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The Study of Hypercapnia

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I. THE CLINICAL USE OF THE MASS SPECTROMETER IN THE STUDY OF HYPERCAPNIA.

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1. Introduction:

A. Problem of carbon dioxide accumulation under general anesthesia.[1-6,18]

In the past 25 years, anesthesiologists have become aware of the necessity for maintaining normal physiology. During this period the increasing complexity of surgical procedures necessitated the development of newer, more potent agents. Both surgical procedure and agent required adoption of closed system methods of anesthetization in order to maintain more even control of anesthetic drug, minimize the quantity of drug used, reduce the hazard of explosion, and provide a quiet surgical field. Operations requiring surgical pneumothorax introduce among other problems the serious effects of reduced tidal volume. The use of the closed system permitted high oxygen tensions but accentuated the problem of hypercapnia. This led to the review and extension of studies on the effects of increased carbon dioxide and the development of methods for the control and detection of hypercapnia.

B. History of preventive measures against carbon dioxide accumulation.[15]

In 1853, Schwann designed the first closed type apparatus using an oxygen supply and lime carbon dioxide absorber for airtight breathing. Benedict, in 1909, did much of his research on human metabolism using sodium hydroxide and lime mixtures for carbon dioxide absorption. In 1915 Jackson[10] applied the carbon dioxide absorption technique to closed system inhalation anesthesia with nitrous oxide and ether vapor for his laboratory animals. The expired gases were forced through an aqueous solution of alkali. This method was applied to clinical anesthesia by Waters[11] in 1923. In 1930 Brian Sword[12] perfected a circle type absorber consisting of a canister connected to a face piece by two tubes. A pair of valves, one at the inlet and one at the outlet of the canister, assured a unidirectional flow of gases. Aside from minor modifications, this arrangement is the most commonly used today.[13, 14]

C. Effects of carbon dioxide[15] and anesthetics[17, 18] on physiology of respiration.

Respiration is controlled reflexly and chemically through the respiratory centers[16a] located in the pons and medulla. These centers give rise to rhythmic impulses analagous to those of the sinoauricular node of the heart. They are influenced by the will, emotional states, chemical agents and peripheral reflexes. Carbon dioxide had a strong stimulating influence on the cells of the respiratory center. A 0.2 per cent rise in alveolar carbon dioxide doubles pulmonary ventilation. When more than 6 per cent carbon dioxide is inspired, no amount of hyperventilation can keep the alveolar air below this figure, so the arterial pCO2 rises to acidemic levels. Because of its role in maintenance of hydrogen ion concentration in the vascular compartments of the body and because carbon dioxide diffuses more slowly within the alveoli than does oxygen, ventilation, which provides enough oxygen to replace oxygen absorbed from the alveolar air, may not be sufficient to remove excess carbon dioxide. In the conscious patient, an accumulation of carbon dioxide results in hyperventilation, and removal of the excess. However, in the anesthetized or hypoxic patient, the respiratory center is depressed and is unable to respond to the chemical stimulation of excess carbon dioxide. During anesthesia the only evidence of excess carbon dioxide may be an elevation of blood pressure. This sign is very often hidden by changes in the homeostasis of circulation due to blood loss and the innumerable reflexes produced

by surgery.

Chemoreceptors are present in the carotid and aortic bodies. When stimulated by mild hypoxia, respiration is increased in rate, depth and minute volume. This response occurs when the oxygen tension falls to 70 mm. Hg. Thus hypoxia directly depresses the respiratory center, but secondarily stimulates respiration via this reflex. Severe hypoxia leads to respiratory failure.

The Hering-Breuer reflex was described in 1868. Afferent impulses travel up the vagus and cause inspiration to be slowed or inhibited when the alveoli are stretched by inflation. Expiration is inhibited when the alveoli are deflated sufficiently. This reflex is important during anesthesia for thoracotomy because collapsing the lung tends to increase the rate and depth of breathing. Heat, cold and pain stimulate breathing; impulses arising in skeletal muscles during active exercise have the same effects. Traction reflexes arising in the abdomen and chest stimulate respiration. Dilatation of the rectal sphincter does the same. Breathing is inhibited during swallowing and by local irritation of the larynx as by strong ether vapor or foreign body. Hypoxia exaggerates the Hering-Breuer reflex, and intense hyperpnea from carbon dioxide excess abolishes this reflex. When respiratory alkalosis is present, the Hering-Breuer reflex is more active and tends to prevent adequate ventilation, which in turn, permits carbon dioxide to accumulate and restore the normal hydrogen ion concentration.

D. Pharmacologic actions of anesthetic agents in relation to hypercapnia.

TABLE I

Pharmacological Effects of Ether, Pentothal and Cyclopropane on the Control of Respiration (after Watrous et al)

	Ether	Pentothal	Cyclopropane
A. On Respiratory Center.			
Stimulus: increased pCO2 of arterial blood. Depression: hypoxia			
1. Response to CO2	Depressed	Depressed Lost in deep anesth.	Accum. c closed sys.
2. Action on center	Incr. min. vol. Center dominant in all planes	Decr. min. vol. Shift of control to chemoreflexes	progressive depres.
3. Vagolytic	Centrally on insp. center	None	Probably at center
4. O2 rich mixtures	No effect	Apnea in deep anes.	Sl. depr. of resp.
B. On Chemoreflexes: Carotid and Aortic Bodies			
Stimulus: Decreased pO2 of arterial blood			
1. Response to hypoxia	Depressed with incr. depth	Not depressed	Depressed CB
C. On Hering-Breuer Receptors and Reflex			
Stimulus: Inflation and deflation of the lung			
1. Action on receptors	Initially more excitable Incr. impulse frequency Lost response in deep planes	Transient decrease impulse frequency	Incr. impulse freq.
2. Action on reflex	Active in light Abolished in deep	Exaggerated	Abolished in deep
D. On Bronchi			
1. Action	Dilatation	Mod. constriction	Constriction
2. Mechanism	Depress s. muscle Vagal paralysis Sympath. stim.	Vagal stimulation	Vagal stimulation

