CLINICAL EXPERIENCE WITH THE MANLEY MULTIVENT: ASSESSMENT OF THE EFFECT OF THE VENTILATOR ON CARDIOPULMONARY STABILITY IN PATIENTS UNDER BALANCED ANAESTHESIA.

BY

DR. MICHAEL STEPHEN MSELLE MD (DAR)

A DISSERTATION SUBMITTED IN PARTIAL FULFILLMENT OF THE REQUIREMENT FOR THE DEGREE OF MASTER OF MEDICINE IN ANAESTHESIOLOGY IN THE UNIVERSITY OF DAR-ES-SALAAM 1996.

I HAVE READ THE DISSERTATION AND APPROVE IT FOR EXAMINATION.

SUPERVISOR: DR. N. J. A. M. MATEKERE (M.B.Ch.B.,M.MED)
DEPARTMENT OF ANAESTHESIOLOGY
FACULTY OF MEDICINE.
UNIVERSITY OF DAR-ES-SALAAM.

MUHIMBILI MEDICAL UNIVERSITY COLLEGE
LIBRARY
DSM.

ABSTRACT

The effects of controlled ventilation by the Manley Multivent Ventilator on the cardiopulmonary stability in patients who underwent various surgical procedures under balanced anaesthesia, were studied in 102 patients of American Society of Anaesthesiologists (ASA) class 1 and 2. The general performance of the ventilator and complications arising from its use were also assessed.

Controlled ventilation by intermittent positive pressure ventilation (IPPV) was found to have no significant effect ($P > 0.10$) on the blood pressures of the patients. The pulse rate changes were also not significant ($P > 0.05$); and a good peripheral oxygen saturation of 96 to 100% was maintained in all patients. The Multivent performed satisfactorily to provide effective ventilation using maximum inspiratory pressures of 20 to 35cmH_2O, tidal volumes of 6.7 to 12.5mls/kg body wt., respiratory rate 12 or 14 cycles/min and I:E ratio of 1:2. These parameters were regulated and adjusted successfully according to individual patient requirements, and adequate and safe ventilation was achieved. The ventilator was simple to operate, versatile, reliable and safe for use during anaesthesia and no complication was associated with its use.

The Multivent with its unique features of flexibility in both power and gas supply, coupled with its low purchasing

and running costs, provides anaesthetist in the developing world with an economical, reliable and safe means of controlled ventilation.

TABLE OF CONTENTS

Title page

Abstract ii

List of Tables v

Acknowledgements vi

List of Abbreviations ix

Introduction 1

PART 1: LITERATURE REVIEW ON ARTIFICIAL VENTILATION

1.1 Introduction 4

1.2 History of Mechanical Ventilation 5

1.3 Physiological Aspects of Controlled Ventilation 7

1.4 Clinical Aspects of Controlled Ventilation 35

1.5 Ventilators and Mechanical Ventilatory Support 45

1.6 The Manley Multivent Ventilator 62

PART 2: CLINICAL STUDY 67

2.1 Introduction 67

2.2 Materials and Methodology 67

2.3 Results 72

2.4 Discussion 81

2.5 Conclusion 86

2.6 References 88

List of Tables

Page

1. Table 1: Age and sex distribution 75
2. Table 2: Procedure and ASA status distribution 75
3. Table 3: Peripheral oxygen saturation of patients during anaesthesia 76
4. Table 4: Tidal volumes (mls/kg) used in patients during anaesthesia 76
5. Table 5: Respiratory rates used in patients during anaesthesia 77
6. Table 6: Inflation pressures (cmH2O) used during anaesthesia 77
7. Table 7: Statistical analysis of pulse rate changes 78
8. Table 8: Statistical analysis of the sytolic blood pressure changes 78
9. Table 9: Statistical analysis of the diastolic blood pressure changes 79
10. Table 10: Operation of the ventilator 79
11. Table 11: Complications arising perioperatively. 80

ACKNOWLEDGEMENTS:

I should like to express my sincere gratitudes to my teachers in the Department of Anaesthesiology, Muhimbili Medical Centre for their continued effort in introducing me to the art of Anaesthesia. I should particularly like to thank Dr. N.J.A.M. Matekere for his constant encouragement and supervision during the clinical study. Special thanks go to Penlon U.K. Ltd. for donating the Manley Multivent, and Sequira Hospital Monitors Ltd. for donating the Novametrix Pulseoximeter, both of which made the study possible.

My appreciation is also extended to the surgeons and theatre personnel who in one way or another contributed to the success of the study. I am also very thankful to Mr. Makwaya who arranged for the statistical data analysis.
I wish to express my thanks to Dr. V.V.G. Mwafongo, Dr. F.W. Mbanga and Dr. H. Lweno for the corrections of the manuscript.

Finally I extend my thanks to Mrs Hafsa Matimbwa for typing the dissertation.

Dr. Michael S. Mselle.

1996

DECLARATION:

I hereby declare that this dissertation is my own original work, and it has not been submitted for a similar degree in any other University.

Date: 29.10.96

Signature: JMMbelle.

COPYRIGHT

All rights reserved. No part of this work may be reproduced, stored in any retrieval system or transmitted in any form or by any means, electronic, mechanical, photocopying, recording or otherwise, without prior written permission of the author or the University of Dar es Salaam in that behalf.

List of Abbreviations

ASA	-	American Society of Anesthesiologists
CPAP	-	Continuous Positive Airway Pressure
CVP	-	Central Venous Pressure
EMO	-	Epstein Macintosh Oxford
FIO_2	-	Fraction Inspired Oxygen Concentration
FRC	-	Functional Residual Capacity
HFJV	-	High Frequency Jet Ventilation
HFPPV	-	High Frequency Positive Pressure Ventilation
HFO	-	High Frequency Oscillation
HFV	-	High Frequency Ventilation
ICU	-	Intensive Care Unit
IMV	-	Intermittent Mandatory Ventilation
IPPV	-	Intermittent Positive Pressure Ventilation
kPa	-	Kilo - Pascals
MMV	-	Mandatory Minute Ventilation
OMV	-	Oxford Miniature Vaporizer
PCO_2	-	Partial pressure of Carbon dioxide
PO_2	-	Partial pressure of Oxygen
Psi	-	Pounds per Square inch
PCWP	-	Pulmonary Capillary Wedge Pressure
PEEP	-	Positive End - Expiratory Pressure.

INTRODUCTION

Anaesthesia in developing countries like Tanzania is faced with major problems of shortage of qualified personnel and anaesthetic equipment. The art of anaesthesia ranges from the 18th century with Humphrey Davy's inhaler of 1779 (Seeley H.F. 1984) to the modern world of electronics where most of the job is done or assisted by machines. Over the years there has been many developments in the field of anaesthesiology involving equipments; techniques; and drugs, which have brought with them increases in costs; complexity; and demand for highly trained personnel for their use.

One of the most striking advances in anaesthesiology is the development of skills of attaining proper ventilation of patient's lungs by means of controlled or artificial ventilation (Mushin, W.W. et al 1980) which can be achieved also by mechanical lung ventilators. Most anaesthetists who work in the developing countries have basic levels of training and little advanced assistance to provide care for their patients. When required to provide artificial ventilation in general anaesthesia or intensive care they usually ventilate their patients manually using a self inflating bag or oxford bellows (Tindiumwemba et al 1992). Anaesthetists who work in advanced hospitals, often have access to the use of automatic lung ventilators, this leaves them with a free pair of hands to attend to other needs during anaesthesia. Lung ventilators have not been

widely used in the developing countries because of their high purchasing and running costs.

A promising breakthrough for developing world countries came in 1990 when Dr. R. Manley, planned to design and produce a prototype of a ventilator more suitable for the needs of the developing world. The Manley Multivent specifications included being simple, inexpensive, and a reliable machine with ability to ventilate adults and children above 20 kilograms of body weight. It would use mains electricity or battery as a source of power. It is cycled by compressed oxygen or air, the volume being equa to 10% of the volume required by the available ventilator which makes it cheaper to use. Through the exhaust port the cycling gas can be introduced into the patients circuit (if the gas is oxygen) to increase the FIO_2 of inspired gases. The ventilator is capable of integrating with draw over systems of anaesthesia like the Oxford Miniature Vaporizer (OMV) using Halothane.

The first Manley Multivent was designed and produced in 1991 and handed to Uganda for clinical trials (Manley, R. 1991). Since then successful clinical trials have been carried out in United Kingdom and various countries of Europe, Asia and Africa (Eltrigham et al 1993). In all clinical trials the ventilator has performed well. Bearing in mind the meagre funds allocated for health delivery in our developing countries, and the responsibility of anaesthetists to provide services to the community in the cheapest, safest and most effective way,

it is evident that this new ventilator has a role to play in the development of anaesthesia as far as artificial ventilation is concerned.

In this study on the Clinical Experience with the Manley Multivent carried out at Muhimbili Medical Centre assessment of the effect of the ventilator on cardiopulmonary stability in patients under balanced anaesthesia, the general performance of the ventilator and complication arising from its use was done.

PART 1:

LITERATURE REVIEW ON ARTIFICIAL VENTILATION

1.1 INTRODUCTION

One of the most striking advances in anaesthesia is the attention and skills now devoted to the attainment of proper ventilation of the patient's lungs by means of controlled ventilation. Spontaneous breathing may become seriously depressed and inadequate as a result of anaesthesia. Controlled ventilation or artificial ventilation may be facilitated by use of muscle relaxants, narcotic analgesics and sedative hypnotics. This will allow intricate intrathoracic surgery, neurosurgery and major laparotomies to be performed with ease and safety.

Rhythmic squeezing of a reservoir bag or self-inflating bag has been the basic method of providing controlled ventilation in life threatening conditions; Anaesthesia; and Intensive care units (ICUs) in developing countries. This has only served the purpose in short anaesthetic procedures, as for the long procedures lasting over three hours; and the extended life support required in ICUs it has not been always possible due to tiring of ventilating personnel and the irregularity of rhythm resulting thereafter. Some of the conditions requiring extended life support by mechanical ventilation include Tetanus; Poliomyelitis; drug poisoning; head injuries; crush injuries of the chest and post operative chest cases. This need has prompted the demand of modern ventilators which

are of increasing complexity now on the market.

Originally mechanical ventilators were designed for use during anaesthesia and were modified or adapted for use in the ICUs. As the clinical values of artificial ventilation in the treatment of respiratory insufficiency become increasingly evident, the main development was such that the majority of ventilators are now intended for use in the ICU.

1.2. HISTORY OF ARTIFICIAL MECHANICAL VENTILATION.

Artificial mechanical ventilation was introduced as early as 1555 by Vesalius. Employing a bellow he forced gas into the trachea of a dog with an open chest (Huston, 1973). One of the most unique attempts at mechanical ventilation was reported in 1776 by John Hunter who utilized a double bellow system assembled so that one bellow would pump ambient air into, and the other bellow aspirate the bad air out of the lungs. A nozzle from each bellow was inserted into the trachea of the dog and its life was maintained by alternating movements.

Despite these observations positive pressure mechanical ventilation was not to receive widespread use until more recent times. By mid nineteenth century, much attention was given to so called negative pressure ventilation. In 1864 Jones introduced the first body enclosing tank ventilator. The spirophore, considered the prototype of iron-lung was developed by Woillez in 1874. The device accommodated a supine patient and included a chest

excursion indicator rod that provided visual confirmation of lung expansion.

In 1928 Drinker and Shaw introduced the first iron lung. This enjoyed wide-spread use. The tank pressure changes were generated by an electronically powered rotary blower. A similar iron lung was built by J.H. Emersons in 1931 and it incorporated many improvements over existing models at approximately one half the cost. It was an air tight cylinder that enclosed the body leaving the head exposed to the ambient pressure. The opposite end of the tank contained a bellow that was powered either by an electric motor or manually in the event of power failure. Expansion of the bellows developed a subambient pressure within the tank and thorax that results in a position transpulmonary pressure gradient to facilitate gas flow into the lungs. Iron lungs saw extensive service during the Los Angeles poliomyelitis epidemic of 1948 to 1949 (Bower, A.G. et al 1950).

Modern positive pressure mechanical ventilation was initiated by Engstrom during the polio epidemic in Denmark in 1952 and in Sweden in 1953 (Lasen, 1953). The Engstrom ventilator provided reliable positive pressure ventilation and contributed significantly to a reduction in mortality. It was adopted subsequently to provide ventilation during anaesthesia and for post operative ventilatory support. (Bjork, V.O. et al 1955).

The value of mechanical ventilation in anaesthesia was soon recognized in Sweden, Britain and some European

countries, but was disputed in United States where reliance on manual assistance persisted. The more stringent ventilatory requirements of cardiac surgery were later to change this view (Seeley H.F. 1984). Since then numerous ventilators have been developed some relatively simple in design and function but suitable and reliable for use for anaesthesia and for patients needing long-term ventilation. Others are quite sophisticated and expensive but are versatile with facilities for treating patients with severe respiratory failure.

1.3. PHYSIOLOGICAL ASPECTS OF CONTROLLED VENTILATION:

1.3.1 Controlled ventilation.

Controlled ventilation is the term with which anaesthetists have become familiar for the process more properly referred to as artificial ventilation of the lungs. Both terms are virtually synonymous with the more limited, but descriptive, intermittent positive pressure ventilation (IPPV). Because we are concerned with anaesthesia the term controlled ventilation will be used.

Controlled ventilation was suggested in 1934 (Guedel, A.E. and Treweek D.N., 1934) as means of providing a quieter operating field in abdominal surgery. Little interest was aroused at the time and it remained for the demands of thoracic surgery (Mushin W.W. 1953) to bring the method to form. Controlled ventilation has now been in daily routine use in anaesthesia for some fifty years. At first it was the means of preventing the harmful effects of

the open chest and thus solving what had been known for nearly a century as the "pneumothorax problem". Later it became the way of compensating for the paralysing effects of curare and other relaxants on the respiratory muscles. By now many millions of patients have been successfully ventilated by this means with little evidence of harmful results. It can, therefore, be concluded that controlled ventilation is a very safe and effective method of ventilating the lungs in respiratory insufficiency.

The efficiency of artificial (or controlled) ventilation in oxygenating the blood is strikingly demonstrated by the speed with which a patient, cyanosed through respiratory insufficiency, flushes a brilliant pink when controlled ventilation is started so long as the cardiovascular system has remained normal.

The adequacy of carbon dioxide removal is shown by the speed with which a raised blood pressure, sweating, tachycardia and wound bleeding during surgery respond in a patient previously suffering from respiratory insufficiency.

The possible harmful effects of controlled ventilation lie principally in the directions of cardiovascular depression because of interference with venous return to the heart, and of disturbances in the distribution of blood and gases through the lungs during ventilation. However, it can be said here that when the mean pressure in the lungs is not excessive, and when the flow of gas through the respiratory tract is within normal limits and when

disease of the lungs with consequent alteration in local areas of compliance and resistance is not gross, the harmful effects, if any, in health subjects, are so small that they can be neglected, (Mushin W.W et al, 1959).

Nevertheless, it is essential to understand, from the physiological point of view the basis of the effectiveness and of the high degree of safety of controlled ventilation and the mechanism underlying the harmful effects which may, in certain circumstances, be produced. Only with such an understanding can those who use automatic ventilators make use of the more exact controls provided by them and employ controlled ventilation in such a way that the patients will derive full benefit thereof, whilst any harmful effects are minimized. Furthermore, with this knowledge it is possible to establish, on a rational basis, criteria for the design of automatic ventilators and their use in patients (Thompson, P.W. 1966).

1.3.2. Physiological Factors Affecting Volume Exchange:

1.3.2.1 Compliance

The relationship between the volume and the pressure of the gas in the alveoli when both are taken as changes from their static levels is determined by the elastic properties of the lungs and chest wall (Grimby, G. et al 1975). The term compliance expresses this relationship. It is defined as the volume change which corresponds to unit of pressure change in the alveoli. Normally this is

given in litres per centimetre of water (Litre/cmH_2O). Thus if an increase in alveolar pressure of 10 cmH_2O occurs with the volume increase within the alveoli of 500mls (0.5 litres), the compliance is 0.5/10 = 0.05 litres/cmH_20.

