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Conscious Volunteers Developed Hypoxemia and Pulmonary Collapse When Breathing Air and Oxygen at Reduced Lung Volume

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Hypoxaemia and Atelectasis Produced by Forced Expiration. By John F. Nunn, Anthony J. Coleman, T. Sachithanandan, Norman A. Bergman, and John W. Laws. Br J Anaesth 1965; 37:3–12. Reprinted with permission.

Ventilation at maximal voluntary reduction of lung volume caused significant desaturation in some healthy subjects breathing air. Saturation rapidly returned to control levels when normal lung volume was regained. These changes are probably due to reversible airway obstruction. During the inhalation of oxygen, ventilation at maximal voluntary reduction of lung volume caused, in one subject, a reduction of arterial P_{O_2} of 243 mmHg. Normal arterial P_{O_2} was not immediately restored on regaining normal lung volume. Chest radiographs showed extensive atelectasis, which persisted for several hours in an ambulant subject. These changes are probably due to absorption of oxygen from alveoli beyond obstructed airways. Reduction of lung volume may be harmful for patients who are breathing oxygen. Caution is therefore necessary in the use of a subatmospheric pressure phase during artificial ventilation and during suction of the tracheobronchial tree.

THE study cited above[1] was a natural sequel to my interest in understanding the factors that influence arterial oxygen tension during anesthesia. In 1964, my coworkers and I wondered whether, during anesthesia, the interference with oxygenation in the lung[2] was explained simply by the reduction of lung volume,[3] or whether it was caused by some other unknown effect attributable to the anesthetic. A photograph is available on the ANESTHESIOLOGY Web site at http://www.anesthesiology.org. It seemed likely that, at very low lung volumes, appreciable areas of lung would suffer total airway obstruction with shunting and reduction of arterial P_{O_2}. This was, in fact, known to occur during breathing with subatmospheric airway pressure or positive extrathoracic pressure, but it was unclear whether similar changes occurred in healthy subjects voluntarily breathing at reduced lung volume. Those days were before the introduction of ethical committees, and it was quite common for investigators to perform experiments on one another that, today, would seriously worry an ethical committee.

In 1964, a small international group of research fellows in the Research Department of Anesthetics at the Royal College of Surgeons of England, in London, set out to determine whether total airway obstruction with shunting and reduction of arterial P_{O_2} would occur in healthy subjects voluntarily breathing at reduced lung volume. Anthony J. Coleman, M.B., B.S., F.F.A.R.C.S., was a Medical Research Council research fellow at The Royal College of Surgeons. This was shortly before his departure for South Africa, where he later became Professor of Anaesthesia in Durban. T. Sachithanandan, M.B., Ch.B., was on study leave from Malaysia, and Norman A. Bergman, M.D. was on sabbatical leave at The Royal College of Surgeons from the Veterans' Administration, Salt Lake City, Utah. Dr. Bergman later became Professor of Anesthesiology at the University of Oregon in Portland. I was about to become Professor of Anaesthesia at the University of Leeds, Leeds, United Kingdom, in 1964. Twelve subjects were recruited, including Dr. Coleman, Dr. Bergman, David Jones (our technician at The Royal College of Surgeons), and myself. The age range was 19–56 yr; the senior volunteer was a distinguished Professor of Anaesthesia from Scotland who just happened to make a brief social visit to the Department and was instantly recruited.

After a control period, the subjects, while breathing air, voluntarily reduced their end-expiratory lung volumes as close as possible to residual volume for a period of 3–10 min. This was made easier by watching their respiratory trace derived from a box–bag spirometer. They were also required to maintain a constant end-expiratory P_{CO_2}, which was not difficult. Changes in saturation were recorded with a Waters ear oximeter, calibrated by rebreathing air to cause hypoxemia.

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There were no changes in saturation in the four subjects aged 24 yr or younger. However, all eight subjects older than 28 yr had decreases in saturation that were complete within a minute and were significantly related to their age. These changes ranged from 1.5–6.1% at a mean decrease in lung volume of 1.38 l. Four subjects showed greater decreases in saturation when breathing at reduced lung volume in the supine position. Saturations always returned to normal within 1 min of returning the end-expiratory lung volume to normal.