Schmidt[16] stated that the balance between chemical and nervous factors in respiratory control is disturbed by narcotic drugs, but the degree and even the type of disturbance varies according to the drug - perhaps also according to the individual. The single common result, of all narcotics in all patients, is a DECREASE in the sensitivity of the respiratory center to increased carbon dioxide in the blood. This results in a depression of the respiratory minute volume. Deep narcotization of the respiratory center shifts the balance of factors involved in respiratory control to the carotid and aortic reflex system. Large doses of barbiturates and morphine do exactly this. Only cyclopropane is uniformly depressant and evokes no compensatory reflex drive. This gas depresses the response of the center to carbon dioxide and interferes drastically with the effectiveness of reflexes from the carotid and aortic bodies. These reflexes would not be activated in any case because of the high oxygen tension with which cyclopropane is routinely administered. This may explain why paralysis of respiration occurs earlier with cyclopropane anesthesia than with other agents now in use.

When hypoxia becomes the chief respiratory stimulus, with barbiturate anesthesia, a breath of oxygen to eliminate chemoreceptor control will depress respiration or cause a brief apnea. Moyer and Beecher[33] believe that during oxygen apnea or when breathing is depressed because of administration of oxygen during barbiturate anesthesia, carbon dioxide accumulates to high narcotic and possibly fatal levels.

Whitteridge and Bulbring[19] observed that all volatile or gaseous anesthetic agents increase the excitability of the pulmonary receptors which are stimulated by inflation. This increases the frequency of respiratory afferent impulses in the vagus by 30-140 per cent over the usual frequency during similar inflation with air. The sensitization is accompanied or followed by a depression, with all agents, except cyclopropane, proportional to the concentration of the agent. Pentothal exaggerates the Hering-Breuer reflex by reason of producing a pharmacologic decerebration and depression of central control.

2. Purpose and scope of preceding studies on hypercapnea and significant findings.

Intermittent hypercapnia is probably a regular accompaniment of general anesthesia as indicated from reviewing the numerous factors affecting respiratory physiology.[1-7] While acute carbon dioxide excess is not as harmful to the patient as acute hypoxia, its actions may subsequently lead to serious effects. Although concentrations between 5-10 per cent only increase the depth of respiration and raise blood pressure, higher concentrations will cause progressive narcotization and so depression of respiration. Concentration of 30 per cent or more of carbon dioxide is capable of anesthetizing dogs. These animals invariably show a rise in blood pressure and early respiratory stimulation followed by depression.[20,21]

Miller and Brown,[8] in studying the effects of hypercapnia on the dog, found that 30 per cent carbon dioxide caused general anesthesia. If the carbon dioxide concentration was increased by 1 per cent per minute, the dogs regularly suffered respiratory arrest at 60-65 per cent and under controlled respiration went into cardiac arrest at 85 per cent. During the procedure the arterial blood pH fell from 7.4 to 6.4, heart rate slowed from 170 to 70 beats per minute and PR interval on the electrocardiogram was gradually prolonged from 0.09 to 0.12 seconds without evidence of interference in conduction. In another study by the same authors, it was found that dogs, carried on high tensions of carbon dioxide for several hours, succumbed to ventricular fibrillation when the pCO_2 was permitted to diminish rapidly. However, when the carbon dioxide levels were slowly reduced, this phenomenon did

not occur.

The phenomenon of post-anesthetic shock following cyclopropane anesthesia has been the subject of much controversial research.[22,23,24] In detailed intermittent blood studies in eleven patients, Dripps[25] has followed the respiratory depressant effect of cyclopropane throughout anesthesia and into the post operative period. He found the arterial blood pCO2 closely paralleled the tidal volume depression. The blood pressure was directly related to rises and falls in pCO2 during anesthesia. "Cyclopropane shock" was prone to occur when an elevated pCO2 returned to normal post anesthetically.

Numerous workers[34-37] have shown that carbon dioxide tends to accumulate in the blood of conscious subjects when breathing oxygen-rich mixtures.

Moyer and Beecher,[33] found that a minute respiratory volume of air, sufficient to maintain a normal arterial oxygen content in animals anesthetized by pentothal, may be insufficient to effect adequate removal of carbon dioxide.