Once the compliance is known, the increase in alveolar pressure which corresponds to a given increase in volume can be easily calculated. Conversely given the increase in pressure the increase in volume necessarily accompanying it can likewise be calculated. The actual value of the compliance varies considerably in different individuals and in different circumstances in the same individual (Nunn,J.F. 1977). The range of the published figures is wide. In anaesthetized, relaxed, supine, patients the total compliance varies from 0.02 litres/cmH_20 to 0.17 litres/cmH_20 (Norlander, O. et al, 1968). The value for conscious individuals is usually quoted as 0.1 litre/cmH_20. This higher value may be partly due to the inability to refrain from some spontaneous inspiration during inflation and to the absence within the lungs, of the changes which occur during anaesthesia.

The total compliance has two components, that of the chest wall and that of the lungs. Again values vary but the average compliance of the lungs alone in anaesthetized patient is 0.1 litre/cmH_20 and that of conscious subject is 0.2 litre/cmH_20. When the chest is opened, the influence of the thoracic wall becomes very small and the total compliance is virtually equal to the compliance of the lungs alone. In clinical practice the compliance may be

modified considerably from moment to moment by such factors as the degree of muscular relaxation, posture, assistants leaning on the chest; retractors pushing against the lungs or diaphragm and, of course by the occlusion of bronchi or bronchioles, during thoracic surgery (Van Lith P. 1967).

Quite apart from the factors which affect total compliance, the compliance varies from one region of the lungs to another. In healthy individuals this depends on the relative positions of different parts of the lungs, in others the presence of disease may markedly affect the compliance of the affected areas (Ferris, B.G. 1952).

1.3.2.2 Respiratory Resistance:

This is the sum of the airway resistance and tissue resistance. The airway resistance expresses the relationship between the pressure difference across the airway (between the mouth and the alveoli) and the rate at which gas is flowing through the airway. It is expressed as pressure difference per unit flow and is usually measured in centimetres of water pressure per litre per second (cmH_2O/litres/sec).

Tissue resistance is the viscous resistance to expansion of the lungs. This arises to a small extent in the lung tissue it-self, but mostly in tissues of the chest wall as a result of friction in the tissues.

In normal conscious subjects, the respiratory resistance is about 4cmH_2O/Litres/Sec.). The resistance is not constant for a given airway, but it increases slightly with

increase in flow rate due to the development of turbulence. (Macintosh, R.R. et al, 1963). The value of airway resistance is increased during anaesthesia. This may arise due to both a narrowing and elongation of the bronchi and to the presence of apparatus such as an endotracheal tube. Thus during anaesthesia, the respiratory resistance from the carina distally is of the order of 4 cmH_2O/litre/Sec. With an endotracheal tube in place the total respiratory resistance is commonly of the order of 6cmH_2O/litre/sec. (Baker, A.B. et al 1974). The presence of disease may increase respiratory resistance for example in conscious subjects. Figures from 3 to 15cmH_2O/litre/Sec. in emphysema, and 13 to 18 cmH_2O in asthma have been found. Still higher values may be expected in gross respiratory obstruction.

During inflation of the lungs the pressure at the mouth must be greater than the pressure in the alveoli or no gas would flow from one to another. Conversely, during the expiratory phase the pressure in the alveoli must be greater, and there is, therefore at least an instant of time between the two phases when the pressure is the same throughout the respiratory tract and no gas flows in either direction. The difference in pressure depends on, and is an indication of, the magnitude of respiratory resistance. During inflation the actual pressure at the mouth at any moment is the sum of the pressure drop across the airway resistance and the alveolar pressure, which corresponds to the volume of gas in the lungs at that moment.

A clear distinction must, therefore always be drawn between the pressure registered at the mouth and the alveolar pressure. In practice during automatic ventilation the manometer is almost invariably situated on the ventilator. However, resistance between the ventilator and the patients mouth, and hence the pressure difference between is usually negligible. Therefore, for all practical purposes, the pressure at the ventilator can usually be taken to be the same as that at the mouth.

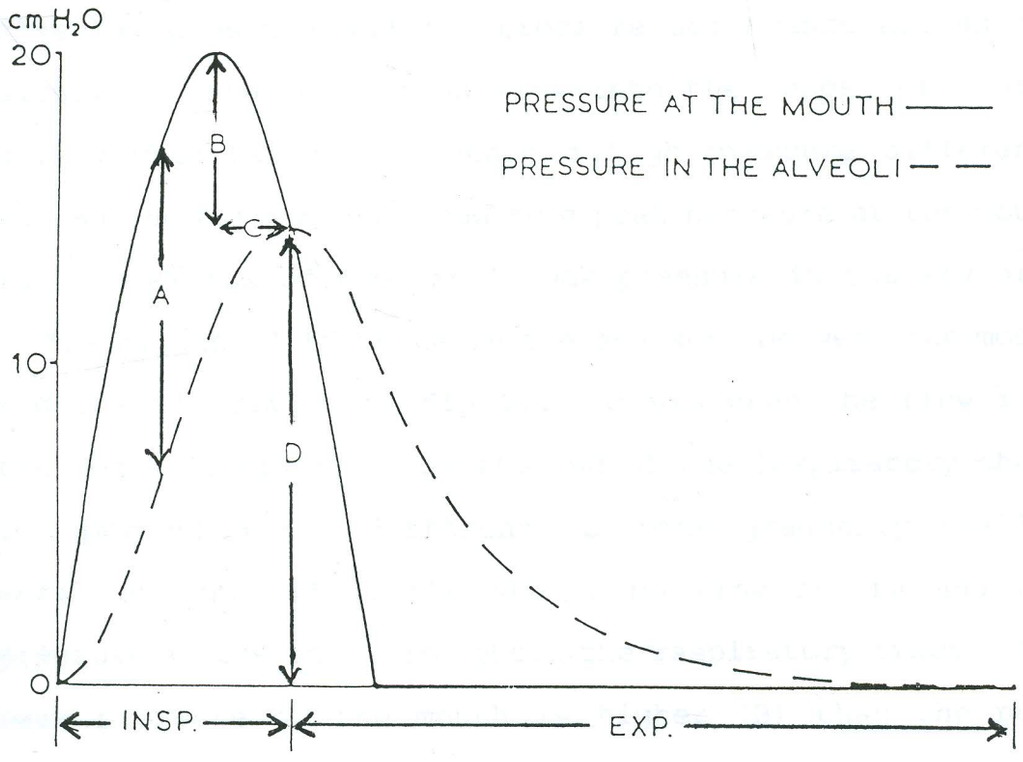


Fig. 1.1 Graph showing the difference that can exist between "pressure at the mouth" and "pressure in the alveoli" (Reproduced from recordings made during a laboratory experiment with an "artificial lung").

The figure illustrates a state of affairs which could

well occur in clinical practice. The pressure as recorded at the "mouth" rises rapidly to 20cmH_2O. At the end of the inspiratory phase when say 700mls have entered the "lungs" and the flow has fallen to zero, the pressure at the mouth and in the "alveoli" are the same (14cmH_2O), the compliance in this case being 0.05 litre/cmH_2O.

Assuming a free path for expiration, the pressure at the "mouth" now falls rapidly to zero (as shown in fig 1:1) whilst that in the alveoli does the same more slowly, since the pressure at the mouth is equal to the sum of the alveolar pressure and the pressure difference across the airway resistance, a high flow into the lungs, or a high airway resistance, and hence a high pressure difference across the airway, can lead to a peak pressure at the mouth much in excess of the final peak pressure in the alveoli.

The biggest difference in the pressure between the mouth and the alveoli (A in fig 1:1) occurs when the flow into the lungs is highest. As the end of the inspiratory phase is approached the difference becomes gradually smaller until at the end of the phase, no flow occurs and the pressure is the same throughout the respiratory tract. The peak pressure at the mouth is higher (B) than the peak pressure reached in the alveoli and it occurs some time(C) before the latter which marks the end of the inspiratory phase. It is possible, by watching a manometer registering mouth pressure, to note the highest a pressure reached there, but not the point (D), at which the mouth pressure is the same as the alveolar pressure. The anaesthetist

must, therefore, exercise caution in translating a manometer reading of pressure at the mouth into volume transferred to the lungs. Figures for compliance relate changes in the volume and pressure in the alveoli (Mushin W.W. et al 1980).

The resistance of unobstructed airway is small and with a slow rhythm of artificial ventilation, the pressure at the mouth during the inspiratory phase is only about 2 - 3 cmH_2O higher than in the alveoli. In these circumstances a practical approximation to the volume exchange can be made from the manometer reading if the compliance of the chest is known or assumed.

A higher pressure at the mouth then indicates that a correspondingly larger volume has been transferred to the patient and this may well be in excess of his normal tidal volume and is therefore undesirable.

There are other occasions, however when a high pressure at the mouth may be needed to produce a tidal volume within the normal range, the airway resistance may be high or the compliance low. A fall in tidal volume resulting from an increase in the airway resistance due to such causes as organic obstruction, increased secretions, or kinks in the tubing can be overcome to a large extent by increasing the pressure at the mouth, thereby maintaining a sufficient flow of gas, so that a tidal volume within the range desired is again transferred into the lung in the required time. Since in such a case expiration will be impeded by the respiratory obstruction and the pressure differences

across the airway at the beginning of expiratory phase is less than that applied at the beginning of the inspiratory phase, either extra time must be allowed for the lungs to empty or negative pressure applied during this phase to increase the pressure difference between the lungs and the mouth and so speed up the flow of gas out of the lungs (Mushin, W.W. et al 1980).

The need of caution in translating manometer readings on the ventilator into tidal volume is also necessary in respect of volume readings taken from a calibrated reservoir bag. The volume expelled from the bag may differ considerably from the volume entering the patients lungs. Losses may occur due to distension of the breathing tube and also the compression of the gas within them. In addition the entry and escape of fresh gases into and from the patients breathing system are responsible for further differences between the two volumes. A careful study of both the ventilation mechanisms and of its physical characteristic is therefore essential if the magnitude of the volume transfer to the patient is to be accurately estimated. A reliable ventilation meter will give an accurate measure of tidal volume and minute volumes if inserted at a suitable point in the expiratory pathway close to the mouth of the patient (Mushin W.W. et al 1980).

1.3.3. The Differences Between Controlled And Spontaneous Ventilation:

1.3.3.1 Normal Spontaneous Ventilation.

The respiratory muscles, principally the diaphragm, enlarge the size of the thoracic cavity, the specific volume (Macintosh, R.R. et al 1963) of the gas within it increases and the pressure within the thorax falls. The difference between intrapleural and alveolar pressure overcomes the elastic recoil of the lungs; the difference between the pressure in the alveoli and the exterior overcomes the airway resistance. The magnitude of these two pressure differences differ from each other. In quiet unobstructed ventilation, the pressure difference across the airway even at the height of gas flow, is small say $2cmH_20$; that between the pleura and the alveoli is larger, ranging from - $10cmH_20$ at the end of inspiration to - $5cmH_20$ at the end of expiration.

1.3.3.2 Intrapulmonary Pressure

During spontaneous ventilation air flows from the outside atmosphere to the inside of the lungs because the pressure difference is created between the exterior and the alveoli. This pressure difference is of comparatively small magnitude since it has only to overcome the airway resistance, the main effort of the respiratory muscles being used to overcome the elastic recoil of the lungs. The pressure difference in a conscious subject breathing quietly is of the order of 1 - $2cmH_20$ (fig. 1. 2) and since

the pressure at the mouth is atmospheric; the pressure in the alveoli during inspiration must be subatmospheric by this amount. By the end of inspiration the pressure within the alveoli has become atmospheric again. When expiration starts, the pressure in the alveoli rises a few cmH_20 above atmospheric and gradually falls to atmospheric again as the lungs empty.

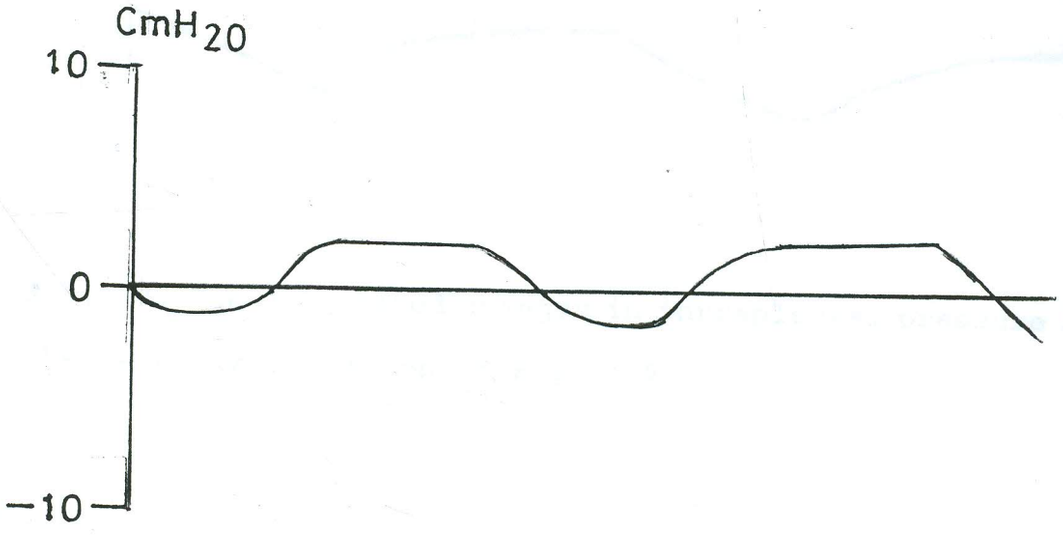


Fig.1.2: The calculated changes in pressure in the alveoli of a conscious patient breathing spontaneously and quickly. Tidal volume 800mls, compliance of lungs and thorax = 0.1 litre/cmH_20, airway resistance = $2cmH_20$/litre/Sec. Respiratory frequency 15/min.

In contrast to this during controlled ventilation (Fig. 1.4) with positive pressure alone, the pressure in the alveoli rises from atmospheric to say + $16cmH_20$ (for a volume of 800mls, assuming compliance of 0.05litre/cmH_20). During the expiratory phase the pressure falls to atmospheric as the lungs empty.



Fig.1.3: The calculated changes in intrapleural pressure in the same circumstances of Fig. 1.2.

