, and this is expressed as narrowing of the lumen of the trachea during expiration actually during quiet breathing.

Expiratory collapse of the small RP cannot be the cause of the appearance of the type of loop we are considering, for in that case there will be a more pronounced nonhomogeneity, due to the appearance of zones of "trapped air," and, as a result, widening of the loop in the region of "zero" flow. The shape of the R_{aw} loop under analysis differs in different patients in its gradient relative to the P_k axis and in the degree of expansion in the expiration region, with no expansion in the zone of "zero" flow.

The physical characteristics of patency of RP in the absence of expiratory constriction of the trachea (or of the extrapulmonary bronchi) is the value of R_{aw} corresponding to in-

spiration and the initial phase of expiration (until maximal flow). A characteristic feature of these parts of the loop in the cases under analysis is their nonlinear, S-shaped character, with a different gradient at the origin of coordinates. We showed previously [4] that nonlinear resistance at inspiration and the beginning of expiration is characterized by two parameters: $R_{aw} = R_0 + bP_A$. In this expression R_0 is determined by the gradient of the tangent to the R_{aw} loop at the origin of coordinates and reflects the resistance of undeformed RP. The parameter "b" reflects the change in resistance of the intrapulmonary RP during deformation of the lungs. This parameter is calculated as the ratio of the difference of resistance at the time of reaching maximal flow and R_0 to the alveolar pressure at that point. Its values can be calculated for inspiration and expiration. In the healthy subject the value of "b" is about 0.1 and the same for inspiration and expiration. The difference of "b" from this value reflects the increase in the degree of deformability of the patients' lungs, which may be due to loss of elasticity of the lungs.

Five examples of the most typical such forms of R_{aw} loops recorded in patients with chronic obstructive bronchitis and with emphysema of the lungs are given in Fig. 3. In all these patients bronchoscopy revealed dyskinesia of the trachea and large bronchi, whereas the elasticity of the lungs was greatly reduced, as shown by a decrease in the lung retraction index (the ratio of the maximal static elastic pressure to the total capacity of the lungs), which amounted to 1.8, 0.9, 1.2, 1.08, and 1.13 cm water·liter^{-1} for each patient (normally over 3.0).

Considering the arguments given above, the qualitative appearance of the R_{aw} loops leads to the conclusion that constriction of the lumen of the trachea takes place during quiet breathing in these patients, in the absence of any marked nonhomogeneity against the background of emphysema of the lungs. Hence it follows that the patency of RP in these subjects is characterized by the shape of the R_{aw} loop in the region of inspiration and beginning of expiration, and its widening reflects the dynamics of the lumen of a certain region of RP during expiration. Despite this, most existing textbooks recommend, in order to obtain the value of R_{aw}, that the loop be approximated, irrespective of whether linear or nonlinear, by a straight line taken through the maximal points of R_{aw} for the whole respiratory cycle or the phases of inspiration and expiration (Fig. 2a) 1, 2, and 5. In this way R_{aw} is calculated for the whole respiratory cycle (R_t), for inspiration (R_{in}), and expiration (R_{ex}). The question of the connection between values of "resistance" determined in this way and the physical value of R_{aw}, characterizing the patency of RP in patients with the forms of loop being analyzed, is not disucssed in the literature.

Table 1 gives values of R_{in}, R_{ex}, and R_t, calculated by the traditional method, and also values of R_0 and b for the patients under consideration.

Since the upper limit of the normal value of R_{aw} in healthy subjects is of the order of 3 cm water·liter^{-1}·sec, we can say that the patency of RP in patients Nos. 1 and 4 was changed only a little, whereas in the remaining patients the change was appreciable. Meanwhile, increased values of the parameter b were observed in all patients, which can be interpreted as an increase in the deformability of RP during the respiratory maneuver. Unlike in healthy subjects, the parameter b differed for inspiration and expiration, evidence of greater deformability of RP during expiration.

Incidentally, with the traditional approach to analysis, R_{aw} of patient No. 1 is apparently favorable (R_{aw} is not much greater than normally). However, the suggested method of analysis shows that although no appreciable change in the lumen of RP was present in this patient, as regards the degree of deformability of RP, he did not differ from the remaining patients.

Thus differences in the analysis of R_{aw} loops may lead to the obtaining of different parameters capable of being called the aerodynamic resistance of RP. Of them only the parameters R_0 and b demonstrate a certain physical meaning when the form of loops under analysis is present. In this case R_0 reflects the change in the patency profiles of RP, whereas b reflects the change in the deformability of RP (increased instability relative to external pressure, as a result of the pathological process of destruction of elastic structures).

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SELECTIVE ELECTRICAL INHIBITION OF C-FIBER SPIKES IN FINE NERVE STRANDS AS A METHOD OF IDENTIFYING A AND C AFFERENTS

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When the properties of receptor endings of an afferent fiber, present in a fine strand teased from a nerve, are studied, it is necessary to determine whether the fiber belongs to type A or to type C. If there are several active fibers in the strand, either the colliding impulses method [2] or electrical stimulation of the afferent fiber close to its ending in the tissues [3] can be used for this purpose. The use of the colliding impulses method requires matching of the times of excitation of the afferent endings and of electrical stimulation of the nerve trunk within a narrow time interval [2]. It can therefore be used in order to study mainly mechanoreceptors, whose time of excitation can be assigned reasonably accurately. The use of the second method requires a high voltage (hundreds of volts), in order to excite C afferents remaining undetected in the tissues [3]. Both these traditional methods are difficult to use in order to investigate afferent chemoreceptive endings in the tissues (tissue chemoreceptors) [1]. In this case it is virtually impossible either to localize the receptor endings precisely or to match the times of excitation of the receptor and of electrical stimulation of the nerve trunk.

In this paper we suggest a simple method of identifying the type of nerve fibers, based on selective inhibition of C-afferent spike generation in a fine bundle of nerve fibers containing A afferents also.

EXPERIMENTAL METHOD

Two regions of the saphenous nerve were isolated in 30 cats anesthetized with chloralose (40 mg/kg) and urethane (600 mg/kg): at the level of the knee, where the nerve was placed on a bipolar stimulating electrode, and in the proximal third of the thigh, where fine strands of fibers were teased from the nerve (Fig. 1). The nerve was tightly ligated proximally. At the site of teasing of the strands the nerve was placed on a platform with grounding electrode and bathed with oxygenated Krebs–Henseleit solution under a layer of mineral oil. Strands containing 2-5 myelinated fibers, distinguishable under the microscope (magnification: 20-40), were isolated by teasing with dissection needles, placed on a polished platinum electrode applied from above, and lifted into the layer of oil. The electrode was connected either to a Park-113 low-interference amplifier (for recording) or to an ÉSU-2 electrical stimulator (to inhibit the fibers selectively). Signals recorded from the fibers were amplified in the 85-2000 Hz band and recorded on magnetic tape for subsequent computer analysis.

EXPERIMENTAL RESULTS

In response to stimulation of the nerve trunk with square pulses 1 msec in duration and 12-15 V in amplitude (3 times higher than the threshold of excitation of C nerve fibers) a

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