Several studies have recently appeared regarding the effect of posture on pulmonary ventilation.[26-32] It has been shown that the vital capacity of a conscious patient is modified to a significant extent by changes in position.[29] Similarly, marked respiratory depression during general anesthesia is attributable to extreme positions on the operating table.[30]

Probably the most pertinent studies in the past few years are those of Beecher.[40-43] First he studied the development of acidosis during thoracic surgery on patients undergoing lobectomy and pneumonectomy.[40] He demonstrated that carbon dioxide retention during ether anesthesia was primarily dependent on the patient's position.[43] He also showed that open thoracotomy played a minor role in the production of respiratory acidosis.[42] He contradicted the opinions of Taylor and Roos[39,41] that the rise in carbon dioxide is due to the action of ether in depressing the center. Beecher asserted that ether is a respiratory stimulant in the light anesthetic levels used for thoracic surgery. He also disagreed with Gibbon,[38, 42] who was of the opinion that the accumulation of carbon dioxide was due to the open thorax per se and not due to the position. Roos and Gabbard[44] recently investigated the effects of premedication, assisted respiration, anesthetic agents and the open thorax. They found that patients under unassisted ether anesthesia with an open thorax invariably produced inadequate alveolar ventilating exchange and respiratory acidosis. This could be remedied by manual assistance. In those non-thoracic procedures where light ether anesthesia was sufficient, adequate ventilation and normal blood values were obtained without assistance to breathing. They showed a quantitative correlation between the degree of inadequacy of ventilation and the magnitude of the respiratory acidosis.

From this selective review of the literature it is evident that the studies were hampered by the limitations imposed by biochemical methods of analysis in determining alveolar carbon dioxide tensions. The methods were at best a discontinuous study subject to the usual errors of blood and alveolar gas analysis.

3. Materials and Methods.

At this University, Dr. A. O. Nier[45,46,47] developed a physical method of quantitative gas analysis dependent upon molecular properties. His method enables a high degree of specificity and high analysis speed. Mixed gases can be ionized at low pressures, accelerated by electric and magnetic fields, and spread out in a mass/charge spectrum. Each gas species can then be separated from the others. The positive ion current derived from one mass/charge position measures the concentration of that species in the entering

gas. Since his instrument can be "tuned" over an extremely wide range of mass/charge ratios, all gases can be measured by the instrument after calibration with known mixtures.

Through the collaboration of our workers in Physics, Physiology, Surgery and Anesthesia,[50,51,52] a Portable Mass Spectrometer was constructed and adapted to the problem of studying respiratory physiology in the operating room.**

Preliminary problems confronting us in carrying our clinical studies involved the sampling site, transport of the sample to the Portable Mass Spectrometer, modification in the sample in the analysis, calibration of the analysis and interpretation of the recording. In the "continuous" method used, there is a steadystate flow of gas from the unanalyzed mixtures in the lung alveoli to and beyond the analysis site, i.e. a flow of gas at the input and a flow of corresponding data at the output. A total of five different gases can be quantitated within an accuracy of 1 per cent every 20 seconds.[48-53] The machine thus provides an instrument capable of rapid, continuous automatic quantitative recording of one or several gases in the ventilatory exchanges of an anesthetized patient.

The basic operating features of this instrument, as used at present, consists of a 40 foot flexible copper tube connecting the endotracheal tube of the patient to the Portable Mass Spectrometer. The internal diameter of this tubing is 0.009 inches and is so minute that gases passing through it virtually have a straight wave front, with minimal mixing of the gases in transit. The greatest value recorded during expiration theoretically should represent the alveolar sample if the expired volume is greater than the respiratory dead-space (150-175 cc.). A vacuum pump delivers 0.22 cc. of gas per minute with a lag time from patient to recorded analysis of approximately 20 seconds. jA graduated molecular leak samples 2 cc of the gas stream per day. This is drawn into the highly evacuated U-shaped (180°) spectrometer tube where the gas molecules are bombarded by an electron beam. The positive ions formed are deflected and focused by means of magnetic and electric fields, so that for a given accelerating voltage, controlled by the operator, only positive ions of a specified mass will traverse a slit on to a highly insulated collecting plate at the distal end of the spectrometer tube. The ion currents are proportional to the amount of the given mass present. This is measured on a galvanometer, amplified and recorded on a standard, commercial potentiometer.

Studies to date have been directed mainly to carbon dioxide levels under various controlled anesthetic and surgical conditions.

The initial problem was to demonstrate true correlation between the alveolar pCO2 as recorded on the spectrometer and the arterial blood pCO2 as derived from the Henderson-Hesselbach equation.[54,55,56]

Once this correlation was established, the blood pH could be predicted from the alveolar pCO2 in a true uncomplicated respiratory acidosis.

The following graph represents the parallelism between the alveolar pCO2 recorded on the spectrometer, and the pH and pCO2 of simultaneously drawn arterial blood.

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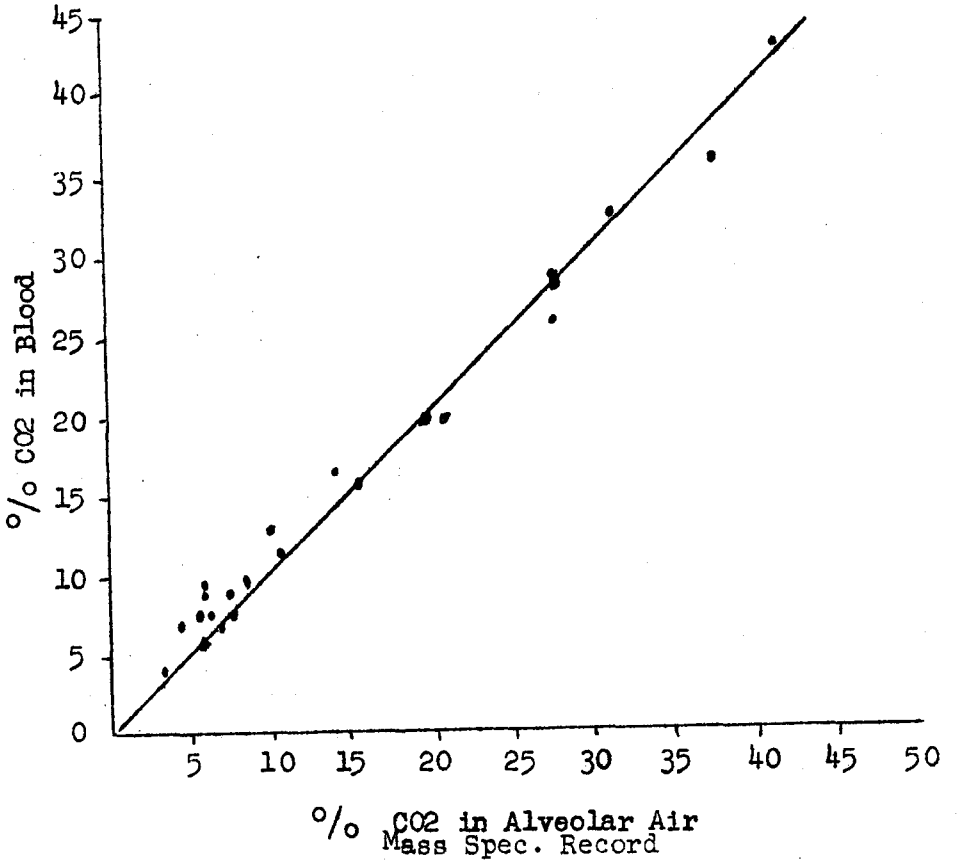