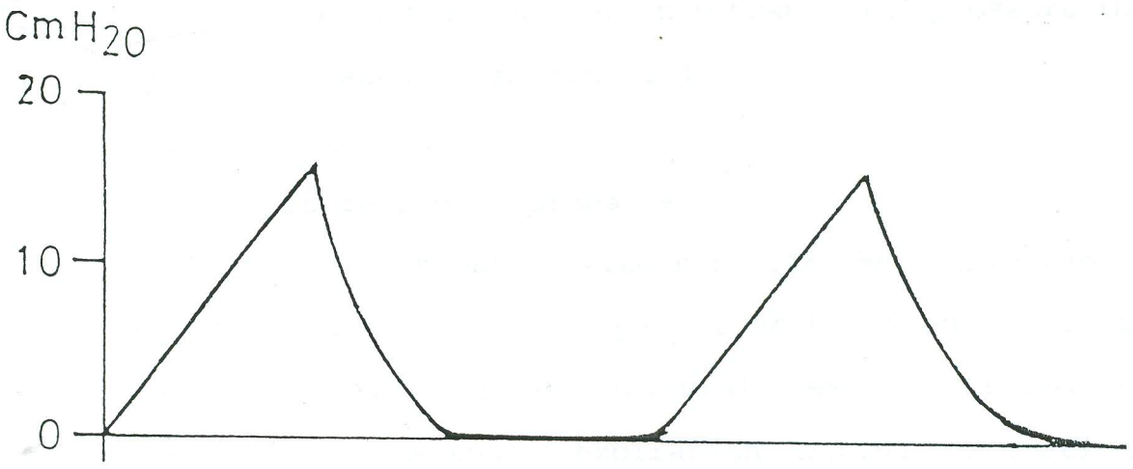


Fig.1.4 The calculated changes in pressure inthe alveoli of a patient with intermittent positive pressure ventilation. Tidal volume = 800mls compliance of lungs and

thorax = 0.05litre/cmH_{20}, compliance of the lung alone = 0.1 litre/cm$H_2$0, resistance (including apparatus) = 4cm$H_2$0/litre/sec, respiratory frequency = 15/min

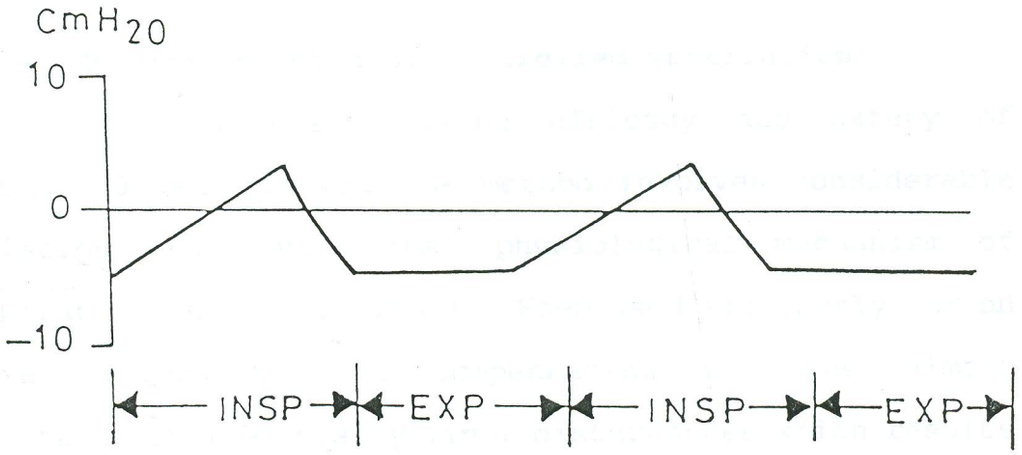


Fig.1.5: The calculated changes in intrapleural pressure in the same circumstances as Fig. 1.4.

1.3.3.3 Intrapleural pressure:

The intrapleural pressure also reflects the considerable difference between the two types of ventilation. During spontaneous breathing the intrapleural pressure is normally about - 5cm$H_2$0 at the end of expiration. During inspiration a further fall occurs to about - 10cm$H_2$0 returning to - 5cm$H_2$0 during expiration (Fig. 1.3).

In controlled ventilation (Fig. 1.5) the same condition as in Fig. 1.4 (tidal volume 800mls, compliance of 0.05litre/cm$H_2$0) the intrapleural pressure rises during the

inspiratory phase from - 5cm$H_2$0 to + 3cm$H_2$0 falling to - 5cm$H_2$0 again during expiration. The rise of 8cm$H_2$0 is half that in the lungs because it has been assumed that the compliance of the lungs alone is double that of the lungs and chest wall.

1.3.4 Harmful effects of controlled ventilation

In spite of the striking efficacy and safety of controlled ventilation, the method involves considerable deviation from the normal physiological mechanism of respiration (Nunn,J.F. 1966). When used improperly, or on patients incapable of compensating for the almost inevitable though usually minor disturbances which results from interference with normal bodily process, controlled ventilation may become significantly harmful.

1.3.4.1 Cardiovascular effects:

The connection between the venous return and the intrathoracic pressure was recognized by Valsalva (Klein, L.J. et al 1964, Stone, D.J. et al, 1965) in the seventeenth century. His experiment of trying to expire against a closed glottis raises the intrathoracic pressure to a positive level. It also interferes with the venous return, causes marked distension of the peripheral veins, especially in the head and neck. If the experiment is persisted with, the subject may lose consciousness as a result of fall of blood pressure and a low cerebral blood flow.

MUHIMBILI MEDICAL UNIVERSITY COLLEGE
LIBRARY
DSM.

(i) The Abolition of the "Thoracic Pump"mechanism:

The fall of pressure with the thorax during spontaneous inspiration not only sucks air into the lungs, but also blood from outside the thorax into the great thoracic veins and the heart (Brecher, G.A. 1956). The interference and the modification of the subatmospheric pressure changes by controlled ventilation disturbs this important mechanism. The normal pressure gradient between the intrathoracic veins and those outside is then upset because the positive pressure within the lungs is transmitted in part to the structures within the thorax as seen in fig. 1.4 and 1.5.

During spontaneous breathing (fig. 1.3) the pressure within the thorax or the intrapleural pressure, at the height of inspiration is say - 10cm$H_2$0. During controlled ventilation (fig. 1.5) this becomes + 3cm$H_2$0. Only during the quiescent part of the expiratory phase is the negative intrapleural pressure the same in both spontaneous and controlled ventilation.

During spontaneous breathing the venous return is therefore greatest during inspiration while in controlled ventilation the situation is reversed and the venous return rises during expiratory phase (Morgan, B.C. et al 1966; Hubay, C.A. et al 1954).

At the end of the inspiration phase of controlled ventilation the central venous pressure is raised. The venous return to the heart and hence cardiac output are reduced and the blood pressure falls. Normally however this is rapidly compensated for by a rise in peripheral

venous pressure which reconstitutes the venous gradient and so re-establishes the venous return to its former level. The restoration of the venous gradient is essential if an adequate cardiac output during controlled ventilation is to be maintained. It is dependent upon the capacity to adjust the distribution of blood between the vascular compartments by changes in vascular tone, and on the presence of an adequate circulating blood volume.

Should either of these factors be interfered with or the inspiratory phase be unduly prolonged, then the untoward circulatory effects of the positive pressure in the lungs will become marked. For example severe haemorrhage markedly reduces the volume of circulating blood. In such a patient extensive vasoconstriction may have already occurred to counteract the hypovolaemia, and further compensation be impossible; the effect of controlled ventilation may then be to hinder the circulation still more.

(ii) Tamponade of the Heart:

During the inspiratory phase of controlled ventilation the heart itself is compressed to some extent between the expanding lungs as a result of which its output suffers. In controlled ventilation at the end of inspiratory phase the heart is subject to a slight positive pressure, whereas in spontaneous breathing the intrapleural pressure is then at its lowest level. The higher the peak pressure and the longer the time during which it acts (or the higher the I:E ratio) the greater is the cardiac tamponade and the

interference with the cardiac output.

Two simple clinical observations will confirm the embarrassing effects on the circulation at initiating controlled ventilation. The drop rate of an intravenous infusion is often seen to slow as the intrathoracic pressure increases during each inflation. In the same way when a central venous pressure line is in use not only does the venous pressure rise, but it is also seen to fluctuate with the variations in the intrathoracic pressure.

(iii) Interference with pulmonary blood flow:

The pulmonary capillary blood pressure is about 11cm$H_2$0 (Wasserman, K. et al 1966). The capillaries will therefore suffer compression in part or whole as the alveolar pressure rises above atmospheric. Even pressure as low as 6.5cm$H_2$0 in the lungs decreases the pulmonary capillary circulation and throw an extra burden on the right ventricle. This is easily tolerated by most patients, but not by one who is on the verge of decompensation, even such a low pressure in the lungs as this may be sufficient to precipitate a right heart failure.

In considering the relative importance of these three ways in which controlled ventilation interferes with the cardiovascular system it should be borne in mind that, while they are all of considerable importance in a patient with an intact thorax, the circumstances alter when the chest is opened. The thoracic pump now no longer operates because the chest is open while any cardiac tamponade if it occurs, will be mainly surgical in origin. The

interference with the pulmonary blood flow is now the most important adverse effect directly due to the controlled ventilation and may itself be sufficient to interfere seriously with the cardiac output.

(iv) Cerebral vasoconstriction:

Hyperventilation leads to cerebral vasoconstriction and to a potentiation of anaesthesia (Clutton-Brock,J.1957). There is little doubt that the success of modern anaesthesia brought about by the widespread use of relaxants, controlled ventilation and small doses of anaesthetic agents, could hardly have occurred without hyperventilation being commonly practiced during anaesthesia (Geddes, I.C. 1959). The mechanism whereby hyperventilation causes cerebral vasoconstriction is not completely understood though it is to large extent dependent on the reduction of the level of circulating carbon dioxide (Ketty S.S. and Schimdt C.F. 1946).

1.3.4.2. Respiratory Effects:

(i) Damage to the Lungs:

The probability of rupture of the alveoli is very small in properly conducted controlled ventilation, unless such condition such as bullous emphysema is present. Rupture even then is probably no more likely to occur during controlled ventilation with properly limited positive pressure than during normal life (Luke, H.C. 1913). Each act of coughing produces an intrapulmonary pressure which may be as high as 80 - 90cmH_2O, while

straining at defecation may give rise to still higher pressure (Adams, W.E. 1940).

The pressure required to rupture the exposed and unsupported lungs of various mammals has been found to be about 40 - 80cmH_2O. When the lungs are supported by the thoracic cage and the abdominal musculature of the living animal a pressure of 80 - 140 cmH_2O is needed. The maximum safe intrapulmonary pressure in intact mammal is about 70 cmH_2O. The pressure developed by squeezing the thin rubber reservoir bag commonly found on anaesthetic apparatus can rarely be made to exceed 40 - 60 cmH_2O (Macklin, C.C.1937). Nevertheless, now that devices capable of producing higher inflating pressures are readily available, the possibility of damage to the lungs must be borne in mind. Rupture of lungs in man leads to interstitial emphysema spreading up the pulmonary vascular sheaths into the mediastinum (Waltz, R.C. et al 1954). Rupture of the pleural in these cases is probably a secondary event, the primary process being the stretching away of the alveoli from the inelastic pulmonary vascular sheaths lying between them. A subsequent leak of air from mediastinum to the intrapleural space may occur. Notwithstanding the rarity of pneumothorax or surgical emphysema as a result of artificial ventilation in patients with intact lungs, these complications occur not infrequently when controlled ventilation is applied to those who have already suffered lung damage as a result of chest injury (Heidrick, A.F. et al 1940).

(ii) Uneven ventilation:

It should be borne in mind that in many patients the distribution of the inflating gas within the lungs is not uniform. When this occurs the normal relationship that must exist between the ventilation of the lungs and the perfusion of the lungs with blood is disturbed. The full implication of uneven ventilation are complex and may be exaggerated during inexpert controlled ventilation. The most serious result of this is the perfusion with venous blood of insufficiently ventilated parts of the lungs and therefore, insufficient oxygenation of this blood. The resulting condition is that of a venous shunt from the right side of the heart to the left (Gray, T.C.1959). In otherwise healthy individuals uneven distribution of gas eventually results in a diminution of blood flow through the less well ventilated parts of the lungs and any venous shunt is thereby minimized. Another possible effect of importance is the ventilation of insufficiently perfused parts of the lungs, such ventilation is largely wasted and represent an increase in the physiological dead space. In such circumstances a "normal" ventilation may result in a raised arterial Pco_2 (Mushin, W.W et al 1980).

Uneven ventilation may be due to localized changes in elasticity or in the patency of the small airway such as are found in asthma, chronic bronchitis and emphysema. Apart from pathological causes within the patient, the lateral posture causes uneven ventilation by decreasing ventilation of the dependent lung. Surgical retractors and

packs may also cause uneven ventilation by limiting the expansion of small or even extensive parts of the lung. The localized accumulation of secretions in the airways will also have the same effect.

In any of these circumstances of uneven ventilation and of disturbed ventilation/perfusion ratio an added risk of alveolar damage exists. At some point in the respiratory cycle, differences of pressure exists between neighbouring alveoli or parts of the lung, and unequal forces act across the alveolar septa tending to rupture them. Although clinical reports have not drawn attention to this type of damage, perhaps because of the difficulty in recognizing it in vivo, it may be more common than is suspected. That vigorous ventilation within clinical limit can produce widespread alveolar damage is shown by case reports (Gray, T.C. 1959, Thompson, P.W. 1963) in which the lungs were examined histologically after death. The possibility of damage to the lungs resulting from uneven ventilation must be regarded as to some extent speculative and it should not deter the anaesthetist in his attempt to ventilate his patient adequately.

(III) Disturbances of Acid-Base Balance:

The Acid base balance of the blood will be disturbed with any deviation of alveolar ventilation from normal. Hyperventilation will cause a fall in Pco_2 and a rise in the pH, that is an alkalaemia conversely, hypoventilation will lead to a rise in the Pco_2 and a fall in pH that is an acidaemia. Ideally the normal equilibrium should be

maintained as being part of the optimum physiological environment.

However there is evidence that retention of carbon dioxide in the body, as indicated by a rise of Pco_2 and a fall in pH, is to be avoided since it leads not only to a general depression of the body, including the central nervous system, ending in coma but also to important undesirable effects on individual organs (Nunn, J.F. 1977). On the heart, for example it enhances the toxic action of many drugs for example cyclopropane and halothane, and of such procedures as hypothermia. Hyperventilation for short periods such as occur during surgery seems to be harmless, though the risk of lung damage should always be remembered. In as much as hyperventilation will, in general, make certain of good oxygenation and carbon dioxide elimination, it is to be preferred to hypoventilation (Mushin W.W. et al 1980).

1.3.4.5 Other Harmful Effects:

Certain other effects may be associated with controlled ventilation particularly if carried out by face mask (Mushin W.W. et al 1958) instead of an endotracheal tube. Then, the possibility of accidental inflation of the stomach arises. The pressure at the mouth in these circumstances should therefore, be carefully limited, thus while pressures of up to $15cmH_20$ rarely distends the stomach, pressure over $20cmH_20$ invariably does so. If this occurs the stomach dilates and may present hindrance to the

surgeon performing an abdominal operation. In addition, unless the gas in the stomach is evacuated by means of a tube the patient may suffer acute discomfort in the post operative period. Rupture of the eardrums has also been reported during controlled ventilation (Whiltigham, J.K.R. 1954).

1.3.5 Minimizing The Harmful Cardiovascular Effects Of Controlled Ventilation:

1.3.5.1 Mean Intrapulmonary Pressure:

Any harmful cardiovascular effects of controlled ventilation are basically the result of abnormally high pressure within the lungs. This eliminates in part, or whole, the subatmospheric pressure normally present within the thorax. The extent to which this occurs depends on the pressure within the lungs and on the lung compliance. If the compliance is very low, then a high intrapleural pressure will be required to achieve adequate ventilation, but only a small fraction of this pressure will be transmitted to the other intrathoracic structures. Whatever the compliance, any means by which the intrapulmonary pressure can be reduced will also reduce the pressure applied to the intrathoracic structures.

Both the magnitude of the positive pressure and the time during which it is applied are of importance. These two factors are combined in the term "Mean pressure". The lower the mean intrapulmonary pressure during the respiratory cycle, the less marked these cardiovascular

effects will be (Cournand, A. et al 1948, Maloney, J.V. et al 1953, Hubay, C.A. 1954, Nunn, J.F. 1969).

The mean pressure is the mean of a very large number of equally intervaled instantaneous readings of the pressure within the lung during one respiratory cycle. (fig. 1.6) To determine the mean it is necessary to take the sum of all the instantaneous readings of the pressure and divide it by the number of readings.

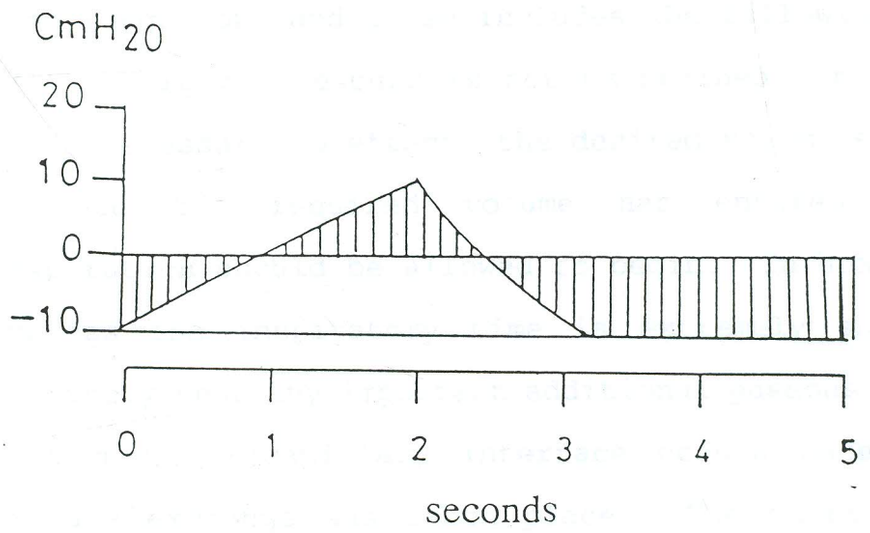


Fig 1.6: Graph illustrating the determination of mean pressure by taking many equally spaced measurements of the height of the curve over one respiratory cycle.

