RES MEDICA
Journal of the Royal Medical Society

Measurement of Breath-holding time in the Acute Respiratory Patient

Donald F. Gardner

Abstract
In 1909, Douglas and Haldane (1) demonstrated the relationship of Pa02 and PaC02 to breath-holding time (BHT). Muxworthy (7) described the direct effect of initial lung volume on breath-holding time in 1951.

At the present, it is felt that breath-holding time is determined by, " . . . the interaction of a number of independent variables which can be classified in two major sets of stimuli: those related to lung volume and those derived from changes in gas tension and pH." (3)

Although a considerable body of knowledge has been acquired about breath-holding in a laboratory setting, very little work has been done to date on breath-holding in a clinical setting with respiratory patients. It was the object of the present study to determine the feasibility of measuring BHT's on acutely ill patients in a clinical situation.

ISSN: 2051-7580 (Online) ISSN: 0482-3206 (Print)
Res Medica is published by the Royal Medical Society, 5/5 Bristo Square, Edinburgh, EH8 9AL

Res Medica, Autumn 1973: 12-14
doi:10.2218/resmedica.v0i0.906

MEASUREMENT OF BREATH-HOLDING TIME IN THE ACUTE RESPIRATORY PATIENT

DONALD F. GARDNER

Introduction :

In 1909, Douglas and Haldane (1) demonstrated the relationship of Pa02 and PaC02 to breath-holding time (BHT). Muxworthy (7) described the direct effect of initial lung volume on breath-holding time in 1951.

At the present, it is felt that breath-holding time is determined by, " . . . the interaction of a number of independent variables which can be classified in two major sets of stimuli: those related to lung volume and those derived from changes in gas tension and pH." (3)

Although a considerable body of knowledge has been acquired about breath-holding in a laboratory setting, very little work has been done to date on breath-holding in a clinical setting with respiratory patients. It was the object of the present study to determine the feasibility of measuring BHT's on acutely ill patients in a clinical situation.

Another goal of this investigation was to study the hypothesis that breath-holding times would be longer in chronic bronchitics during periods of exacerbation than during periods of remission. This was felt to be true because of the decreased sensitivity to arterial CO2 manifested by these patients during periods of exacerbation. This was thought to be especially likely if any hypoxic stimulus could be eliminated by an inspiration to total lung capacity of pure O2 prior to breath-holding.

It was also felt that in chronic bronchitics during exacerbation that increased importance of the hypoxic stimulus relative to the hypercapnic stimulus would be reflected in a greater ratio of BHT on air to that on O2 as the patient improved; that is, breath-holding times on O2 would be relatively longer during exacerbation when the hypercapnic stimulus was relatively weak.

In addition to chronic bronchitics several other types of respiratory patient were incorporated into the study. These included patients with pneumonia and asthma, and one patient with spontaneous pneumothorax. They were all chosen because they shared the characteristic of an acute illness which would be expected to show a good response to therapy and improvement in pulmonary function.

Methods :

Breath-holding times were measured daily at approximately the same time for each patient. A total of four BHT's was done each time, two following an inspiration to total lung capacity of air, and two following a similar inspiration of pure O2. Patients were instructed to inspire maximally to total lung capacity from an unmarked rubber bag containing air or O2 and then to exhale to a spontaneously chosen lung volume most comfortable to them (an approximation of functional residual capacity). Timing was begun by an observer at this point and continued to breaking point. Nose clips were kept on throughout the procedure. Breath-holds on air were alternated with those on O2, and the order of gases was changed each day.

Peak expiratory flow rate (PEFR) was also measured daily at the same time as the breath-holds on a Wright Peak Expiratory Flow Meter.

Arterial blood gases were drawn on days one, seven, fourteen, and/or day of discharge in patients with abnormal blood gases, and more frequently if the clinical situation warranted. No attempt was made to draw blood at the exact time of breath-holding, only during the afternoon of the same day.

Results :

Breath-holding times were obtained on a total of twelve patients. Of these, five had exacerbations of chronic bronchitis, three had bacterial pneumonia, three had acute exacerbations of asthma, and one had spontaneous pneumothorax.

There is an excellent correlation of mean breath holding time after a breath of air to that after a breath of O2 in all patients (Fig. 1). Likewise, as has been demonstrated by other observers (5), mean BHT on O2 was significantly longer than on air in all but one patient (Jon).