4. Clinical Studies

With the establishment of this parallelism, attention was directed to an evaluation of the known factors predisposing to and causing respiratory acidosis during anesthesia. Our goal is to discover the most ideal methods of maintaining carbon dioxide clearance and controlling the patient's respiratory physiology within normal limits.

TABLE II

Maximum CO2 values recorded by PMS on 34 cases Sample from $\frac{1}{4}$" polyethylene tube (100 cc/min.) (Mixed Inspiratory and Expiratory Gas Samples)							
Procedure	Anesth.	# Cases	<5%CO2	5-6%	6.5-8%	8-10%	>10%
Pneumonectomy	Ether	1	-	-	-	-	1
Lobectomy	PC,N20	2	1	-	-	-	1
Miscell. Thoracotomy	Ether	1	-	1	-	-	-
	PC,N20	12	4	1	3	3	1
Laparotomy	Ether	10	5	2	3	-	-
	PC,N20	5	1	1	3	-	1
Miscell.	Ether	1	1	-	-	-	-
	PC,N20	2	-	1	-	1	-
Maximum CO2 values recorded by PMS on 21 cases Sample from 0.009" copper cable (0.22 cc/min.) (Unmixed Alveolar Gas Samples)							
Procedure	Anesth.	# Cases	5-6%CO2	6-8%	8-10%	10-15%	>15%
Pneumonectomy	Ether	1	-	-	-	-	1
Miscellaneous Thoracotomy	PC,N20	1	-	-	-	1	-
	Ether	1	-	1	-	-	-
Laparotomy	PC,N20	6	1	2	-	2	1
Miscellaneous	Ether	2	2	-	-	-	-
	PC,N20	10	2	2	2	4	-

Miller (Table II) studied unselected cases undergoing surgical procedures with varied anesthetic agents. In the first thirty-four cases, samples were withdrawn from the endotracheal tube via $\frac{1}{4}$ inch polethylene tube, analyzed in the Portable Mass Spectrometer and returned to the anesthetic circuit. Due to mixing of inspired and expired gases, a correction factor had to be applied to the readings, thereby limiting their accuracy. The sampling method was improved at this point. The following 21 cases showed even closer correlation between both Portable Mass Spectrometer alveolar carbon dioxide and that calculated from the blood. These studies clearly demonstrated that respiratory acidosis accompanies practically all general anesthesias.

Subsequently, Buckley et al studied the mechanical limitations of the Portable Mass Spectrometer and its recording system,[53] as well as attempting to sort out the numerous ancillary factors which predisposed to the respiratory acidosis.

Thus far, 66 cases[57] have been studied in an effort to relate hypercapnia to such predisposing factors as position, open thoracotomy, depth of anesthesia and anesthetic agent. These patients were unselected. Their anesthesia was managed with an endotracheal airway and respiration was assisted when their own ventilation appeared inadequate. (Table III and IV)

TABLE III

Maximum CO2 values recorded by PMS on 25 cases undergoing Thoracotomy in Varied Positions

Position	Anesth.	# Cases	<6%CO2	6-7.5%	7.5-10%	>10%
Supine	Ether	6	1	3	1	1
	PC,N2O	1	-	-	-	1
Prone c̄ roll (semi-prone)	Ether	4	-	1	-	3
	PC,N2O	10	-	2	1	7
Lateral	Ether	2	-	-	-	2
	PC,N2O	2	-	-	-	2
Total	Ether	12	1	4	1	6
	PC,N2O	13	-	2	1	10

TABLE IV

Maximum CO2 values recorded by PMS on 41 cases not involving Thoracotomy (Varied Positions)

Position	Anesth.	#Cases	<6%CO2	6-7.5%	7.5-10%	>10%	Remarks
Supine	Ether	13	3	2	5	3	
	PC,N2O	15	-	4	3	8	
Prone c̄ rolls	Ether	-	-	-	-	-	Laminectomy cases
	PC,N2O	3	-	-	1	2	2 long 6" rolls in
Prone s̄ rolls	Ether	-	-	-	-	-	anterior axillary
	PC,N2O	1	-	-	-	1	line for freedom of chest motion
Full lateral Kidney rest up Lat. flexion	Ether	-	-	-	-	-	Patient's head and feet lower than operative site
	PC,N2O	7	1	1	2	3	
Lithotomy	Ether	1	-	1	-	-	
	PC,N2O	1	-	1	-	-	
Totals	Ether	14	3	3	5	3	
	PC,N2O	27	1	6	6	14	

This study demonstrated, unequivocally, the much higher occurrence of respiratory acidosis in patients undergoing surgical pneumothorax. During the conduct of these cases, it was demonstrated, that most of the patients could be adequately cleared of carbon dioxide in the supine and lateral positions by assisted respiration. Semiprone cases were more difficult to aid. Some cases did not respond despite all apparent favorable factors.