The former is proportional to the area between the pressure curve and the line of zero pressure; the number of readings is proportional to the length of the baseline which represents the duration of one cycle. The mean pressure is therefore equal to the area enclosed between the pressure curve and the line of zero pressure for one respiratory cycle, divided by the duration of the cycle.

Should there be any negative pressure in the lungs during the respiratory cycle the pressure curve will fall below the zero line. Areas enclosed by the pressure curve below the zero line should be taken as negative and subtracted from the area enclosed by the pressure curve above the zero line.

1.3.5.2 Reduction of the mean intrapulmonary pressure:

Several ways may be used to keep the mean pressure in the lungs low, and these includes the following.

(i) Positive pressure is not maintained for longer than necessary to effect the desired volume exchange.

Once the required volume has entered the lungs expiration should be allowed to begin. In healthy lungs, unless the inspiratory time is extremely short, it is unlikely that any important additional gaseous exchange at the alveolar/capillary interface occurs once the tidal volume exchange has taken place. The maintenance of a plateau in the pressure curve after the tidal volume exchange has occurred, may aid more even alveolar distribution of the inflating gas in patients with pulmonary disease. However, this benefit will, to some extent, be offset by the circulatory embarrassment which may also result.

(ii) Inspiration is shorter than expiration:

It is important to avoid undue limitation of the time allowed for expiration. The expiration phase should occupy more than half the respiratory cycle (Cournand, A. et

al, 1947; Astrup, P. et al 1954; Nunn, J.F. et al 1969 and Gordon, A.S. et al 1956). A shorter expiratory phase and a longer positive pressure inspiratory phase will tend to cause a reduction of cardiac output (Motley, H.L. et al 1948 and Spalding, J.M.K. 1955). Many ventilators allow the ratio of inspiratory to expiratory time to be varied between 1:1 and 1:4. A common setting is about 1:2.

An important benefit of a longer expiratory phase is that it allows the lungs to deflate so that the intrapulmonary pressure falls to zero. The heart is free from continued tamponade and its output then rises, the intrathoracic pressure falls, and the great veins and heart fill (Gordon, A.S. et al 1956). If the expiratory period is too short these events are curtailed and the next inflation commences while the lungs are still partially inflated; the heart is subjected to continuous tamponade, the venous return is continuously hampered and the pulmonary circulation is continuously impeded. The mean pressure within the lungs is raised, and even in a healthy adult, a short expiratory period may cause gradually increasing cardiovascular embarrassment. In chronic bronchitic and emphysematous patients in whom there is already a degree of cardiorespiratory failure a short expiratory period may cause even further deterioration in cardiovascular function (Werko. L. 1947).

On the other hand, there is a reason for not using an inspiratory: expiratory ratio which is too small (Nunn,

J.F. 1969). There is evidence that, at least in the case of pressure generators when the inspiratory period is reduced to about 0.5 second, that is the inspiratory: expiratory ratio approaches about 1:6 the physiological dead space may exceed 50% of the tidal volume.

(III) The lungs are inflated with a rapid flow:

The faster the flow of gas into the lungs the shorter the inspiratory phase can be. Limits, however, are set to this by mechanical designing problems; by the danger of alveolar rupture; and uneven ventilation. However in general terms the tendency should be to use higher rather than lower flows during inflation (Pask, E.A. 1955 and Nunn, J.F. 1969).

(iv) The Expiratory resistance is kept low:

Any resistance to the outflow of gas from the lungs will delay the fall of pressure during the expiratory phase and hence raise the mean intrapulmonary pressure and its consequences.

(v) Minimizing the dead space:

The dead space may be reduced by the insertion of an endotracheal tube or tracheostomy tube; and by the careful design of the connections between the ventilator and the patient. By taking this steps the tidal volume needed to produce a given alveolar ventilation at a given respiratory frequency can be reduced. This will reduce the peak and hence the mean intrapulmonary pressure (Mushin, W.W. et al 1980).

(vi) Negative pressure is applied during the expiratory phase:

The application of negative pressure during the expiratory phase lowers the mean intrapulmonary pressure in two different ways. First way is that if the inspiratory phase starts with a negative pressure of a few cmH_20 in the lungs, the required pressure difference; and hence volume exchange occurs with a lower peak of positive pressure. This is what occurs in automatic ventilation in which a negative pressure is maintained throughout the expiratory phase. The second way is that a negative pressure can also accelerate the fall in pressure in the lungs during expiration (Maloney J.V. et al 1953; Hubay. C.A. et al 1954; Maloney. J.V. and Handford. S.W. 1954).

In practice most or all of these various methods of reducing the mean intrapulmonary pressure and maintaining the venous return are employed in the same patient. The patient suffers less cardiovascular harm if his ventilatory requirements are satisfied with the least mean intrapulmonary pressure. Whether one factor is more important than the other is still not clear.

1.4.0 CLINICAL ASPECTS OF CONTROLLED VENTILATION:

1.4.1. Indications for Controlled Ventilation:

The commonest and most important indication for controlled ventilation is respiratory insufficiency. This term is used for any condition in which either respiratory activity is completely absent, or if present is

insufficient to maintain either adequate oxygenation or carbon dioxide clearance.

I) Respiratory insufficiency associated with Anaesthesia:

During anaesthesia the deliberate production of apnoea is the common reason for controlled ventilation (Guedel A.E. 1949). Originally such a state; produced by a combination of anaesthetic overdose and hyperventilation was only considered really necessary during open chest surgery (Nosworthy, M.D. 1941; Neff, W., Phillips, W. and Gunn, G. 1942). Nowadays relaxants have made the induction of apnoea easy, and this technique is employed, not only in thoracic and abdominal surgery, but in a wide range of operations in which profound relaxation and the abolition of spontaneous respiration is advantageous.

Post operatively, respiratory insufficiency or even respiratory arrest may need controlled ventilation (Bjork, V.O. and Engstrom C.G. 1955; Rudy, N.E. and Crepean, J. 1958, Becher, A. et al 1960). Prolonged or disordered action of relaxant drugs; the combined effects of circulatory and respiratory depressants; the effect of oedema of the brain and of raised intracranial pressure generally, may all produce respiratory insufficiency during this period (Bartleth, R.H. et al 1973).

Indeed, apnoea is sometimes deliberately maintained for one or two days after open-heart operations in order to reduce the patient's muscle metabolism, and hence reduce the need for oxygen (McConnel, D.H. et al 1974).

ii) Respiratory insufficiency in diseased states unconnected with surgery or anaesthesia:

Spino - bulbar poliomyelitis (Lassen, H.C.A, 1953; Harries, J.R and Lawes, W.E, 1955; Kelleher W.H. et al 1956) and other neurological conditions in which respiratory activity is impaired such as polyneuritis and myelitis (James, J.L. and Park, H.W.J. 1961); overdose of narcotic, hypnotic or other drugs (Freier, S. et al 1957); and severe cases of myasthenia gravis (Griffin, S.G. et al 1956) are important indications for controlled ventilation. Tetanus is also an indication in certain circumstances (Honey, G.E. et al 1954; Ablett, J.J.L, 1961; Glossop, M.W and Low M.D.W. 1957 Adams, E.B. et al 1966). The spasms are controlled by relaxants or other drugs, the consequent respiratory insufficiency needing controlled ventilation.

Patient suffering from carbon dioxide accumulation due to respiratory disease, may be tided over a critical period (Bjorneboe, M et al 1955). The method is commonly used not only in the respiratory crises (Misuraca, L. 1966; Ambiavagar, M. et al 1967) of such diseases as chronic bronchitis, asthma, and emphysema in the adult; but also in respiratory distress syndrome" of the newborn (Reid, D.H.S. and Tunstall M.E., 1965; Reid, D.H.S and Tunstall, M.E. 1966). Beneficial effects may be obtained by treating patients with crush injuries of the chest by controlled ventilation (Boyle, A.K. et al 1957, Clarkson, W.B. and Robinson, J.S. 1962; Hunter, A.E. 1964; Garden, J. and Mackenzie A.I. 1963). In these cases the paradoxical

movement of the chest wall has the same significance, and is just as harmful, as paradoxical spontaneous movement of the lungs during open-chest surgery (Mushin, W.W. and Rendell Baker, L. 1953).

iii) Other Uses of Controlled Ventilation:

a) In Neurosurgery the patient derives benefit not only from the respiratory effects of controlled ventilation, but also from what would in other circumstances be regarded as side effects (Marubini, M.B. et al 1964). Controlled ventilation lowers the intracranial pressure and reduces both the brain volume and the CSF pressure. An increase in the minute volume ventilation produces a fall in the alveolar PCO_2, the systolic blood pressure and the ventricular fluid pressure.

b) Induced hypotension - Controlled ventilation tends to exaggerate the hypotension produced by ganglion - blocking drugs used to lower the blood pressure when this is considered desirable during anaesthesia. This effect is due to a combination of its cardiovascular effects and hypocapnia.

c) In Pulmonary oedema (Miller W.F. and Sproule, B.J. 1959) of whatever the cause. This condition represents a disturbance of the normal balance of forces across the alveolar membrane. Controlled ventilation, by its ability to increase the mean intra alveolar pressure, increases the force tending to drive liquids back into the circulation, and to

prevent a further transudation into the alveoli.

d) The use of controlled ventilation has from time to time been reported for other conditions than those already mentioned, for example in drowning (Redding J, et al 1960; Toland, C, 1972, Model J.H. et al 1974); hypoventilation of obesity (Hargrave S.A. et al 1973; Waltermath, C.L. 1974) fat embolism (Galloon, S and Chakravanty K, 1967; List W.F. 1969) and snake bite (Casale F.F. and Patel S.M. 1974).

1.4.2 Volume Exchange:

The tidal volume delivered by a ventilator may be inadequate for the needs of the patient. This may be the result of an incorrect setting; failure to recognize the development of mechanical faults in the apparatus; or of leaks between apparatus and patient, which may cause the volume entering the lungs to be less than that leaving the ventilator. However, even when the patient's tidal volume has been estimated correctly and is entering the lungs, the accumulation of secretions, or the presence of emphysema or areas of atelectasis, may interfere with proper distribution of the gas and with the efficient gas exchange in the alveoli.

Poor oxygenation of the arterial blood and inadequate elimination of carbon dioxide usually occur together. However, oxygenation may be adequate while at the same time a high, and sometimes dangerous, levels of carbon dioxide may occur as in diffusion respiration during bronchoscopy

(Roth L.W. et al 1947). A patient who is made apnoeic with relaxant, and from whom the nitrogen has been eliminated, remains oxygenated for periods of even over half an hour, so long as the respiratory tract is kept filled with oxygen. Carbon dioxide accumulates steadily and severe respiratory acidosis may occur at the end of the half hour.

Any patient who is hypoventilated but is receiving a gas mixture containing a high concentration of oxygen, may be bright pink yet have a high blood carbon dioxide content. The high oxygen content and the appearance of well-being mask the evil effects of hypoventilation.

1.4.3 Hypoxia

The danger of hypoxia needs no emphasis and its recognition is generally easy. Cyanosis is usually present, though anaemia, carboxyhaemoglobin, pigmented skin, or even cosmetics, are examples of pitfalls for the unwary. All these mask, or prevent the development of cyanosis. Further, even amongst skilled observers, there may be a wide variation in the estimation of cyanosis (Comroe, J.H. and Botelho, S. 1947). It is therefore unsafe to assume that oxygenation is adequate simply because cyanosis is not clearly evident.

The degree of suboxygenation can only be accurately determined by estimating arterial oxygen levels. This is sometimes done by measuring the oxygen saturation of the arterial blood, but the shape of the oxygen dissociation curve is such that the method is not particularly valuable

in detecting minor degrees of suboxygenation. Cyanosis is rarely clearly visible until the oxygen saturation is below 80% a figure which corresponds to half the normal oxygen tension on the dissociation curve.

Measurement of oxygen saturation in practice, therefore though valuable as a guide to the effectiveness of controlled ventilation, is of secondary importance in initial clinical assessment of the need for instituting such treatment (Mushin. W.W. et al 1980).

1.4.4 Hyperventilation:

Hyperventilation appears to be less serious and is less readily recognized than hypoventilation, which is usually easily detected by the serious clinical signs of suboxygenation or carbon dioxide accumulation (Rollason, W.N. and Parker, J. 1957). In general, controlled ventilation performed by hand tends towards hyper-ventilation. The anaesthetist usually squeezes the bag too vigorously and too often. When performed by machine, however, with the controls set in empiric manner, it may all too easily tend in the opposite direction. Hyperventilation is of serious consequence if prolonged or if the patient suffers from certain underlying respiratory diseases. Continued hyperventilation in subjects suffering from chronic respiratory acidosis due to chronic bronchitis and emphysema will quickly remove any hypoxic drive to respiration because of the good volume exchange and lowering of the blood carbon dioxide below what is

probably an already high threshold value (Nunn, J.F.1977). If such a patient is hyperventilated even for a short time as during surgical operation, he may well remain apnoeic and fail to start to breath spontaneously at the end of operation.

Gross hyperventilation may cause tetany due to alkalosis which results from excessive elimination of carbon dioxide. Whether tetany can be detected depends on how paralysed or how deeply anaesthetized the patient is.

Another effect of hyperventilation is cerebral vasoconstriction with a fall in cerebrospinal fluid pressure and reduction in brain size. These effects may be found of value in neurosurgical anaesthesia. However, in patients with cerebral arteriosclerosis in whom the cerebral blood flow may already be impaired, any further reduction in cerebral blood flow due to hyperventilation may lead to irreversible damage. It may be that the serious personality changes sometimes leading to dementia seen in old people after operation may, at least in part, be due to this mechanism (Stroddart, J.C. 1967).

Hyperventilation also causes a gradual fall in the blood pressure with peripheral vasoconstriction and pallor (Rad, L.N. 1970). In the abscence of clear clinical signs, estimation of the arterial PCO_2 is the only certain means of confirming suspected hyperventilation.

1.4.5 Estimation of ventilation requirements:

By whatever means the ventilation requirements of a

patient is calculated the best checks on its accuracy are the repeated estimation of arterial Pco_2, oxygen saturation and arterial PO_2. (Pontoppidan H. et al 1965) The value of this estimations for the benefit of a patient suffering from respiratory insufficiency is so great as to outweigh any possible hazards of repeated arterial puncture or the insertion of indwelling arterial catheter. The means for performing this and other investigations are fortunately becoming readily available. Nevertheless, the laboratory estimations do not remove the need of frequent and meticulous clinical assessments of the patient for especially hypoxia, hypercarbia and hypocarbia, which will go far to detect pulmonary complications at an early stage.

The basal tidal volume for any patient may be obtained from normograms which give estimations for patients of various weights and heights (Radford, E.P.et al 1954). These tables are based on accumulated data linking respiratory requirements with body surface area and hence with metabolic activity. A tidal volume and frequency chosen from a range on the normogram corresponding to the weight of the individual patient, is claimed to maintain an arterial PCO_2 of 40mmHg (5.3 kPa) within reasonable limits (Radford, E.P. 1955).

The actual minute volume resulting from various combinations of tidal volume and frequencies is larger with high rate of ventilation. The lower the ventilation frequency, the nearer the alveolar ventilation approaches the total volume of ventilation (Woolmer, R. and Dobkin,

A.B.1958). The normograms gives basal tidal volumes and lists a number of corrections which must be applied in various conditions such as the presence of fever, metabolic acidosis during anaesthesia and change in altitude.