No patient with chronic bronchitis showed a decreased breath-holding time on air or oxygen with improvement of clinical status or pulmonary function or length of hospitalization.

The author regrets that an adequate number of blood gas analyses as recommended by the protocol

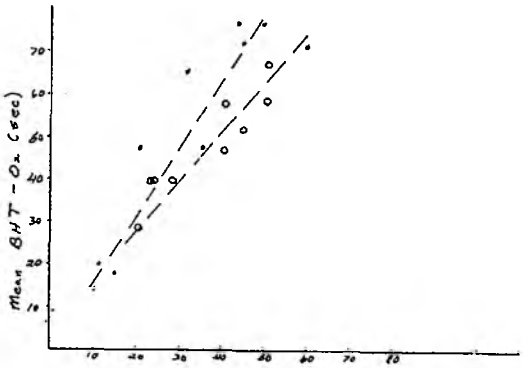

Fig. 1 MEAN BHT — Air

could not be made on all patients for technical and other reasons. In those patients in whom several blood gas analyses were made, no correlation between BHT and arterial blood gas concentrations drawn on the same day could be determined.

In three patients with chronic bronchitis who showed significant improvement in mean BHT, two, (Jon) and (Mau), had an increased PaCO2 drawn on the same day, and one, (Mur), was unchanged. Two, (Mur) and (Jon), had an increased PaO2 as would be expected, but one had a decreased PaCO2 (Mau). Likewise, in six other patients who showed significant improvement in mean BHT no correlation between mean BHT and arterial gas tensions could be determined.

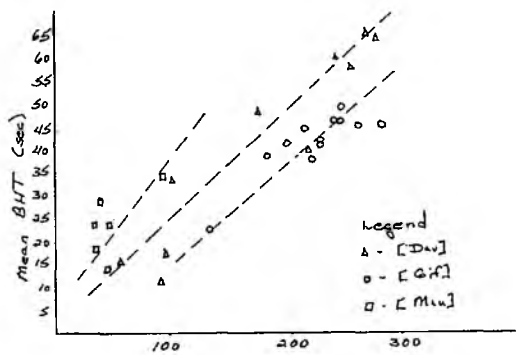

Fig. 2 PEFR (Litres/sec)

Peak expiratory flow rate was normal in three patients (>450 1/min). In three patients PEFR was greatly reduced (<150 1/min) and showed no improvement with therapy. The remaining six patients all demonstrated improvement in PEFR with treatment (ranging from 123 to 483%). In these patients PEFR tended to correlate with changes in MBHT (Fig. 2). In three patients (Dav), (Gif), and (Mau), the correlation appears highly significant, the correlation coefficients of PEFR plotted against mean BHT being .94, .79, and .72 respectively. In three other patients the correlation is less good, but still apparent, with r values of .43, .40, and .18 when PEFR is plotted against mean BHT.

No general correlation could be shown between mean BHT and length of hospitalization. In the six patients who demonstrated a correlation between PEFR and mean BHT, only one (Ser) showed a good correlation between mean BHT and days of hospitalization and three, (Gif), (Max), and (Jon), showed no correlation between these parameters. Of the other six patients in the study, two did show a correlation of mean BHT with length of hospitalization and four did not.

No correlation could be shown between the ratio of mean BHT on air to that on O2 (mean BHT-air/mean BHT-O2) and mean BHT. Two patients, (Dru) and (Mur), showed a dcrease in mean air/mean O2 as the mean BHT increased and one patient (Ess) showed an increase in mean air/mean O2 as mean BHT increased.

There was a failure rate of 19% due to the voluntary withdrawal of three patients from the experiment. One patient had to be withdrawn because of worsening clinical status.

Discussion :

This study has demonstrated that BHT can be measured in a practical way at the bedside and is of potential usefulness in the clinical setting. It must be recognised that breath holding does require a large degree of patient co-operation and is therefore excluded in the extremely ill, stuporous, or uncoperative patient.

Evidence has been obtained which suggests that there is a correlation of mean BHT on air and O2 to PEFR (and hence to airways conductance) in patients of all types who, (1) have airways obstruction upon admission, and (2) have significant change in airways resistance during therapy. Such a relationship might be explained by the length-tension inappropriateness hypothesis of Campbell for the etiology of dyspnea (4). More study is needed in this area with a larger number of selected patients.