The non-thoracotomy cases also pointed out the effects of extreme positions on the operating table as predisposing to ventilatory depression.

Another current study has been directed to correlating the occurrence of hypercapnia during surgery with the resistant postoperative hypotensive states which have been specifically ascribed to anesthetic agents - especially cyclopropane. In 24 cases (Table V) under light

TABLE V

Preliminary Report on Maximum CO2 Values Recorded on PMS and Circulatory Effects of Cyclopropane Anesthesia

Case	Age	Operation	Anes. Time	MAX.%CO2 Duration	SYST.BP MAX.RISE of Surgery	POSTOPERATIVE MAX.FALL S & D BP	DURATION LOW B.P.	REMARKS
A. Respiration Unassisted								
1-7	-		-	8.5-18.0	37 mm Hg	65 mmHg	-	
555	16M	Nerve repair	270	13.0	44	34S-80D	19hrs	Anuria 28 hrs.
561	19F	Ing. Hernia	180	20.0	32	30S-16D	18	
446	42F	Saph.v.lig.	180	6.0	30	34S-66D	6	Shock stat po
USV	64M	Ing. Hernia	115	12.5	63	52S-24D	16	Shock 5hrs.po
478	22F	Breast Biopsy	120	9.0	30	28S-80D	32	
503	64M	Umb&Ing Hernia	225	8.5	30	46S-10D	23	
USV	32M	Ing. Hernia	150	11.5	30	82S-55D	21	Shock 1 hr.po
B. Respiration Assisted								
"	42	Ing. Hernia	375	7.5	20	8S-10D	2hrs	
"	12	Ing. Hernia	95	6.5	10	4S-0	0	
"	30	Saph.v.strip.	135	6.0	0	0	0	
"	77	Cystostomy	120	6.5	0	54S-16D	16	Gradual Fall
"	66	Mastectomy Rad.	150	6.5	0	20S-0	30	500 cc blood
	66	Leg Amputation	315	7.5	50	35S-14D	12	Trans.Reaction
	36	Saph.v.strip.	215	7.5	16	18S-20D	3	Position change
	38	Ing. Hernia	240	5.5	70	0	0	
	67	Leg Amputation	165	5.5	40 Fall	52S-30D	8	Blood Loss high
	83	Ing. Hernia	225	4.5	60	0	0	

cyclopropane anesthesia, we attempted to control the effects of blood loss and trauma by selecting only minor surgical procedures. All patients were intubated prior to commencement of the operative procedure and the alveolar carbon dioxide analysis was continuously recorded. The cases were divided into two groups. In the first group respirations were assisted. The recorder was placed near the anesthesiologist and alveolar pCO2 was kept normal to subnormal thruout the case. In the second group respirations were unassisted and alveolar pCO2 was allowed to accumulate. Frequent intermittent arterial blood pressure readings were taken by the oscillometric method with a cuff applied over the brachial artery. At the conclusion of the surgical procedure, the anesthesia machine was disconnected and the patient then allowed to breathe room air while the endotracheal tube and spectrometer sampling tip were left in place. In this way alveolar carbon dioxide tensions could be recorded during

emergence from anesthesia, at least to the point where the patient could no longer tolerate the orotracheal tube. Blood pressure readings were frequently recorded during the immediate postoperative period and for about 12 hours thereafter.

Of the 14 cases who were allowed to breath spontaneously, 13 showed a significant elevation of alveolar carbon dioxide tension ranging from 8.5 - 20 per cent with an average of 12 per cent. These cases likewise showed a uniform blood pressure response to hypercapnia, exhibiting an average elevation above their preoperative levels of 37 mm. Hg systolic.

Upon cessation of the anesthesia, most of the cases exhibited a prompt and sometimes alarming drop in pressure. Several were delirious, cold and clammy and with weak pulses. No pulse irregularities were noted clinically.

In 10 cases the alveolar carbon dioxide tensions were maintained within the normal range by manual assistance to respiration. Several were found to have significantly elevated alveolar carbon dioxide levels when the Portable Mass Spectrometer was connected. The situation was remedied immediately through assisted respiration.

Aside from the complicating surgical factors that occurred, there was no significant change in the blood pressure during the conduct of surgery or during the postoperative period. These patients all had a smooth recovery period and were easier to manage than the unassisted group.

Our findings to date indicate that the cyclopropane shock syndrome may be explained by the effects of hypercapnia and its rapid postoperative elimination. The underlying basis for the respiratory acidosis noted in these patients was the depression of tidal exchange during anesthesia. It also showed that hypercapnia causes relative hypertension during surgery and the rapid elimination of carbon dioxide postoperatively causes a hypotension which persists for many hours.