1.4.6 Measurement of Tidal Volume:

It is evident that when the tidal volume has been selected it is still necessary to ensure that this volume is infact transferred to the patient's lungs. Therefore it is necessary in cases of long-continued ventilation to incorporate some type of ventilation meter. This is best placed close to the patient in the path of his expiration, so that the volume of gas measured has actually come from the patient's lungs. Such a meter must be of a type which is not upset by the condensation of water within its mechanism. If it is, either an effective barrier to water such as a condenser humidifier (Walker A.K.Y and Bethune, D.W. 1976) must be inserted or, as an expedient, the meter must be placed in the path of the inflating gas, carefully guarding against leaks as a source of error.

As with any sensitive measuring device, regular checks should be made of the accuracy of the calibration of the meter (Byles, P.H. 1960, Nunn, J.F. and Ezi-Ashi T.I. 1962). In a well established unit it is wise to have on robust gas meter against which at least a rough checking of the more fragile meters can be made. The makers of the ventilation meters provide calibration graphs for both constant and sinusoidal flows, so that the necessary

comparisons and corrections can be made.

1.5.0 VENTILATORS AND MECHANICAL VENTILATORY SUPPORT:

Ventilators are devices used for mechanical ventilation to provide ventilatory support or controlled ventilation in critically ill patients in Intensive care or during anaesthesia.

Mechanical ventilatory support is a positive airway pressure therapy that is applied primarily to enhance carbon dioxide excretion. Research and development of positive pressure ventilators that could minimize the physiological disadvantages of positive pressure ventilation and provide the greatest degree of reliability and versatility have led to several modes of mechanical ventilatory support. The importance of the available technologies rests not in their ability to achieve adequate ventilation but rather in the degree to which they minimize disruption of normal cardiopulmonary physiology (Roy. D.C, Shapiro, B.A. 1985).

1.5.1 Desirable Features for an Ideal Ventilator:

There are numerous ventilators available for controlled ventilation and each have different working principles, behaviour and special functions (Bersten A.D.; Skowronuki, G.A, Oh, TE 1986). Despite all those differences an ideal ventilator should provide for

i) Ability to ventilate all sizes of patients, from neonate to adults.

ii) Versatility of operation, or ability to produce optimal patterns of ventilation for each clinical situation by variation of mode and parameters such as inflating pressure, tidal volume, rate and the inspiratory : expiratory (I:E) ratio.

iii) Delivery of accurate, predetermined, stable oxygen concentrations from 21 - 100%.

iv) Complete delivery of all preset volumes despite changes in patient's lung characteristics.

v) Minimal circuit resistance and impedance to allow spontaneous breathing modes without incurring excessive work of breathing.

vi) Effective and safe humidification of gases and nebulization of bronchodilator drugs.

vii) Monitoring of respiratory variables and alarm facilities. These two requirements are complementary in modern ventilators for example reliable and sensitive disconnect alarms relates to continuous monitoring of airway pressure and expired volume.

viii) Applications of positive end expiratory pressure (PEEP) and continuous positive airway pressure (CPAP).

ix) Adequate safeguard against cross infection, such as simplicity of sterilization, use of bacterial filters or disposable tubings.

x) Electrical and gas safety features including continuing function in the event of an electrical

or gas source failure; or high pressure relief valves to prevent barotrauma.

xi) Economical to purchase, simplicity of use and maintenance, reliability and robustness.

1.5.2 Driving Mechanism:

All ventilators require a power source or driving mechanism. The driving mechanism may deliver the gas to the patient directly (single circuity) or by way of another mechanism (double circuity) such as ventilator compression of a bag or bellows, which in turn delivers the gas to the patient. The types of driving mechanism used in ventilators may be:

1. Electrical motors which may drive either:
   a) An air compressor, with the gas pressure produced being used to ventilate the patient or
   b) A piston, with the piston drive mechanism being rotary or linear drive.
2. Pneumatic which may be further subdivided into
   a) High pressure, utilizing wall source air or oxygen, or
   b) Low pressure, with wall source gas passing through a reducing valve or a venturi injector.
3. Spring tension driving a bag or bellows which then delivers the gas.
4. Weight on bellows or mass utilized as a generating pressure source.

1.5.3 Control Mechanism:

Every ventilator perform in four phases; inspiratory, inspiratory to expiratory (I to E) cycling, expiratory and expiratory to inspiratory (E to I) cycling. The control mechanism which initiates each phase of ventilation may operate mechanically, electronically, via fluid components, by microprocessors, or by any mixture of these. Microcomputers are being increasingly used by new ventilators to control complex electronic valves and regulators often using servo-controlled (feedback) flow mechanisms.

Panel controls for ventilatory parameters vary from ventilator to ventilator. Gas volumes can be preset by direct control of any two of the three parameters of inspired minute volume, tidal volume and respiratory rate. The duration of inspiration and expiration can be set by any of the following controls:-

1) Direct control of rate with a fixed I:E ratio (such as 1:2).
2) Direct rate control with variation of I:E ratio by altering the inspiratory time. This is accomplished by tidal volume and flow (or volume per unit time) changes. High flows with small tidal volumes will decrease inspiratory time and decrease the I:E ratio.
3) Direct control of inspiratory time (which may also include control of pause time) or both inspiratory and expiratory times by timing mechanisms.

1.5.4. Cycling:

Some ventilators use the same mechanism to initiate both inspiration and expiration, but others use one mode of cycling to end inspiration and another to end expiration.

1.5.4.1 Inspiratory to Expiratory Cycling:

Inspiration can be terminated by the following modes

a) Time cycling. Cycling is controlled by a timing mechanism.

b) Pressure cycling. Cycling occurs when airway pressure reaches a predetermined value.

c) Volume cycling. Expiration is started after a fixed tidal volume has been delivered. Some ventilators which are time or pressure cycled use a double circuit "bellow in bottle" delivery system, with the tidal volume being set by limiting the bellows travel. If the inflating pressure or cycling pressure in these machines is set very high (relative to airway pressure) then the preset tidal volume will always be delivered. Hence, machines when used in this manner are strictly speaking, volume limited or pressure cycled, but may also be referred to as being volume cycled.

d) Flow cycling. Inspiration is terminated when gas flow to the patient falls to a critical preset value. It is an uncommonly used mechanism.

e) Mixed cycling. More than one mode of cycling is possible because of independent cycling mechanisms, or

a variable performance of a single mechanism.

1.5.4.2 Expiratory to Inspiratory Cycling:

Expiratory to inspiratory cycling may use any of the methods terminating inspiration. In addition, there is patient cycling, in which the patient starts to inhale and in doing so, initiates inspiration. This is also called "triggering", and the cycling mechanism depends on the slight negative pressure or gas flow generated by the patient. The sensitivity of triggering mechanism can vary, and there is always a slight delay between sensing the inspiratory attempt and initiating inspiration. Patient triggering is thus usually unsatisfactory at high respiratory rates. Patient cycling also decreases the expiratory time and hence the I:E ratio rises from the preset value.

Triggering is also known as the "assist" mode, and a patient cycled machine can be referred to as an assistor. A ventilator which controls inspiration by time cycling is called a controller. An assist/control mode then allows triggering, but which also initiates mechanical ventilation if spontaneous respiration ceases. Most ventilators allow assist/control as assist only machines are hazardous if the patient fails to initiate spontaneous breathing efforts.

1.5.5.0 Classification:

Classification of ventilators facilitates the understanding of their functional characteristics. The

anaesthetist can then choose the most appropriate ventilator for his needs, use the ventilator optimally, and predict the effect of lung changes on ventilator function. Various classifications have been described (Smallwood, R.W.1986) and they include:

1.5.5.1 Volume or Pressure Preset (Hunter, A.R. 1961):

Volume preset machines can be set to deliver a fixed tidal volume, and do so despite changes in lungs characteristics. Increase in the airway resistance or decrease in compliance will result in an increased airway pressure. Any leaks in the airway will be uncompensated and will result in fall in inspired minute volume.

Pressure Preset Machines generate a preset pressure and Inspiratory to Expiratory cycling occurs when a predetermined airway pressure is reached. Reduced compliance or increased airway resistance will usually result in fall in minute volume (measured as expired volume). Small leaks in the circuit will, however, be compensated for because the pattern of airway pressure remains unchanged. In general pressure preset ventilators are not useful in the ICU. This classification, although simple and useful is no longer adequate.

1.5.5.2 Force Generators (Norlander, O.P. 1964):

Ventilators are classified based on a concept of force, assessed as a product of flow and pressure using a test lung. The two major groups are constant force

generators (Subdivided into those with adjustable or non-adjustable flow) and increasing force generators (subdivided into those with direct or indirect action). This classification is not useful as cycling and some decreasing force generators are ignored.

1.5.5.3 Stable or Flexible Volume and Flow

(Grogono, A.W. 1972).

In this classification, minute volume, tidal volume and inspiratory flow are classified as "stable" if constant values are maintained, and as "flexible" if the values change with changes in the conditions of the patients lungs. However, "stable" characteristics in normal patients may become "flexible" with grossly abnormal lungs, and also modes of cycling are not classified.

1.5.5.4 Flow Pattern, Force and Cycling (Baker, A.B, Muray Wilson, A. 1974):

In this classification ventilators are considered as flow producers. Flow controlled or load independent ventilators can maintain a predetermined inspiratory flow pattern against patient resistance. Flow uncontrolled or load-dependent machines cannot maintain a flow pattern, that is flow is affected by the patient's lung changes. In general flow control is more likely to be achieved by high driving force or generating pressure. An arbitrary generating force of 50mmHg (6.7kPa) was chosen to divide flow-controlled from flow-uncontrolled.

Other characteristics are grouped into force patterns (constant, varying, and adjustable) which in turn, are subdivided into the methods of inspiratory to expiratory cycling.

The division into flow controlled or uncontrolled is dependent upon the generating pressure. Since a high value was selected, this classification is more suited to abnormal lung mechanics. Nonetheless, this classification is useful to predict how a ventilator will perform in a clinical setting, but unfortunately it is not commonly used.

1.5.5.5 Flow or Pressure Generators (Mapleson 1969, Mushin W.W. et al 1980).

This widely used classification considers inspiratory and expiratory flow with their methods of cycling, although clinically, usefulness involves mainly inspiratory flow and Inspiratory to Expiratory cycling.

A flow generator is a ventilator with a high generating pressure, producing an inspiratory flow which is unaffected by patients lung changes. The flow pattern of a flow generator may be constant or non-constant (such as a sine wave flow pattern produced by a rotary drive piston).

A pressure generator has a lower generating pressure to produce an inspiratory flow which can be influenced by changes in lung characteristics. Although the flow pattern may vary, the pressure wave form remains the same, and may

be either constant or non constant (such as increasing or decreasing pressure generators). Both flow and pressure generators are subdivided into methods of cycling.

Although this classification divide ventilators according to the generating pressure produced to drive gas into the lungs, it is actually a spectrum, with transition from one group to the other at some intermediate point. A ventilator behaves like a flow generator when the generating pressure is high enough to disregard patient impedance. A pressure generator may become a flow generator if its generating pressure is increased to exceed alveolar pressure by 10 times. Some ventilators allow control of generating pressure, and these machines may then be used either as flow generators or pressure generators.

1.5.5.6 High frequency ventilation:

High frequency ventilators are not covered in the above classifications. There are three main basic techniques of high frequency ventilation which are in use. They include High Frequency Positive Pressure ventilation (HFPPV); High Frequency Jet Ventilation (HFJV) and High Frequency Oscillation (HFO). The ventilators used in this techniques are as yet to be classified in a clinically useful system.

1.5.5.7 Minute Volume Dividers:

A few ventilators, in addition to be classified into any of the above classifications, can be grouped as "minute

volume dividers" or "flow choppers". The minute volume of gas is preset from the flow meters of the gas source. The tidal volume and inflation pressure are determined, thus deriving the rate. No increase in minute volume can be achieved by the patient, even by triggering the ventilator.

1.5.6 Modes of ventilation:

The practice of artificial ventilation has developed from the use of simple Intermittent Positive Pressure ventilation (IPPV) to include a range of new techniques. These include the use of Positive-End-Expiratory Pressure (PEEP) or Continuous Positive Airway Pressure (CPAP), and the combination of spontaneous breathing and automatic ventilation modes as Intermittent Mandatory Ventilation (IMV), Mandatory Minute Volume (MMV) and High Frequency Ventilation (HFV) (Lin, E.S. and Hanning, C.D. 1990).

1.5.6.1 Patient triggering or Assist/Control mode of ventilation:

This is a system whereby the ventilator begins inspiration only after a spontaneous breath by patient reduces airway pressure and initiates the ventilation cycle. The tidal volume is preset and the patient determines the rate. For safety reasons, ventilators offer only an assist/control mode whereby the ventilator will deliver predetermined breaths if there is no spontaneous breathing by the patient. The theoretical advantage of triggering is to reduce inspiratory pressure and coordinate

patient and ventilator. In practices, hyperventilation and patient exhaustion are commonly seen and triggering is of little use other than to implement weaning or recovery from respiratory failure secondary to central depression (Cameron, P.D. Oh TE. 1986).

1.5.6.2 Pressure support/Assist mode of ventilation:

This is a mode whereby the patient initiates inspiration and the ventilator delivers a breath up to a constant preset positive airway pressure, that is the technique provides a preset airway pressure at the start of inspiration.

Pressure support levels of 5-15cm$H_2$0 (0.5 - 1.5 kPa) are usually used which ceases after a given fraction of inspiratory time or when inspiratory flow falls bellow a predetermined fraction of the initial inspiratory flow rate. Expiration is passive and the pressure support level is gradually decreased as the patient improves. Although there has been isolated case reports, consistent, clear clinical advantages over intermittent mandatory ventilation or vice versa has not been proven (Brochard, L.; Pluskwa, F and Lemaire, F. 1987).

1.5.6.3 Intermittent Mandatory Ventilation (IMV):

This system allow spontaneous breathing between the mandatory ventilator delivered ventilation. The mandatory ventilation is further reduced by a reduction in frequency as the patient ability to increase his own spontaneous

minute volume improves. The spontaneous tidal volume is directly related to the patients effort, whereas with triggering it remains a function of the ventilator. By allowing for some spontaneous breathing IMV offers the following advantages.

a) There is a reduced need for sedation, and muscle relaxants can be avoided.

b) A slower ventilation rate is possible thus allowing for potentially less barotrauma.

c) Venous return via the thoracic pump mechanism is augmented by spontaneous breathing. Cardiac output is less depressed.

d) The weaning process is facilitated, in part by the graduated exercising of respiratory muscles.

Synchronized Intermittent Mandatory Ventilation (SIMV) is a modification whereby the mechanical breaths are provided by triggered ventilation. If no breath is triggered within a predetermined time period a mandatory ventilation breath is automatically delivered. There does not appear to be any clear cut clinical advantages of SIMV over IMV in critically ill patients. (Downs, J.B. et al, 1975).

1.5.6.4 Mandatory Minute Volume (MMV)

This system incorporates a modified circuit so as to deliver a mandatory preset minute volume which is the sum of spontaneous and mechanical ventilation. The patient breathes spontaneously and the ventilator makes up for the

deficit in the minute volume that is mechanical ventilation varies minute by minute, inversely to spontaneous respiration. MMV aids weaning, as the patient improves, his own spontaneous efforts contribute more to the minute volume, and ventilation by the machine automatically, gradually decreases. In contrast to IMV, progressive manual adjustments to the ventilator are unnecessary that is the patient "self weans". However, the MMV circuits are relatively complicated. Spontaneous shallow and therefore ineffective tidal volumes may not be differentiated by the ventilator, which would only sense the total spontaneous minute volume, and thus inappropriately contribute to an inadequate mandatory volume (Cameron, P.D.; Oh, TE. 1986; Hewlet, A.M., Platt. A.S., Terry, V.S. 1977 and Ravenscroft, P.J. 1978).

