On The Respiratory Changes Of The Intrathoracic Pressure, Measured In The Mediastinum Posterior

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It is a well-known pathological fact that tuberculosis shows a decided preference for the apices of the lungs. This was often explained by the assumption that the apex is not participating fully in the respiratory movements, and that the diminished ventilation of the said locality is the cause of its predisposition for the disease. But as it is now an established teaching that an invasion of tubercle bacilli in the lungs is at the bottom of the disease, we should rather expect, that if the apex breathes less, the bacilli will have less chances of getting in there, and in consequence this part of the lungs, instead of being predisposed for the disease, should rather show a certain immunity against it. To overcome this obstacle A. Hanau\(^1\) undertook to modify the hypothesis, by assuming that the expiration is indeed less, whereas the inspiration is rather better in the apex than in the other parts of the lungs, therefore all corpuscular elements of microscopical dimensions, like the bacilli, dust etc., have a greater chance to get in the alveoli of the apex, while their chances to get out of these by the expiration is reduced. This hypothesis would cover indeed the pathological phenomenon satisfactorily. Unfortunately for this hypothesis, the normal breathing also has to be taken in consideration. What would be the fate of the apex, if the inspiration should considerably overbalance the expiration, if more air should get into the alveoli than could come out?

Instead of speculating upon the normal status from pathological facts, we should endeavour firstly to study the normal process in due

\(^1\) A. Hanau. *Zeitschrift für klinische Medizin*. Bd. xii.
manner by physiological methods. It is a matter pertaining to physiology to decide, if in normal breathing all parts of the lungs are equally sharing in the respiratory act. But there, in physiology, the point in question is even not raised to the rank of a problem. I failed to find in physiological literature, even a serious discussion of our question. In the face of the decided predilections, however, shown by certain parts of the lungs to certain pathological processes (Tuberculosis, Pneumoniosis, Emphysema, etc.) physiology cannot afford to ignore this problem, or to solve it with mere theoretical speculations like the application of the physical laws of elasticity on the lungs, i.e. that an elastic band shows equal distensions in all its parts\textsuperscript{1}.

As it is very difficult to find an exact method to ascertain directly the degree of the distensions of the apex, as compared with those of the other parts of the lungs during normal breathing, I turned my attention firstly to a phenomenon steadily accompanying those distensions; it is the change of the intrathoracic pressure coinciding with the inspiration and expiration. There are at present three different methods for determining the intrathoracic pressure. The first, and oldest method, and as it was shown by Heynzius\textsuperscript{2}, probably the most reliable one, is that which was introduced by Donders\textsuperscript{3} and Hutchinson\textsuperscript{4}: to connect a manometer with the trachea of a dead animal and to open both the pleurae. We could of course not employ this method, which informs us only of the sum of negative pressure prevailing in the whole pleural cavity while the respiratory muscles are entirely at rest. Another method which seemed to be more appropriate for our purpose, is to connect a manometer carefully direct with the pleural cavity as it was practised by Fredericq\textsuperscript{5}, Bernstein\textsuperscript{6}, Weil\textsuperscript{7} and others. For my purpose, I should have to connect two manometers at different levels of the chest wall of a living animal. I have tried it; but soon convinced myself that, just as Heynzius supposed it to be, we never get by this method the exact intrathoracic pressure as it prevails in a normal condition. By removing carefully all the muscular tissue in one or two intercostal spaces, I could observe through intact pleura the motions of

\textsuperscript{3} *Zeitschrift für rat. Medicin.* Bd. iii.
\textsuperscript{4} Hutchinson in Todd's *Encyclopaedia of Anatomy and Physiology.*
\textsuperscript{5} Fredericq. *Archiv de Biologie.* 1882.
\textsuperscript{7} Weil. *Archiv für Klinische Medicin.* Bd. xxviii.
the lungs, and thus could control the state of the lungs. In this way I found out that every attempt of mine to connect the pleural cavity with a manometer was met with some degree of retraction of the lung, which if not for the direct observation, does not manifest itself otherwise.

The third method, which was introduced by Luciani and I. Rosenthal, and which seemed to be indeed the most appropriate for my purpose, is to measure the intrathoracic pressure in the thoracic part of the oesophagus. This can be done in a living animal without further preparation, by introducing a tube (katheter) through the mouth into the oesophagus. The tube has to be of some thickness to fill out the lumen of the oesophagus, but then on one hand it interferes with the breathing, and on the other hand it makes the oesophagus contract, which of course alters the pressure within it. I therefore made an incision in the oesophagus, pushed in through the opening a small katheter, and tied the oesophagus and katheter with a rubber thread, so as to have the oesophagus air-tight around the katheter and at the same time to be still able to push the katheter up and down within the oesophagus. This was done in three rabbits. I shall briefly state that I could not discover any constant difference in the negative pressure between the upper and lower parts of the oesophagus. At the same time these few experiments were sufficient to convince me of the correctness of the statement made by Heynsius, that the pressure within the oesophagus does not correspond to the exact pressure of the pleural cavity, the pressure in the oesophagus being, as it was proven by Heynsius, not only considerably less, but also inconstant, and consequently unappropriate to throw light on our subject. The thickness of the oesophagus wall, the muscular tissue and its irregular contraction provoked by the presence of a foreign body (katheter) are probably the cause, as Heynsius points out, of the reduction of the pressure and its irregularity. I may add, that every act of deglutition, which occurs quite often on account of the moving of the katheter, changes the pressure within the oesophagus considerably, the change lasting for some time.

Looking out for a new method by which I could compare the intrathoracic pressure at different levels of the thorax, my attention was turned to the mediastinum posterior, just by the last-mentioned experiments made on the oesophagus. The only objectionable features

1 Luciani. Archivi per le scienze medichi. 1875.
3 loc. cit.