5. Conclusion:

The Portable Mass Spectrometer has provided us with a physical means for analyzing alveolar gas concentrations with great speed and sensitivity enabling us, not only to diagnose the pathological process at its inception, but also to remedy the condition almost as soon as it arises. It has given us some insight into the controversial factors causing hypercapnia and has aided us in evaluating the effects of assisted respiration. Our results to date are in full agreement with the findings of Watrous et al, who advocate that the minute volume of respiration be kept up to (or slightly above) normal by assisted or controlled respiration during general anesthesia administered by a closed or semiclosed system. This eliminates the depressant effect of all anesthetic agents and premedicating sedatives on the carbon dioxide sensitivity of the respiratory center, and so avoids hypercapnia and hypoxia and their sequelae.

6. Summary:

1. Review of the problem of hypercapnia during general anesthesia.

2. Brief history of preventive measures against carbon dioxide accumulation during closed system rebreathing.

3. Review of the relationship of carbon dioxide accumulation to the physiological and pharmacological control of the respiratory system.

4. Significant findings of previous studies of hypercapnia.

5. Physical vs. chemical approach to the study of alveolar pCO_2.

6. Development of the Portable Mass Spectrometer and problems encountered

in its clinical application to the study of hypercapnia.

7. Review of clinical applications of the Portable Mass Spectrometer to the problem of hypercapnia during various anesthetic and surgical situations.

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II. MEDICAL SCHOOL NEWS

Coming Events

April 7-9	Continuation Course in Surgery for General Physicians
April 8	George E. Fahr Lectureship; "Coarctation of the Aorta," Dr. Robert E. Gross, Ladd Professor of Children's Surgery, Harvard Medical School, and Surgeon-in-Chief, Children's Hospital, Boston; Owre Amphitheater; 8:15 p.m.
April 14-19	Continuation Course in Proctology for General Physicians
April 17-19	Continuation Course in Obstetrics for Specialists
April 18	Duluth Clinic Lectureship; "Newer Concepts for Fetal Circulation," Dr. S.R.M. Reynolds, Carnegie Laboratory, Baltimore, Maryland; Owre Amphitheater; 8:00 p.m.
April 21-23	Continuation Course in Pediatrics for Specialists
April 21	Clarence M. Jackson Lecture; "Respiratory Diseases -- Changing Concepts," Dr. John M. Adams, Professor of Pediatrics, University of California, Los Angeles; Museum of Natural History Auditorium; 8:15 p.m.

* * *

Continuation Course in Proctology

Dr. Garnet W. Ault, Professor, Department of Proctology, Georgetown University School of Medicine, Washington, D.C., will be our distinguished visiting faculty member for the continuation course in Proctology to be held at the Center for Continuation Study from April 14 to 19, 1952. The course is intended primarily for physicians engaged in general practice and as in past Proctology courses, all aspects of ano-rectal disease will be considered. Two half-days will be devoted to operative clinics in which the registrants for the course will take an active part.

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Faculty News

Dr. Forrest Adams, Assistant Professor, Department of Pediatrics, has been appointed Associate Professor of Pediatrics, University of California Medical School in Los Angeles. On July 1, 1952, he will assume his new position in which he will be associated with Dr. John Adams, formerly Associate Professor of Pediatrics at the University of Minnesota Medical School, who is now Professor and Head of the department at the Los Angeles institution. Dr. Forrest Adams' friends and colleagues join in congratulating him on this much deserved recognition. We are sure that sunny California will prove an excellent setting for his continued clinical and research activities.

At the annual meeting of the American Association of Anatomists, held March 19 to 21, in Providence, Rhode Island, the University of Minnesota Medical School was represented by Doctors Edward A. Boyden, Lemen J. Wells, Berry Campbell, J. Francis Hartmann, W. Lane Williams, and Mr. Dennis J. Kane, Mrs. Maria Ryzen, and Mr. Richard H. Swigart.

Dr. David T. Imagawa, Instructor, Department of Bacteriology and Immunology, recently gave a paper entitled, "Cytotoxic Studies on Mouse Mammary Cancer," at the meeting of the Second National Cancer Congress in Cincinnati, Ohio.

III. UNIVERSITY OF MINNESOTA MEDICAL SCHOOL
WEEKLY CALENDAR OF EVENTS

Physicians Welcome

March 31 - April 5, 1952

Monday, March 31

Medical School and University Hospitals

9:00 - 9:50 Roentgenology-Medicine Conference; L. G. Rigler, C. J. Watson and Staff; Todd Amphitheater, U. H.

9:00 - 10:50 Obstetrics and Gynecology Conference; J. L. McKelvey and Staff; W-612, U. H.

10:00 - 12:00 Neurology Rounds; A. B. Baker and Staff; Station 50, U. H.

11:30 - Tumor Conference; Doctors Kremen, Moore, and Stenstrom, Todd Amphitheater, U. H.

12:15 - Obstetrics and Gynecology Journal Club; Staff Dining Room, U. H.

1:30 - 2:30 Pediatric-Neurological Rounds; R. Jensen, A. B. Baker and Staff; U. H.

4:00 - Pediatric Seminar; Carbohydrate Metabolism in Scurvy; C. Stewart; Sixth Floor West, U. H.

4:30 - 5:30 Dermatological Seminar; M-346, U. H.

4:30 - Public Health Seminar; 15 Owre Hall.

5:00 - 6:00 Urology-Roentgenology Conference; C. D. Creevy, O. J. Baggenstoss, and Staff; Eustis Amphitheater.

Minneapolis General Hospital

7:30 - Fracture Grand Rounds; Dr. Zierold; Sta. A.

10:30 - 12:00 Tuberculosis and Contagion Rounds; Thomas Lowry; Station M.