1.5.6.5 Positive End Expiratory Pressure (PEEP):

PEEP is provided by incorporating an expiratory resistance in the breathing circuit. This can be achieved with a constriction, a spring loaded-valve, a weighted valve, a venturi valve, an electronically controlled scissor valve or a pressure actuated solenoid valve placed in the expiratory part of the circuit. All devices produce some retardation of expiratory flow before the final pressure is reached, with a resultant increase in mean airway pressure.

A more favourable distribution of inspired gas occurs with the use of PEEP. In general PEEP is indicated when an

adequate fractional inspired oxygen concentration (FIO_2) fails to maintain satisfactory oxygenation (e.g. PaO_2 60mmHg or 8kPa or less). It improves oxygenation probably by reducing terminal airway closure and increasing Functional Residual Capacity (FRC) leading to a reduction in physiological dead space (Duncan, A.W., Oh, TE., Hillman, D.R. 1986).The usual range employed is 5 - 15cmH_2O (0.5-1.5 kPa) but higher values have been used.

With the use of higher pressures the fall in cardiac output caused by the raised intrathoracic pressure may counteract the beneficial effects of PEEP on oxygenation (Asbough, D.G., Petty, T.L. 1973). Optimal levels of PEEP can be found with the use of the pulmonary capillary wedge pressure (PCWP); cardiac output, and mixed venous oxygenation tension measurements, and with appropriate use of inotropic agents. However, PCWP readings in the presence of PEEP need to be interpreted with caution (Green, R.T. 1977). When Central Venous Pressure (CVP) and PCWP measurements are not available, lung compliance and the clinical indices of cardiac output may be used to derive an optimal PEEP level (Suter, P.M. Fairley, H.B. Isenberg, M.D. 1975).

1.5.6.6 Continuous Positive Airway Pressure (CPAP):

In this system there is continuous positive airway pressure maintained throughout the respiratory cycle. CPAP or constant pressure breathing allows spontaneous respiration at an elevated baseline pressure when the

ventilator is used in the assist mode. It may also be used with Intermittent Mandatory Ventilation, becoming effective during the spontaneous breathing phase. It has been advocated in a variety of clinical condition, including respiratory distress of the newborn and following cardiac surgery. The physiological lung effects and disadvantages of CPAP are similar to PEEP (Duncan A.W., Oh, TE., Hillman D.R. 1986).

1.5.6.7 High Frequency Ventilation (HFV):

a) High Frequency Positive Pressure Ventilation (HFPPV).

HFPPV delivers humidified gas through an insufflation catheter, bronchoscope, or cuffed endotracheal tube at a preferred rate of 1 - 2 Hz (60-120/min). Adequate alveolar ventilation and arterial oxygen can be achieved with a reduction of peak airway pressure and an improvement in the pulmonary gas distribution. It has been successfully used during laryngoscopy; bronchoscopy; and in adult and neonatal intensive care. The performance of HFPPV may be specific to the device, as reported features refers to a device of small internal volume and static compliance (Crawford, M.R. 1986).

b) High Frequency Jet Ventilation (HFJV)

This is probably the most widely used form of HFV used today. HFJV delivers dry gas under high pressure (about 345 kPa) at rates of 2 -10 Hz (120-600/min), by means of fluidic solenoid, or rotating valve flow controllers. The

driving pressure, frequency and I:E ratio are usually adjustable. Expiration is passive and PEEP may be added. Gas exchange occurs by ordinary convective gas transport, possibly enhanced by some turbulent mixing generated by the jet, as tidal volumes are extremely small at 1.0 - 2.5mls/kg.

Although peak airway pressure is reduced, barotrauma has occurred, since exhalation is dependent on passive lung and chest wall recoil driving gas out of the endotracheal tube. HFJV has been successfully used to treat bronchopleural or tracheal disruption, bronchopleural fistula, ventilator weaning and emergency percutaneous trans-tracheal ventilation (Crawford, M.R. 1986).

c) High Frequency Oscillation (HFO)

High frequency Oscillation uses a piston, loudspeaker or similar device to oscillate at 3 - 20 Hz (180 - 1200/min) and drive a very small volume of gas into the airway and suck an equal volume back out. A steady fresh gas flow at the top of the endotracheal tube provides oxygen and removes carbon dioxide. Alveolar gas exchange is postulated to be due to facilitated diffusion. A higher mean airway pressure can be achieved, but less than peak airway pressure by conventional means. Ventilation perfusion inequalities may be minimized (Butler, W et al 1980).

1.6.0 THE MANLEY MULTIVENT VENTILATOR:

(Penlon Ltd.1993).

This is a gas powered, electronically controlled ventilator, that is designed to operate in world's most challenging medical environment. It is a robust machine, designed to be reliable and very economical to run and provides the users with many unique features and benefits. It can accept AC electrical power supply ranging from 88 volts to 264 volts and is protected from unacceptable power surges by a built in circuit breaker. The internal battery will automatically keep the ventilator running for at least 100 hours if there is a power cut. The battery needs to be recharged for 1 hour to provide a minimum of 50 hours use.

The driving gas can be compressed oxygen or air at 20 to 73 psi and its gas consumption is approximately 1/10th of the minute volume. The ventilator has a flexible approach to gas supply and can be driven by any of the following:

. Compressed oxygen from a standard cylinder or from pipeline supply and the oxygen can be recycled to the patient's circuit to give approximately 33% inspired oxygen.

. Compressed air from a pipeline.

. The Penlon Multivent Power Pack- an air compressor which runs on mains electricity with its own back up battery, that will power the compressor for 24 hours.

. A specially adapted De Vilbis Oxyvent Oxygen Concentrator can supply compressed air to drive the

ventilator while simultaneously delivering oxygen to the patient's circuit.

The ventilator is equally suitable for use with continuous flow anaesthetic machines or draw-over vaporizers such as the Penlon OMV or EMO. It is compatible with non-rebreathing and partial rebreathing circuits such as the Bains, Ayres T-Piece and circle system. This highly versatile ventilator can be used for automatic or manual ventilation of patients from children to large adults in the operating theatre, the intensive care unit or in an ambulance.

The Multivent is available in two versions. The Non-ether compatible version which has the bellows module and the electronic control connected directly together is not for use with inflammable anaesthetic agents. The Ether compatible version can be used with inflammable agents as the electronic control module has been raised above the bellows module.

The controls of the ventilator allow for respiratory rates ranging from 5 to 35 cycles/min., tidal volumes from 100 to 1200mls, maximum inspiratory pressures from 10 to 50cmH_2O and inspiratory/expiratory ratio from 1:1 to 1:6 (Preset to 1:2 on the Ether model).

The inspiratory pressure relief valve is set at 60cmH_2O which is the maximum safe pressure. It has audio and visual alarms for airway pressure; disconnection; and low battery charges. The high airway pressure alarm is set at 58cmH_2O and cannot be muted, the low airway pressure and

disconnection alarm is set at 5cmH_2O and can be muted for 1 minute. The low charge battery alarm allows for a minimum of 1 hour use and can be muted for 4 minutes.

To assist a clinician who is not familiar with the ventilator on how to set the controls rapidly in an emergency, blue spots are marked at the vertical position on the rate; I:E ratio controls on the tidal volume, and the maximum inspiratory pressure scale. Selection for these gives values suitable for an average adult male as follows:

Rate	12 BPM
I:E Ratio	1:2
Tidal Volume	0.6 Litres
Inflation Pressure	20cm H_2O

The controls may then be reset to values to suit the particular patient when a more stable condition exists.

1.6.1 Theory of Operation:

The controls for rate, tidal volume and I:E ratio plus adjustment of the inflation pressure are available and these parameters should be set in accordance with the clinical requirements. For the Ether compatible model there is no I:E ratio control as this parameter is preset at a ratio of 1:2. The rate and I:E ratio are electronically controlled and so are directly set by the controls.

The tidal volume control has an eyebrow scale as it is affected by two variables and is set by electronic timing

which controls the time allowed for the bellows to rise. The tidal volume achieved depends upon the speed at which the bellow is raised, this speed in turn is dependent upon the driving gas pressure and the positioning of the inflation pressure weight. The higher the maximum inflation pressure is set, the slower the speed at which the bellow is raised, and so the lower the achievable rate.

The ventilator is designed to supply a maximum tidal volume of 1.2 litres, at maximum inflation pressure with a driving gas pressure between 20 and 73 psi (140 - 500 kPa). Outside these limits the ventilator will still work, but its performance will be impaired. The I:E ratio is also electronically controlled but for it to be accurately achieved the other parameters should be properly set.

The ideal setting of the ventilator's parameters is that the inflation pressure is adjusted so that the required tidal volume is delivered just in time for the set rate to be achieved, thus keeping the inspiratory pause to a minimum. If the inflation pressure is set too high, or the tidal volume too low, the inspiratory pause will be increased. If the patient circuit is too compliant the ventilator will be unable to maintain the inflation pressure to the patient, reducing the accuracy of the I:E ratio setting. When the inflation pressure is set too low the indicated tidal volume will not be fully delivered.

If the tidal volume is set too high for the patient, the inflation pressure increases once the patient's lung have become fully inflated. The full tidal volume would

not be completely delivered to the patient, but the increase in inflation pressure which results may cause some form of trauma. At very low tidal volumes there may not be sufficient airway pressure to prevent the low pressure alarm from triggering. This may indicate that either the patient circuit is too compliant and needs to be reduced in volume or that the patient is too small to be ventilated by the ventilator.

PART 2: CLINICAL STUDY.

2.1 Introduction.

A study was done to assess the effect of the Manley Multivent ventilator on the cardiopulmonary stability in patients under balanced anaesthesia. The general performance of the ventilator and complications arising from its use were also assessed in this study which was done at Muhimbili Medical Centre operating theatres.

2.2 Materials And Methodology.

2.2.1 Patients:

Patients included in the study were healthy adults (ASA I and II) with relatively normal lung function and no significant cardiovascular disabilities. They were aged between 16 and 65 years and were undergoing surgical operations of the extremities or the trunk (except thoracotomies and head and neck operations). The surgeon's experience and attitudes towards mechanical ventilation and the duration of the operation were also considered for convenience. Sex and weight were not considered important in the choice of patients.

2.2.2 The Multivent:

The Multivent (Ether compatible Model) was used in conjunction with a draw over system comprising of an Oxford Miniature Vaporizer (OMV) for Halothane and an Ambu-E non-rebreathing valve to ventilate the patients. It was run by

compressed oxygen in a cylinder (which was recycled into the patient circuit to increase the FIO_2 of inspired gases to about 33%) and powered by mains electricity or the inbuilt battery.

A 3 metre long Driving gas hose with fittings to suit the oxygen cylinder was used. For the patient breathing circuit a 1 metre long corrugated breathing hose was used, while another 1 metre long corrugated hose with an Oxygen enrichment T-piece was used as a reservoir hose or connection to the draw over vaporizer.

2.2.3 Induction of Anaesthesia:

After obtaining written consent all patients were induced with atropine sulphate 0.6 - 1mg; Sodium thiopentone 4-5mg/kg body wt., followed by 1 - 1.5mg/kg body wt., succinylcholine to facilitate tracheal intubation. All drugs were administered through an intravenous line with normal saline or Ringer lactate solution. Patients were ventilated manually while checking for the correct placement of the endotracheal tube, pressure in the cuff and fixing of the tube. A long acting muscle relaxant; atracurium in a dose of 0.3mg/kg body wt was administered intravenously and the ventilator was switched to automatic ventilation. The OMV was switched on and halothane 1% administered for 15 minutes.

2.2.4 Maintenance of Anaesthesia:

Patients were maintained by automatic mechanical ventilation with oxygen 33%, halothane 0.5% and intravenous Pethidine 50 - 100mg. Additional doses of muscle relaxant atracurium in a dose of 0.15mg/kg body wt, and fluids as well as blood replacements were given to each patient according to their individual requirements.

2.2.5 Monitoring:

Vital signs were monitored throughout the anaesthetic procedure using the standard non-invasive methods. The parameters included the pulse rate; blood pressure; and peripheral oxygen saturation.

An aneroid sphygmomanometer was used to monitor the systolic and diastolic blood pressures which was done initially at intervals of 5 minutes from the beginning of anaesthesia up to 15 minutes after initiation of automatic ventilation. Thereafter it was checked at intervals of 10 minutes. The pulse rate and peripheral oxygen saturation were monitored continuously by a Novametrix pulseoximeter model 511. The adequacy of oxygenation was monitored by peripheral oxygen saturation levels.

The adequacy of anaesthesia was judged by the maintenance of a stable blood pressure and pulse rate at or slightly divergent from (lower or higher) pre-induction levels. Close observation of blood loss and fluid maintenance and replacement was done and appropriate measures taken as the situation dictated.

2.2.6 End of Anaesthesia:

Close to the end of the operation, halothane levels were gradually reduced and stopped completely at the end of skin closure. All patients were reversed by neostigmine 2.5mg preceded with atropine 1mg, and ventilation changed from automatic to manual ventilation as soon as the patient started efforts to breath. Spontaneous respiration was then allowed to resume with intermittent assisted ventilation. Suction and extubation was done and an oropharyngeal airway left in place. Oxygen supplements were given only when indicated as guided by the peripheral oxygen saturation levels and the patient's condition.

2.2.7 Recovery Room:

Routine recovery room patient monitoring was done with every patient. Monitoring included observations on the pulse rate, blood pressure, respiratory rate and peripheral oxygen saturation until the patient was fully awake and stable. Patients were then transferred to their wards and all were reviewed 24 hours after anaesthesia to note any complications or untoward effects of automatic mechanical ventilation.

2.2.8 Recording of data and other relevant Information:

Recordings were made of the patients' age, sex, weight, tidal volume and inspiratory inflation pressures applied, respiratory rate used, duration of anaesthesia, automatic ventilation and surgery, and the vital

parameters. Information concerning the assembling, setting of controls and their accuracy, operation of the bellows and alarms and the general performance of the ventilator during anaesthesia was recorded. Also all complications or critical incidences during anaesthesia and post-operative period were noted. The changes in pulse rate, systolic and diastolic blood pressure were statistically analysed by the 2 way Analysis Of Variance (ANOVA) for F-value.

2.3 RESULTS:

A total of one hundred and two patients were included in the study, fourty seven patients (46.1%) were males and the remaining fifty five (53.9%) were females. Their ages ranged from 15 years to 65 years (Table 1). The type of surgical procedures and ASA status classification for the patients are shown in table 2. General Surgery procedures were 46 (45.1%); Gynaecological procedures 36 (35.3%); and Orthopaedic procedures 20 (19.6%). Sixty four (62.7%) of the patients were ASA I and thirty eight (37.3%) were ASA II.

Peripheral oxygen saturation levels of the patients during anaesthesia are shown in table 3. The lowest saturation levels were of the order of 96%, and the maximum levels ranged between 99% and 100%. Therefore, during anaesthesia all patients maintained saturation levels of 96% and above which indicate good oxygenation. The tidal volumes used during controlled ventilation in mls/kg body weight are shown in table 4, ranged from 6.7ml/kg to 12.5mls/kg body weight. The majority of the patients (97.1%) were ventilated satisfactorily with tidal volumes of between 6.7 mls/kg and 10.5 mls/kg body weight.

The respiratory rates and inflation pressures used in the patients are shown in table 5 and 6 respectively. The respiratory rates used in this study were 12 and 14 cycles/minute. 75 patients (73.5%) were ventilated at a rate of 12 cycles/min and 27 patients (26.5%) at a rate of 14 cycles per minute. Both rates were found to be

satisfactory. The inflation pressures applied ranged between 20 cmH_20 and 35 cmH_20. In most of the patients (91%) the inflation pressures used were either 25 cmH_20 or 30cmH_20 (Table 6).

The results of statistical analysis of pulse rate changes during the procedures are shown in table 7. The differences between the pulse rates at the three points of observations (that is initial pulse rate, Pulse rate at 15 minutes and pulse rate at the end of anaesthesia) were not significant ($P > 0.05$) indicating that the pulse rate was not significantly affected by intermittent positive pressure ventilation.