Breathholding times in chronic bronchitics with elevated PaCO2's did not decrease with clinical improvement and no correlation between breath-holding times and arterial PO2 and PCO2 drawn on the same day could be demonstrated. Therefore, the original hypothesis that chronic bronchitics with elevated PaCO2 and a decreased sensitivity to arterial PCO2 would have longer breath holding times than during periods of remission could not be supported.

Since no correlation of the ratio of BHT's on air to those on oxygen could be shown with either mean BHT or PEFR, it could not be demonstrated that the hyercapnic stimulus to respiration became more important relative to the hypoxic stimulus with improvement in patient respiratory function.

The author is especially indebted to the ideas and help of Dr. G. J. R. McHardy of the pulmonary physiology section of the City Hospital, Edinburgh, and to the Medical Student Council of the University of Edinburgh for making the Edinburgh-Illinois student exchange possible.

Summary :

Breath-holding times were measured daily during an acute phase of illness in twelve patients with different types of respiratory disease. A direct correlation between airways conductance as measured by the peak expiratory flow rate (PEFR) and

mean breath-holding time was suggested. No relationship could be shown between breath-holding time and arterial blood gas concentrations taken on the same day. It was demonstrated that breath-holding time can be measured and may be useful in the clinical situation.

References

(1) *Douglas, C. G., and J. S. Haldane. The regulation of normal breathing. J. Physiology, London 38:420-440, 1909.*

(2) *Mithoefer, J. C., C. D. Stevens, H. W. Ryder, and J. McGuire. Lung volume restriction, hypoxia, and hypercapnia as interrelated respiratory stimuli in normal man. J. Appl. Physiol 5:797, 1953.*

(3) *Mithoeffer, J. C. Breath-holding. Handbook of Physiology, American Physiological Soc. Section III, vol. II 1011-1025, 1966.*

(4) *Campbell, E. J, M., and Howell, J. B. L. The sensation of breathlessness. Br. Med. Bull., 19, 36-40. 1963.*

(5) *Engel, G. L., E. B. Ferris, J. P. Webb, and C. D. Stevens. Voluntary breath-holding. II. The relation of the maximum time of breath-holding to oxygen tension of the inspired air. J. Clin. Invest. 25:729-733, 1946.*

(6) *Fowler, W. S. Breaking point of breath-holding. J. Appl. Physiol. 6:539-545, 1954.*

(7) *Muxworthy, J. F. Breath-holding studies: relationship to lung volume. Air Force Technical Report 6528, pp. 452-456. Ohio: Wright-Patterson Air Force Base, 1951.*

ITEM OF MEDICAL INTEREST

22d. To the Crown Tavern behind the Exchange, and there met the first meeting of Gresham College since the plague. Dr. Goddard did fill us with talk in defence of his and his fellow physicians going out of town in this plague time; saying that their particular patients were most gone out of town; and a great deal more etc. But what, among other fine discourse pleased me most was Sir G. Ent, about respiration; that it is not to this day known, or concluded upon among physicians, nor like to be done either, how the action is managed by nature, nor for what use it is.

23d. Good news beyond all expectation of the decrease of the plague, being now but 79, and the whole but 273. So home with comfort to bed. A most furious storm all night and morning.

From The Diary of Samuel Pepys. January 1666.

ABOUT THE AUTHORS

Heather Davis is a Research Associate in the Department of Pharmacology. She earned her Ph.D. in 1971 for a thesis entitled "The release of prostaglandins in vivo".

Neil Douglas is the Senior President of the Royal Medical Society.

Elizabeth Edmond is a medical graduate researching into the epidemiology of Infectious Mononucleosis in the Edinburgh student population.

Donald Gardner is a medical student at the University of Illinois.

Iain Palin is a medical student at Edinburgh University.

ANSWERS TO THERAPEUTICS PROBLEM

Yes, this question is relevant. Certain cooking oils contain methyl polysiloxane, a silicone that apparently interfers with the absorbtion of anti-coagulants. Similar compounds are used in some preparations prescribed for hiatus hernia. (E.G. "Crisp n'Dry" and "Asilone") Reference: Talbot, J. M. & Meade, B. W. LANCET (1971) i 1293.