11:00 - Pediatric Rounds; Franklin H. Top; 7th Floor.

12:30 - Surgery Grand Rounds; Dr. Zierold; Sta. A.

1:00 - X-ray Conference; Classroom, 4th Floor.

1:30 - Pediatric Rounds; Robert Ulstrom; 4th Floor.

Veterans Administration Hospital

9:00 - G. I. Rounds; R. V. Ebert, J. A. Wilson, Norman Shrifter; Bldg. I.

11:30 - X-ray Conference; Conference Room; Bldg. I.

2:00 - Psychosomatic Rounds; Bldg. 5.

3:30 - Psychosomatic Rounds; C. K. Aldrich; Bldg. I.

Tuesday, April 1

Medical School and University Hospitals

9:00 - 9:50 Roentgenology-Pediatric Conference; L. G. Rigler, I. McQuarrie and Staff; Eustis Amphitheater, U. H.

9:00 - 12:00 Cardiovascular Rounds; Station 30, U. H.

12:00 - 1:30 Selected Topics, Permeability and Metabolism; Nathan Lifson; 129 Millard Hall.

12:30 - 1:20 Pathology Conference; Autopsies; J. R. Dawson and Staff; 102 I. A.

4:00 - 5:00 Pediatric Rounds on Wards; I. McQuarrie and Staff; U. H.

4:30 - 5:30 Clinical-Medical-Pathological Conference; Todd Amphitheater, U. H.

5:00 - 6:00 X-ray Conference; Presentation of Cases by Mt. Sinai Hospital; Dr. Friedman; Eustis Amphitheater, U. H.

Ancker Hospital

8:00 - 9:00 Fracture Conference; Auditorium.

1:00 - 2:30 X-ray-Surgery Conference; Auditorium.

Minneapolis General Hospital

8:00 - Pediatric Rounds; Spencer F. Brown; 5th Floor.

10:30 - 12:00 Medicine Rounds; Thomas Lowry and Staff; Station F.

11:00 - Pediatric Rounds; Erling S. Platou; 7th Floor.

Veterans Administration Hospital

7:30 - Anesthesiology Conference; Conference Room, Bldg. I.

8:30 - Infectious Disease Rounds; Dr. Hall.

8:45 - Surgery Journal Club; Conference Room, Bldg. I.

9:00 - Liver Rounds; Drs. Nesbitt and MacDonald.

9:30 - Surgery-Pathology Conference; Conference Room, Bldg. I.

10:30 - Surgery Tumor Conference; Conference Room, Bldg. I.

1:00 - Surgery Chest Conference; T. Kinsella and Wm. Tucker; Conference Room, Bldg. I.

2:00 - 2:50 Dermatology and Syphilology Conference; H. E. Michelson and Staff; Bldg. III.

3:30 - 4:20 Autopsy Conference; E. T. Bell and Donald Gleason; Conference Room, Bldg. I.

Wednesday, April 2

Medical School and University Hospitals

8:00 - 8:50 Surgery Journal Club; O. H. Wangensteen and Staff; M-109, U. H.

8:00 - 9:00 Roentgenology-Surgical-Pathological Conference; Norman Jacob and L. G. Rigler; Todd Amphitheater, U. H.

11:00 - 12:00 Pathology-Medicine-Surgery Conference; Medicine Case; O. H. Wangensteen, C. J. Watson and Staff; Todd Amphitheater, U. H.

5:00 - 5:50 Urology-Pathological Conference; C. D. Creevy and Staff; Eustis Amphitheater, U. H.

5:00 - 6:00 Vascular Conference; Todd Amphitheater, U. H.

5:00 - 7:00 Dermatology Clinical Seminar; Dining Roo , U. H.

7:00 - 8:00 Dermatology Journal Club; Dining Room, U. H.

8:00 - 10:00 Dermatological-Pathology Conference; Review of Histopathology Section; R. Goltz; Todd Amphitheater, U. H.

Ancker Hospital

8:30 - 9:30 Clinico-Pathological Conference; Auditorium.

3:30 - 4:30 Journal Club; Surgery Office.

Minneapolis General Hospital

8:00 - Pediatric Allergy Rounds; Lloyd Nelson; 4th Floor.

10:30 - 12:00 Medicine Rounds; Thomas Lowry and Staff; Station D.

11:00 - Pediatric Rounds; Franklin H. Top; 7th Floor.

12:00 - Surgery Seminar; Dr. Zierold; Classroom.

12:30 - Pediatric Staff Meeting; Meningococcal Disease; Robert Disenhouse; 4th Floor Annex.

12:30 - EKG Conference; Boyd Thomes and Staff; 302 Harrington Hall.

1:30 - Pediatric Rounds; E. J. Huenekens and Robert Ulstrom; 4th Floor.

Veterans Administration Hospital

8:30 - 10:00 Orthopedic X-ray Conference; Conference Room, Bldg. I.

8:30 - 12:00 Neurology Rehabilitation and Case Conference; A. B. Baker.

2:00 - 4:00 Infectious Disease Rounds; Conference Room, Bldg. I.

Wednesday, April 2 (Cont.)

Veterans Administration Hospital (Cont.)

4:00 - 5:00 Infectious Disease Conference; W. Spink; Conference Room, Bldg. I.

7:00 p.m. Lectures in Basic Science of Orthopedics; Conference Room, Bldg. I.

Thursday, April 3

Medical School and University Hospitals

8:00 - 9:00 Vascular Rounds; Davitt Felder and Staff Members from the Departments of Medicine, Surgery, Physical Medicine, and Dermatology; Heart Hospital Amphitheater.