Table 8 and 9 shows the results of the statistical analysis of the systolic and diastolic blood pressure changes during the procedures respectively. The differences between the systolic and diastolic blood pressure at the three points of observation (that is initial, at 15 minutes and at the end of anaesthesia) were not significant ($P > 0.10$) indicating that the blood pressure was not significantly affected by intermittent positive pressure ventilation.

Information about the general performance of the ventilator during anaesthesia are in table 10. It was easy to set up and operate; reasonably accurate, flexible and safe for use in all stages of anaesthesia that is induction, maintenance and reversal of anaesthesia.

Table 11 shows complications which occurred during the procedures. Two patients had severe bleeding during

surgery (myomectomy and renal cystectomy) with consequent transient hypotension which was corrected by appropriate volume replacement using blood and crystalloid fluids. One patient had a prolonged recovery from anaesthesia for a hip operation. Ventilation was continued manually until she could breath spontaneously. There was no complication associated with the use of the Manley Multivent.

TABLES OF RESULTS:

Table 1: AGE AND SEX DISTRIBUTION

Age Range	Male	Female	Totals	% of Total
15 - 20	2	4	6	5.6
21 - 30	15	7	22	21.6
31 - 40	9	27	36	35.3
41 - 50	11	14	25	24.5
51 - 60	7	3	10	9.8
60 - 65	3		3	2.9
Totals	47(46.1%)	55(53.0%)	102	100

Table 2: PROCEDURE AND ASA STATUS DISTRIBUTION

Procedures	ASA I	ASA II	Total	% of Total
General Surgery	25	21	46	45.1
Gynaecological	23	13	36	35.3
Orthopaedic	16	4	20	19.6
TOTAL	64(62.7%)	38(37.3%)	102	100

Table 3: PERIPHERAL OXYGEN SATURATION OF PATIENTS DURING ANAESTHESIA

Minimum Oxygen Saturation %	No.of Patients	%	Maximum Oxygen Saturation %	No.of patients	%
96	1	1	99	75	73.5
97	15	14.7	100	27	26.5
98	70	68.6			
99	16	15.7			
Totals	102	100		102	100

Table 4: TIDAL VOLUMES (ML/KG) USED IN PATIENTS DURING ANAESTHESIA

Tidal volume range (mls/kg body wt)	No. of Patients	% of Total
6.5 - 8.5	58	56.9
8.6 - 10.5	41	40.2
10.6 - 12.5	3	2.9
Total	102	100

Table 5: RESPIRATORY RATES USED IN PATIENTS DURING ANAESTHESIA.

RR/min	No. of Patients	% of Total
12	75	73.5
14	27	26.5%
Total	102	100

Table 6: INFLATION PRESSURES (cmH_2O) USED DURING ANAESTHESIA

Inflation Pressure cmH_2O	No. of Patients	% of Total
20	6	5.9
25	64	62.8
30	29	28.4
35	3	2.9
Total	102	100

Table 7: STATISTICAL ANALYSIS OF PULSE RATE CHANGES:

Source of Variation	df	SS	MSQ	F	P(value)
Patients	101	10599.00	104.94	0.86	
Periods	2	611.25	305.63	2.5	> 0.05 NS
Residual	202	24771.75	122.63		
Total	305	35982.00			

$\overline{X_1}$ = 98.8)
$\overline{X_2}$ = 99.6) $\pm$ 1.1 (SE)
$\overline{X_3}$ = 96.3)

NS = No significant changes

Table 8: STATISTICAL ANALYSIS OF THE SYSTOLIC BLOOD PRESSURE CHANGES

Source of Variation	df	SS	MSQ	F	P(value)
Patients	101	97400.25	964.36	0.74	
Periods	2	84.50	42.25	0.03	> 0.10 NS
Residual	202	263118.3	1302.56		
Totals	305	360603.05			

$\overline{x_1}$ = 114.5
$\overline{x_2}$ = 115.3 $\pm$ 3.6 (SE)
$\overline{x_3}$ = 114.0

NS = No Significant changes

Table 9: STATISTICAL ANALYSIS OF THE DIASTOLIC PRESSURE CHANGES

Source of Variation	df	SS	MSQ	F	P(value)
Patients	101	44514.38	440.74	1.05	
Periods	2	2	1	0.002 > 0.10 NS	
Residual	202	84730.62	419.46		
Totals	305	129247			

$\bar{x}_1$ = 71.9 NS = No significant change_

x_2 = 71.8 $\pm$ 2.0 (SE)

$\bar{x}_3$ = 71.6

Table 10: OPERATION OF THE VENTILATOR

	EASY/ACCURATE	NOT EASY/ACCURATE
Assembling of Ventilator	102	-
Setting of Controls and their accuracy	102	-
Change from Auto to Manual	102	-

Table 11: COMPLICATIONS ARISING PERIOPERATIVELY

	Complication	No Complication	Totals
Anaesthesia	1	101	102
Automatic ventilation	0	102	102
Surgical	2	100	102

2.4 DISCUSSION:

The assessment of the effects of the Manley Multivent ventilator on the cardiopulmonary stability in patients under balanced anaesthesia was carried out in patients with relatively normal lung function and no significant cardiovascular disabilities. The selection of patients was aimed at minimizing the influence of the patients cardiopulmonary status on the outcome of the study.

The results of the study showed that intermittent positive pressure ventilation (IPPV) by the Manley Multivent has no significant effect on the systolic and diastolic blood pressure during anaesthesia with IPPV. The pulse rate was stable and oxygenation was adequate. Thus the cardiopulmonary stability in the patients under balanced anaesthesia was not significantly affected by controlled ventilation.

Despite of the striking efficacy and safety of controlled ventilation, the method involves considerable deviation from the normal physiological mechanism of respiration (Nunn,J.F. 1966). The deviation is associated with interference with normal physiological processes which may become harmful to a significant degree when controlled ventilation is used improperly or in patient incapable of compensating for the disturbances. One of the dangers of controlled ventilation by IPPV lies on the strain it places on the circulation by impeding blood flow into the right side of the heart (Cournand A. et al 1948; Spalding J.M.K. 1955) During the inspiratory phase of spontaneous

ventilation the fall of intrathoracic pressure sucks air into the lungs and blood from outside the thorax into the great thoracic veins and the heart (Brecher, G.A. 1956). Controlled ventilation interferes with and modifies the subatmospheric pressure changes and disturbs this important mechanism.

The heart is also subjected to a slight positive pressure during the inspiratory phase of controlled ventilation. The resulting cardiac tamponade effect is determined by the inflation pressures applied and the duration of its action.

The inflation pressures used in this study ranged between 20 cmH_20 and 35 cmH_20. In most of the patients pressures of 25 cmH_20 and 30 cmH_20 were used and these were safe and adequate in providing effective ventilation for the patients.

Motley, H.L. (1948) demonstrated that during IPPV using pressures of between 20 cmH_20 and 30 cmH_20, there was no essential change in heart rate, and the changes in systolic, diastolic and mean pressures were of low magnitude. The peripheral venous resistance was raised and the cardiac output reduced. Maloney J.V. et al (1953) confirmed Motley's findings when he showed that cardiac output was lower in IPPV than with negative pressure breathing. The results of this study supports the findings in the previous studies by demonstrating that IPPV is not associated with significant changes in blood pressure and the pulse rate, when inflation pressures of between 20 and

30cmH_2O are applied.

It is clear from these observations that any harmful cardiovascular effects of controlled ventilation results from an abnormally high pressure within the lungs which eliminates in part, or whole, the subatmospheric pressure present within the thoracic cavity. The extent to which these occur depends on the magnitude of the pressure and the duration of action of this pressure which is determined by the inspiratory/expiratory ratio used in the ventilator.

In controlled ventilation the filling of the heart occurs during the expiratory phase when intrathoracic pressure fall to zero, and thus a long expiratory phase is mandatory for adequate venous return to occur. To minimize the harmful effects of the positive intrathoracic pressure the respiratory cycle should have a short inspiratory phase and a long expiratory phase occupying more than half of the cycle. The short inspiratory phase will reduce the duration of the intrathoracic pressure, while a sufficiently long expiratory phase will allow the intrathoracic pressure to fall to zero, abolish the cardiac tamponade effect and hence improve the venous return and cardiac output.

Most ventilators allow for the I:E ratio to be varied between 1:1 and 1:4 (Mushin W.W. 1980). The ventilator in this study used the I:E ratio of I:2 which is the common setting in most ventilators.

The effects of IPPV on cardiac output are related to the mean intrathoracic pressure over a complete respiratory

cycle (Morgan B.C. et al, 1956). An I:E ratio of 2:1 with modest positive pressure of 20 cmH_20 reduced the cardiac output than a higher pressure of 30 cmH_20 with an I:E ratio of 1:2. IPPV with I:E ratio of 1:2 provide optimal ventilation with the least deleterious effects to the circulation (Gordon A.S. 1956).

An inspiration longer than 1.5 seconds provides little useful increase in tidal volume, while a respiratory rate exceeding 16 - 20 cycles per minute in an adult has very little effect on the increase in ventilation (Spalding, J.M.K. 1956).

Respiratory rates of 12 and 14 cycles per minute were used in the study, both were adequate to maintain normal oxygenation as shown by the peripheral oxygen saturation levels ranging between 96 and 100%.

Controlled ventilation with respiratory rate of 12 cycles per minute provide normal oxygenation and elimination of carbon dioxide in most adult patients (Gordon A.S. 1956).

The tidal volumes used in this study ranged from 6.7 mls/kg/body weight to 12.5mls/kg/body weight and were sufficient to provide effective ventilation and normal oxygenation during anaesthesia. Singh, B.B. et al.(1995) using the Manley Multivent in patients under general anaesthesia found that a tidal volume of approximately 10mls/kg/body weight, and respiratory rate of 12 cycles/min. was sufficient to maintain the pulse rate and blood pressure within normal limits. There were no

incidents of cyanosis or ventilatory failure and operative conditions were satisfactory. IPPV in prolonged controlled ventilation is frequently complicated by deterioration in gas exchange because of uneven ventilation and collapse of terminal alveoli. The resulting ventilation perfusion mismatch can lead to insufficient oxygenation of the blood. The collapse of terminal alveoli or atelectasis can be prevented by using larger tidal volumes of 10 to 15 mls/kg and respiratory rates of 10 to 12 cycles per minute to avoid hypocarbia (Hinds, C.J. 1994).

The Manley Multivent was powered by cylinder oxygen which after driving the ventilator, it was ducted back to the patient to provide oxygen supplementation. The ventilator proved satisfactory in providing effective ventilation in the wide range of clinical conditions. By adjusting and regulating the inflation pressure, tidal volume and respiratory rate to suit the requirements of each individual patient it was possible to provide a safe and smooth anaesthesia with adequate oxygenation and good operating conditions in all patients.

The machine was straight forward to set up, easily operated, accurate and could be used safely during induction, maintenance and reversal of anaesthesia. When the power was switched off, the manual control of the bellows was simple to establish using the handle along the bellows arm. There was no complication which could be attributed to the functioning of the ventilator. These results confirm the original findings by Tindiumwebwa

(1992) and Eltrigham (1993), that the performance of the ventilator in a variety of clinical conditions was satisfactory.

Eltrigham, R.J. (1993) showed that it functioned well when used with the cycle system, the Bain system and even in children with an Ayre T-piece system in which the ventilator substituted the reservoir bag and tidal volumes and respiratory rate adjusted according to requirements.

It was also used successfully in the recovery room demonstrating its potential as a versatile machine for both anaesthetic and intensive care situations. The low flow of oxygen (0.6 - 1L/minute) required to drive the ventilator combined with the flexibility of power supply and facility to use it manually makes the ventilator very useful in our situation in which the erratic supply of electricity and shortages of oxygen are not uncommon. The fact that it is less expensive than any comparable ventilator and more versatile over a wide range of clinical situations presents us with an extremely economical method of providing controlled ventilation.

2.5 CONCLUSION:

The Manley Multivent has proved that it can be used safely to provide controlled ventilation in patients under balanced anaesthesia. The cardiopulmonary stability was maintained and no complications were associated with its use. It is economical, versatile easy to operate, and therefore is ideal for use in situations where the supply

of electricity and oxygen are unreliable as found in most of the developing countries. It is the appropriate ventilator for the remotely placed hospitals which are always in short supply of both resources and manpower.

2.6 **REFERENCES:**

1. Ablett, J.J.L. (1956). Tetanus and the anaesthetist. A review of symptomatology and the recent advances in treatment. Br. J. Anaesth. 28, 258.

2. Adams, E.B; Holloway, R; Thombiran, A.K; and Desai, S.D. (1966) Usefulness of intermittent positive pressure. Lancet 2, 901.

3. Adams, W.E (1940): Differential pressures and reduced lung functions in intrathoracic operations. J. Thorac. Surg. 9, 254.

4. Ambiavagar, M; Jones, E.S. and Roberts, D.V. (1967) IPPV in severe asthma. Anaesthesia 22, 134.

5. Asbaugh, D.G. and Petty, T.L. (1973): PEEP-Physiology, indications and contraindications. J. Thorac. Cardiovasc. Surg. 65, 165.

6. Astrup, P; Totzche, H. and Neukirch, F. (1954) Laboratory investigations during treatment of patients with poliomyelitis and respiratory paralysis. Br. Med. J. 1, 780.

7. Baker, A.B and Murray-Wilson, A. (1974): Toward a better classification of lung ventilators. Anaesth. Intens. Care 2, 151.

8. Baker, A.B; Wilson,A. and Hahn, C.E.W.(1974). Alveolar pressure response to top hat gas flow pressure wave in artificial ventilation. Resp. Physiol. 22, 217.

9. Bartlett, R.H; Gazzaniga, A.B. and Geraghty, T.R. (1973) Respiratory manourvers to prevent postoperative complications. J.A.M.A. 224, 1017.

10. Becker, A; Baraka, S; Braun, E. and Myers, M.P. (1960) The treatment of postoperative pulmonary atelectasis with IPPB. Surg. Gynaec. Obstetr. 111, 517.

11. Berstein, A.D; Skowronski, G.A. and Oh TE.(1986) New generation of ventilators. Anaesth. Intens. Care. 14, 293.

12. Bjork, V.O. and Engstrom, C.J. (1955). The treatment of ventilatory insufficiency after pulmonary resection with tracheostomy and prolonged artificial ventilation. J. Thorac. Cardiovasc. Surg. 30, 356.

13. Bjorneboe, M; Ibsen, B; Astrup, P. and Evebert, G. (1955)Active ventilation in treatment of respiratory acidosis in chronic disease of the lungs. Lancet 2, 901.

14. Bower, A.G; Benett, V.R. and Dillon, J.B. (1950) Investigations of the care and treatment of poliomyelitis patients. Ann. West. Med. Surg. 4, 461.

15. Boyle, A.K; Gallie, J.R. and Murray, D.B. (1957) Crush injury of the chest. A report on two cases. Anaesthesia 12, 453.

16. Brecher, G.A.(1953) Venous return during intermittent positive- negative pressure respiration with a new catheter flowmeter. Am. J. Physiol. 174, 299.

17. Brecher , G.A (1956) Venous Return. pg 71 New York : Grune and Starton.

18. Brochard , L. , Pluskwa, F. and Lemaire, F. (1987) Improved efficiency of spontaneous breathing with inspiratory pressure support. Am . Rev. Resp. Dis. 136,411.

19. Butter, W., Bohn, D., Bryan, A. and Froese, A.(1980) Ventilation by high frequency oscillation in humans. Anesth. Analog. 59, 577.

20. Byles, P.H. (1960) Observation on some continuouslyacting spirometters. Br. J. Anaesth. 32, 470

21. Cameron, P.D. and OhTE (1986) Newer modes of mechanical ventilatory support. Anaesth. Intens. Care 14, 258.

22. Clarkson, W.B and Robinson J.S. (1962) Deliberate hyperventilation in the treatment of the crush injury of the chest. Br. J. Anaesth. 34, 471.