It crossed our minds that the desaturation at residual volume might be an artifact due to circulatory stagnation in the ear, caused by increased intrathoracic pressure. We dispelled this hypothesis by placing sphygmomanometer cuffs around the necks of three subjects and inflating them to 55 cm H_2O. The indicated saturation was unchanged, but I was surprised to find that my trachea was totally occluded! This question was finally settled when Dr. Bergman, then 37 yr old, volunteered (once again) for arterial puncture and recorded a decrease in arterial P_{O_2} from 103 to 65 mmHg at reduced lung volume, while the oximeter showed the saturation to decrease from 96 to 87%.

These results left us with little doubt that breathing at minimal lung volume resulted in airway occlusion, the extent of which was related to both age and posture. We therefore conjectured that there would be a danger of absorption atelectasis if the experiment were undertaken while the subject was breathing 100% oxygen. Dr. Bergman volunteered to breathe 100% oxygen at a residual volume for two periods of 3 min. During the first period at residual volume, his arterial P_{O_2} decreased from a control value of 676 to 560 mmHg and only slowly returned to 595 mmHg after 15 min at functional residual capacity. During the second period at residual volume, arterial P_{O_2} decreased to 433 mmHg (corresponding to a shunt of about 13%) and only returned to 580 mmHg 20 min after return to normal lung volume (corresponding to a shunt of about 5%).

The production of persistent absorption atelectasis by breathing oxygen at residual volume now seemed highly likely. We all believed that it would be useful to clinch this hypothesis by performing chest radiography. At 3:00 PM on March 17, 1964, I, then 38 yr old, breathed 100% oxygen at residual volume for two periods of 5 min. Since there were no facilities for radiography at The Royal College of Surgeons, St. Paul's Hospital, less than a quarter of a mile from the College, kindly took a radiograph 20 min after I had breathed oxygen at residual volume. This showed atelectasis of the basal segments of both lower lobes and partial atelectasis of the right middle lobe. Total lung capacity was estimated from the radiographs to be 3,860 ml. That evening, there was a meeting at the Royal Postgraduate Medical School, involving a walk of about a mile and a 30-min journey on the underground railway. A second radiograph at about 7:00 PM showed some reexpansion, but the medial segment of the right middle lobe and basal segments of the right lower lobe remained atelectatic. The radiologist, John Laws, who was a Consultant Radiologist at the Royal Postgraduate Medical School at Hammersmith in London, England, in 1964, now became interested and formally joined the study.

On March 18, 1964, I was due to lecture in Glasgow, Scotland, and took the night sleeper train from London to Glasgow. After my lecture, another chest radiograph showed almost complete reexpansion. A final radiograph, back in London on March 19th, showed both lungs fully expanded, and the estimated total lung capacity was 5,690 ml, essentially my normal value.

This study undoubtedly showed that age-dependent airway closure occurred while breathing at residual volume. This resulted in desaturation due to perfusion of unventilated alveoli. It was therefore not surprising that breathing oxygen at residual volume could result in radiologically detectable absorption atelectasis, which persisted for several hours. The atelectasis was not painful, but there was a curious "tearing" sensation in the chest when attempting to inspire to normal total lung capacity.

A later study[4] in six subjects (aged 38–54 yr) breathing 100% oxygen at minimal lung volume confirmed the essential findings. In addition, we were able to show a reduction in functional residual capacity in four of the six subjects, which was reversed by taking five maximal inspirations; this initially caused some substernal discomfort.

It was a privilege to be involved in clinical research in those days. Studies could be set up at short notice, as the inspiration arose. No ethical clearance was expected or required, and I do not recall that I ever made an application for a research grant in the last 35 yr of my career. We used the equipment that was on hand or that which we had made ourselves in the departmental workshops. Colleagues gave freely of their time to work on a project that appealed to them. Times have changed, and I look back with nostalgia to those golden years.

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