9:00 - 11:50 Medicine Ward Rounds; C. J. Watson and Staff; E-221, U. H.

11:00 - 12:00 Cancer Clinic; K. Stenstrom and A. Kremen; Todd Amphitheater, U. H.

1:30 - 4:00 Cardiology X-ray Conference; Heart Hospital Theatre.

3:30 - Medicine-Pediatric Infectious Disease Conference; Heart Hospital Auditorium.

4:00 - 5:00 Physiology-Surgery Conference; Todd Amphitheater, U. H.

4:30 - 5:20 Ophthalmology Ward Rounds; Erling W. Hansen and Staff; E-534, U. H.

5:00 - 6:00 Radiology Seminar; Osteopoikilosis, Bernard Halper; Marble Bones, Jack Friedman; Eustis Amphitheater, U. H.

7:30 - 9:30 Pediatric Cardiology Conference and Journal Club; Review of Current Literature 1st hour and Review of Patients 2nd hour; 206 Temporary West Hospital.

Minneapolis General Hospital

8:00 - Pediatric Rounds; Spencer F. Brown; 5th Floor.

8:30 - Neurology Rounds; William Heilig; 4th Floor.

11:00 - Pediatric Rounds; Erling S. Platou; 7th Floor.

1:00 - Fracture-X-ray Conference; Dr. Zierold; Classroom.

Veterans Administration Hospital

8:00 - Surgery Ward Rounds; Lyle Hay and Staff; Ward 11.

9:15 - Surgery Grand Rounds; Conference Room, Bldg. I.

11:00 - Surgery Roentgen Conference; Conference Room, Bldg. I.

Friday, April 4

Medical School and University Hospitals

8:30 - 10:00 Neurology Grand Rounds; A. B. Baker and Staff; Station 50, U. H.

9:00 - 9:50 Medicine Grand Rounds; C. J. Watson and Staff; Todd Amphitheater, U.H.

10:30 - 11:50 Medicine Rounds; C. J. Watson and Staff; Todd Amphitheater, U. H.

10:30 - 11:50 Otolaryngology Case Studies; L. R. Boies and Staff; Out-Patient Department, U. H.

11:45 - 12:50 University of Minnesota Hospitals Staff Meeting; Malignancy of the Tonsil; Dale Parshall and Karl Stenstrom; Powell Hall Amphitheater.

1:00 - 2:50 Neurosurgery-Roentgenology Conference; W. T. Peyton, Harold O. Peterson and Staff; Todd Amphitheater, U. H.

2:00 - 3:00 Dermatology and Syphilology Conference; Presentation of Selected Cases of the Week; H. E. Michelson and Staff; W-312, U. H.

3:00 - 4:00 Neuropathological Conference; F. Tichy; Todd Amphitheater, U. H.

3:30 - 4:30 Advanced Neurophysiology Seminar; E. Gellhorn; 111 Owre Hall.

4:00 - 5:00 Dermatology Seminar; W-321, U. H.

5:00 - Urology Seminar and X-ray Conference; Eustis Amphitheater, U. H.

Ancker Hospital

1:00 - 3:00 Pathology-Surgery Conference; Auditorium.

Minneapolis General Hospital

11:00 - Pediatric Rounds; Franklin H. Top; 7th Floor.

11:00 - Pediatric-Surgery Conference; Dr. Wyatt, Forrest Adams; Classroom, Sta. I.

12:00 - Surgery-Pathology Conference; Dr. Zierold, Dr. Coe; Classroom.

1:00 - 3:00 Clinical Medical Conference; Thomas Lowry; Classroom, Station M.

1:30 - Pediatric Rounds; Robert Ulstrom; 4th Floor.

Veterans Administration Hospital

10:30 - 11:20 Medicine Grand Rounds; Conference Room, Bldg. I.

1:00 - Microscopic-Pathology Conference; E. T. Bell; Conference Room, Bldg. I.

1:30 - Chest Conference; Wm. Tucker and J. A. Meyers; Ward 62, Day Room.

3:00 - Renal Pathology; E. T. Bell; Conference Room, Bldg. I.

Saturday, April 5

Medical School and University Hospitals

7:45 - 8:50 Orthopedic X-ray Conference; W. H. Cole and Staff; M-109, U. H.

9:00 - 10:30 Pediatric Grand Rounds; I. McQuarrie and Staff; Eustis Amphitheater.

9:00 - 11:50 Medicine Ward Rounds; C. J. Watson and Staff; Heart Hospital Amphitheater.

9:15 - 10:00 Surgery-Roentgenology Conference; L. G. Rigler, J. Friedman, Owen H. Wangensteen and Staff; Todd Amphitheater, U. H.

10:00 - 11:30 Surgery Conference; Todd Amphitheater, U. H.

10:00 - 12:50 Obstetrics and Gynecology Grand Rounds; J. L. McKelvey and Staff; Station 44, U. H.

Minneapolis General Hospital

8:00 - Pediatric Rounds; George Lund; 5th Floor.

11:00 - 12:00 Medical-X-ray Conference; O. Lipschultz, Thomas Lowry, and Staff; Main Classroom.

11:00 - Pediatric Clinic; C. D. May and Floyd Denny; Classroom, 4th Floor.

Veterans Administration Hospital

8:00 - Proctology Rounds; W. C. Bernstein and Staff; Bldg. III.

8:30 - Hematology Rounds; P. Hagen and E. F. Englund.