23. Crawford, M. R. (1986) High frequency ventilation. Anaesth. Intens. Care. 34, 471.

24. Clutton-Brock, J. (1957) The cerebral effects of over-ventilation. Br. J. Anaesth. 29, 111.

25. Comroe, J.H. and Botelho, S. (1947) Unreliability of cyanosis in recognition of arterial anoxemia. Am. J. Med. Scienc. 214, 1.

26. Cournand, A; Motley, H.L. and Werko, L. (1947) Mechanism underlying cardiac output change during IPPB. Fed. Proc. -Am. Soc. Expt. Biol. 6, 92.

27. Cournand, A; Motley, H.L; Werko,L. and Richard, D.W.(1948) Physiological studies of effects of IPPB on cardiac output in man. Am. J. Physiol. 152, 162.

28. Dobkin, A. B. (1958) Regulation of controlled respiration: recent concepts important to the anaesthetist. Br. J. Anaesth. 34, 502.

29. Downs, J.B.; Klein, E.F; Desantales, D; Modell, J.H and Kirby, R.R. (1975) IMV : a new approach to weaning patients from mechanical ventilators. Chest, 64, 551.

30. Duncan, A.W; Oh TE and Hillman, D.R. (1986) PEEP and CPAP. Anaesth. Intens. Care. 14, 236.

31. Eltrigham, R.J. and Khuwaja, A.(1993) Progress with the Manley Multivent. World Anaesth. Newsletter 10, 130.

32. Ferris, B.G JR.; Mead, J., Whittenberger, J.L. and Saxton, G.A. (1952) Pulmonary function in convalescent poliomyelitis patients. III. Compliance of the lungs and thorax. New England J. of Med. 247, 390.

33. Freier, S., Neal, B. W., Rees, G.J. and Wilson, F. (1957) Salicylate Intoxication treated with IPPR. Br. Med. J. 1, 1333.

34. Galloon, S. and Chakravarty K. (1957) Fat embolism treated with IPPR. Report of three cases. Br. J. Anaesth. 30, 71.

35. Garden, J. and Mackenzie, A.I (1963) The stove - in chest: Another approach to treatment. Br. J. Anaesth. 35 , 731

36. Geddes, I.C. and Gray, T. C. (1959) Hyperventilation for the maintenance of Anaesthesia. Lancet, 2, 4.

37. Glossop , M.W and Low, M.D.W. (1957) Some observation on severe tetanus treated with paralysis and IPPR. Br.J. Anaesth. 29, 326.

38. Gordon, A.S., Frye, C. W. and Langston, H.T. (1956) The cardiorespiratory dynamics of controlled respiration in the open and closed chest . J. Thorac. Surg. 32, 431.

39. Gray, T.C. (1959) A clinical note on the difficulties of maintaining adequate pulmonary ventilation during hypothermia. A symposium on pulmonary Ventilation, pg 43. Altrincham : Sherratt.

40. Green , R.T (1977) Interpretation of Pulmonary artery wedge pressure when PEEP is used. Anesthesiology, 46, 383 .

41. Griffin, S. G., Nattrass, F.J. and Pask , E.A. (1956) Respiratory failure during Thymectomy in a case of myopathy with myasthenia gravis. Lancet 2, 704 .

42. Grimby, G. Hedenstierna, G. and Lowfstrom,B (1975) Chest wall mechanics during artificial ventilation. J. Appl. Physiol. 38, 576.

43. Grogono, A.W. (1972) The classification of intermittent positive pressure ventilator. Br. J. Anaesth. 44, 405.

44. Guedel, A.E. and Treweek, D.N.(1934) Ether Apnoeas. Anaesth. Analg. Curr. Resear. 13, 263.

45. Guedel, A. E. (1940) Cyclopropane anaesthesia. Anaesthesiology 1, 13.

46. Hargrave, S.A., Legge , J.S. and Palmer K.N.V. (1973) Post-Operative ventilatory failure in a patient with primary alveolar hypoventilation. A case report. Br. J. Anaesth. 45, 111 .

47. Harries J. R. and Lower, W.S. (1955) IPPR in bulbo-spinal poliomyelitis. Use of the Radcliffe respiration pump. Br. J. Med. J. 1, 448 .

48. Head, J. (1964) Respiratory failure after thymectomy for myasthenia gravis Ann. Surg. 160, 123.

49. Heidrick, A.F. , Adams, W.E. and Livingstone, H.M (1940) Spontaneous pneumothorax following positive pressure intratracheal anesthesia: a case report. Arch. Surg. 41, 61.

50. Hewlett , A.M., Platt, A.S. and Terry V.G. (1977) Mandatory Minute Volume. A new concept in weaning from Mechanical Ventilation Anasthesia , 33, 246

51. Hinds, C.J. Intensive Care Medicine. In: Kumari, P. and Clark, M. Clinical Medicine 3rd Ed. Baillie're Tindall 1994 pg.709-736.

52. Honey, G.E., Dwyer, B.E. ,Smith A.C., Spalding, J. M.K. (1954) Tetanus treated with tubocurarine and IPPR Br. Med J. 2, 442 .

53. Hubay, C.A., Waltz, R.C., Brecher, G.A. and Hingson R.C. (1954) Circulatory Dynamics of Venous Return during positive-negative pressure ventilation. Anesthesiology, 15, 445 .

54. Hunter, A.R. (1961) The Classification of Respirators. Anaesthesia, 16. 231.

55. Hunter, A.R. (1964) Artificial ventilation of the lungs in combined head and chest injury Lancet, 2, 279.

56. Huston, K.G. (1976) Resuscitation : An historical perspective ASA meeting - 1976 .

57. James, J.L. and Park, H.W.J. (1961) Respiratory failure due to polymyositis treated by IPPR Lancet, 2, 1281 .

58. Kelleher, W.H., Medlock, J.M. and Powell, D.G.B. (1956) Maintenance of respiratory functions in poliomyelitis and other neuromuscular disorders Lancet, 2, 68.

59. Kety, S.S. and Schmidt, C.F (1946) The effects of active and passive hyperventilation on cerebral blood flow, cerebral oxygen consumption, cardiac output and blood pressure of young men. Journ. of Clinical Investigations 25, 107 .

60. Klein, L.J., Saltzman, H.A. , Heyman, A. and Sieker, H.O. (1964) Syncope induced by the valsalva maneurve. American Journal of Medicine. 37, 263.

61. Lassen, H.C.A. (1953) A preliminary report on the 1952 epidemic of poliomyelitis in Copenhagen with special reference to the treatment of acute respiratory insufficiency. Lancet, 1, 37 .

62. Lin, E.S. and Hanning, C.D. Artificial Ventilation of the Lungs In: Nimmo W.S. and Smith, G. Anaesthesia 1st Ed. Oxford. Blackwell Scientific Publications 1990 pg 343-367.

63. List, W.F. (1969) Assisted respiration in fat embolism .Anaesthesia, 18, 215.

64. Macintosh, R.R , Mushin, W.W. and Epstein, H. G. (1963) Physics for the anaesthetist 3rd ed. pg 97 Oxford : Blackwell.

65. Macklin, C.C. (1937) Pneumothorax with massive collapse from experimental local over inflation of lung substance. Canadian Medical Association Journal, 36, 414

66. Manley, R. (1991) A new ventilator for developing countries and difficult locations. World Anaesthesia Newsletter. (15) 1991

67. Maloney, J.V., Elam, J.O., Handford, S.W., Balla, G.A and Ten Pas, R.H. (1953). The Importance of negative pressure phase in mechanical respirators. J.A.M.A 152, 212 .

68. Maloney, J.V. and Handford, S.W. (1954) Circulatory response to intermittent positive and alternating positive-negative pressure respirators. Journ. Appl. Physiol. 6, 453.

69. Marchand, J.F. (1954) Care for respiratory paralysis in poliomyelitis. J.A.M.A. 155, 1297.

70. Marubini, M.B., Rossanda,M. and Tretola, L. (1964) The role of artificial hyperventilation in the control of brain tension during neurosurgical operations. Br. J. Anaesth. 36, 415 .

71. McConnell, D.H., Maloney, J.V. and Pukberg, G.D. (1974) Post-operative IPPB treatments. Journ. Thorac.Cardiovasc. Surg. 68, 944 .

72. Miller, W.F and Sproule, B.J. (1959) Studies on the role of IPPB/I:O_2 in the treatment of pulmonary oedema. Disease of the Chest 35, 469.

73. Misuraca, L. (1966) Mechanical ventilation in Status Asthmaticus New Engl. Journ. Med. 275, 318.

74. Modell, J.H., Calderwood, R.W., Ruiz, B.C. and Down, J.B (1974) Effects of ventilatory patterns on arterial oxygenation after near drowning in sea water. Anaesthesiology, 40, 376.

75. Morgan, B.C., Martin, W.E., Horbein, T.F. and Gunteroth, W.G. (1966). Haemodynamics effects of IPPR. Anaesthesiology, 27, 584.

76. Motley, H.L., Coumand, A., Werko, L., Dresdale, D.T. and Richards D.W. (1948) IPPB; a means of administering respiration in man. J. A.M. A. 137, 370.

77. Mushin, W.W. and Rendell-Baker, L. (1953) Principles of thoracic anaesthesia 1st ed. pg 14 Oxford: Blackwell.

78. Mushin, W.W. and Morton, H.J.V. (1958) Correspondence. Br. Med J. 1, 215.

79. Mushin, W.W., Rendell-Baker, L., Thompson, P.W. (1959) Automatic Ventilation of the lungs 1st ed. Oxford : Blackwell Scientific Publications.

80. Mushin, W.W., Rendell-Baker, L., Thompson, P.W. (1980) Automatic Ventilation of the lungs 3rd ed.pg 2-44 Oxford : Blackwell Scientific Publications .

81. Neff, W., Phillips, W. and Gunn, G. (1942) Anaesthesia for pneumonectomy in man. Anesthesiology, 3, 314.

82. Norlander, O.P., Herzog, P., Norden, I. and Hossh, G. (1968) Compliance and airway resistance during anaesthesia and controlled ventilation Acta Anaesth. Scan. 12, 135

83. Norlander, O.P. (1964) Functional analysis of force and power of mechanical ventilations. Acta. Anaesth. Scan. 8, 57

84. Nosworthy, M.D (1941) Anaesthesia in chest surgery, with special reference to controlled respiration and cyclopropane. Proc. Royal. Soc. Med. 34, 479.

85. Nunn, J.F. (1960) Ventilation Normograms during anaesthesia Anaesthesia 15, 65.

86. Nunn, J. F. and Ezi-Asho T.I (1962) The accuracy of the respirometer and ventigrator. Br. J. Anaesth. 34, 422.

87. Nunn, J. F. (1966) Symposium - Physiological effects of artificial ventilators. Abstracts: Sec.Europ. Cong. Anaesth. -Copenhagen. pg. 86

88. Nunn, J.F. (1969) Applied Respiratory Physiology 1st Ed. pg 126-132 London : Butterworths .

89. Nunn, J.F. (1977) Applied Respiratory Physiology 2nd Ed. pg 94 - 138 London : Butterworths.

90. Pask, E.A. (1953) The maintenance of respiration in Respiratory Paralysis Proc. Royal. Suc. Med. 48, 239.

91 Penlon Ltd. (1993) The Manley Multivent Ventilator - User's Manual.

92. Pontoppidan, H., Hedley - Whyte. J., Bendexin, H.H. and Radford , E. P. (1965) Ventilation and oxygen requirements during prolonged artificial ventilation in patients with respiratory failure. New Engl. Journ. Med. 273 , 401.

93. Rad, L.N. (1970) Vagal activities in canines :a possible connection to the hyperventilation syndrome. Anasth. Analg. Curr. Res. 49, 351.

94. Radford E.P. (1955) Ventilation standards for use in artificial respiration. J. Appl. Phys. 7, 451.

95. Radford, E.P., Ferris, B.G. and Kriete, B.C. (1954) Clinical use of normograms to estimate proper ventilation during artificial ventilation. New. Engl. J. Med. 251, 877

96. Ravenscroft, P.J. (1978) Simple Mandatory Minute Volume. Anaesthesia 33, 246.

97. Redding, J., Voigt, G.C. and Safar, P. (1960) Drowning treated with IPPB. Journ. Appl. Physiol. 15, 849 .

98. Reid, D.H.S. and Tumstall, M.E. (1960) Treatment of Respiratory Distress Syndrome of New-born with nasotracheal intubation and IPPR. Lancet, 1, 1196 .

99. Reid, D.H.S. , Tumstall , M.E. and Mitchell, R.G. (1967) A controlled trial of artificial respiration in the respiratory distress syndrome of the New-born Lancet, 1, 532.

100. Rollason, W.N. and Parkes, J. (1957) Anaesthesia, Hyperventilation and peripheral blood. Anaesthesia, 12, 61 .

101. Roth, L.W. , Whitehead,R.W. and Draper, W.B. (1947) Studies of diffusion respiration II. Survival of the dog following a prolonged period of respiratory arrest. Anesthesiology , 8, 294 .

102. Roy, D.C. and Shapiro, B.A. (1985) Mechanical Ventilatory Support J.A.M.A. 254, 1.

103. Rudy, N.E and Crepean , J. (1958) Role of IPPB post-operatively . J.A.M.A. 167, 1093 .

104. Seeley, H.F. Artificial Ventilation. In: Churchil-Davidson, H.C. A Practice of Anaesthesia 5th Ed. Lloyd-Luke Ltd. 1984 pg 254-294.

105. Singh, B.B; Gautan, M.P; Gurung,A; Chand, M.B.and Shrestha, B.M.(1995) Field trial of the Penlon Manley Multivent and the De Vilbiss Oxygen Concentrator. World Anaesth. Newsletter 14 pg. 4-5. 106.

106. Smallwood, R.W. (1986) Ventilators - reported classification and their usefulness. Anaesthe. Intens. Care 14, 251.

107. Spalding J. M. K. (1955) Pressure and duration of inspiration during artificial respiration by Intermittent positive pressure. Lancet, 1, 1099.

108. Stoddert, J.C. (1967) E.E.G. activity during voluntary controlled alveolar hyperventilation Br. J. Anesth. 29, 2.

109. Suter, P.M., Fairley, H.B., Isenberg, M.D. (1975) Optimum end - expiratory airway pressure in patient with acute respiratory failure. New. Engl. J. Med. 292, 284 .

110. Tindimwebwa, J.V.B. and Mijumbi, C. (1992) Preliminary Experience with the Manley Multivent in Mulago Hospital Uganda World Anaesth. Newsletter (7), pg 5

111. Thompson, P.W. (1966) Classification and function of Automatic Ventilators. Abstract: Second Congress. Anaesth. Copenhagen (1966) pg 118.

112. Thompson, P.W. (1963) Anaesthetic problems associated with certain surgical procedures. Thoracic anaesthesia. 1st ed. pg 350. Oxford : Blackwell.

113. Toland, C. (1972) Treatment of a case of near - drowning in chlorinated water. Anaesthesia, 44, 616

114. Van Lith, P. (1967) Respiratory elastances in relaxed and paralysed states in normal and abnormal man J. Appl. Physiol. 23, 275 .

115. Walker, A.K.Y. and Bethune , D.W. (1976) A comparative study of condenser humidifiers. Anaesthesia 31, 1086 .

116. Wattermath, C.L. (1974) Respiratory Compliance in Obese Patients. Anesthesiology 41, 84.

117. Wasserman, K., Bulter, J and Van Kessel, A.(1966) Factors affecting the pulmonary capillary blood flow pulse in man. J. Appl. Physiol. 21, 890 .

118. Waltz, R.C., Hubay, C.A., Ankeney, J.L. and Merril J. (1954).Experimental Study of pulmonary histopathology following positive and negative pressure respirations Surg. Gynaec. Obstet. 99, 580.

119. Werko, L. (1947) The influence of positive pressure breathing on circulation in man. Acta. Med. Scand. 128 suppl. 193.

120. Wolmer, R. (1956) The Management of Respiratory
ej insufficiency. Anaesthesia 11, 281.

MED.DISS
RD 87
.3C37
.M84
1996

MIAMI MEDICAL UNIVERSITY COLLEGE
LIBRARY
DSM.