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Electrophrenic Respiration

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G. KEITH STILLWELL, M.D., Research Fellow, and

FREDERIC J. KOTTKE, M.D., Associate Professor and Director,

Division of Physical Medicine
University of Minnesota Medical School

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Address communications to: Staff Bulletin, 3330 Powell Hall, University of Minnesota, Minneapolis 14, Minn.

I. ELECTROPHRENIC RESPIRATION

G. K. Stillwell
F. J. Kottke

The cessation of spontaneous respiration is a major emergency, the management of which may be thrust upon any of us at any time. The problem resolves essentially into obtaining an adequate patent airway and promoting the exchange of an adequate volume of respiratory gases through it, without at the same time jeopardizing the life of the patient through deleterious side-effects of the method employed, or through waiting too long before starting. Wiggin, Saunders, and Small[1] point out that the medical profession has often tended to relegate these procedures to lay personnel who lack the educational background to judge the medical situation. They mention the calls to lay rescue squads from operating and delivery rooms as a particular example of this defection.

(I) Methods of Artificial Respiration

Many methods have been devised to achieve satisfactory artificial respiration, but the efficiency of some of these leaves much to be desired. It is particularly regrettable that the Schafer method, which has probably been most widely taught to the lay population, is probably also the least efficient of the various manual methods of artificial respiration. The Eve method (which enjoyed some vogue in the English-speaking navies in World War II and embodies the principle used in the rocking-bed) is almost as inefficient in producing a sufficient tidal volume.

It is now felt that the only reliable way to measure the efficacy of a method of artificial respiration is to employ it on a subject in whom voluntary respiration, and preferably consciousness, is actually suspended. Cordier[2], reviewing the evaluations of the Schafer, Silvester and Eve methods previously published, showed the variability of the results in subjects with voluntary suspension of respiration and the total inadequacy of the Schafer method applied to the non-rigid cadaver (Table 1). He also drew attention to the fact that, in subjects with voluntary suspension of respiration, the tidal volume obtained was nearly identical to that obtained with voluntary respiration at the same rate by the same subject. The inefficiency of the Schafer method he thought to be due to the fact that the diaphragm and chest wall in death are already in the expiratory position and that positive intracardiac pressures of 9 to 13 cm. of water interfered with cardiac filling.

Gordon, Raymon, Sadove and Ivy[3] also found rather striking differences between the results obtained with the various methods of manual artificial respiration when applied to non-rigid warm corpses, totally apneic normal adults (rendered thus with curare and anesthesia) or to normal subjects with passive suspension of respiration. They suggest that there is more elastic recoil in the living chest in the young adult, and that "the smaller values attained with warm corpses are probably due to total absence of reflex activity and a non-functioning circulatory system, whereas the greater values in normal passive subjects reflect the inability to completely suppress normal respiratory mechanism and the desire to breathe." Asmussen and Nielsen[4] studying normal adults, found similar results with the Schafer method, but good ventilation with the Eve method. It seems likely, as Ivy et al suggest, that the administration of artificial respiration to recently deceased subjects prior to the onset of rigor mortis more nearly approaches the usual emergency situation.

Schwerma et al[5] resuscitating acutely asphyxiated dogs found the manual methods gave no better results than were obtained with no respiratory assistance, while mechanical resuscitation gave a significantly higher survival rate. Blood and D'Amour[6], working with rats found positive pressure devices and the barospirator to be better than the Schafer and Eve methods. They also observed that fever may be a critical factor in survival from hypoxia, the increased metabolic demand for oxygen aggravating the effect of the asphyxia.

Table I.

Tidal Volumes Obtained by Various Methods of Artificial Respiration on Various Types of Subjects. (c.c.)

Author and Type of Subject	Schafer Method	Silvester Method	Eve Method	Schafer-Emerson-Ivy Method
1. Voluntary Suspension of Respiration				
Committee on Suspended Animation 1903*	366	458		
Schafer 1904*	520	178		
Ploman 1906*	500-600	1200-2000		
Eve 1932*	1000			
Killick & Eve 1933*	350-550	200	400-830	
Gordon 1950 (3)	810	1529	1106	1649
2. Post-hyperventilation Apnea				
Liljestrand 1913*	170	190		
Hederer 1934*	100-150	250-280		
Gordon 1950 (7)	1000	1800	950	2070
3. Anesthesia, etc., Apnea				
Waters & Bennet 1936*	180-662 (F) (M)	214-933 (F) (M)		
Gordon 1950 (3)	500	1145	751	1199
4. Non-rigid Cadaver				
Bruns 1927*	20	200		
Bruns & Thiel 1930*	20	200		
Hederer 1934*	40-50	200		
Cordier 1943 (2)	20	200		
Motley 1948 (21)	50-140		50-60	
Gordon 1950 (3)	185	520	300	530

*From Cordier, reference #2, Table 1.
Numbers in parentheses refer to bibliography.

A recent review of the problem for the American Medical Association by Gordon, Fainer and Ivy[7] showed that with fresh human cadavers the prone rocking method (Eve) gave 42% and the prone pressure method (Schafer) gave 35% of the ventilation obtainable by other less-renowned manual methods. The Emerson method, consisting of lifting the hips some 4 inches from the ground to promote inspiration (a "pull" method), was shown to be more effective than the Schafer method (a "push" method). A combination of this with the Schafer procedure (Schafer-Emerson-Ivy method - a "push-pull" method) was as efficient as any of the manual methods employed, giving a nearly normal respiratory pattern with a tidal volume of some 500 cc. It has the advantage of requiring (a) only a small modification of a method already familiar to many of the population, (b) only one person to administer it, and (c) no apparatus. The importance of the last mentioned factor should not be overlooked, since the survival time without respiration is notoriously short and usually does not permit the assembling of equipment for resuscitation, even in a hospital.

Many devices have been conceived to cause the exchange of respiratory gases by causing the pressure within the respiratory tract to be periodically greater than the pressure acting upon the thoracic wall from without. Among them are the Burns valve, or Pneumatic Balance Resuscitator (Pneophore) which is actuated by the pressure of the inflowing gas and cycles automatically. Being automatic and compact, it was popular with the army air forces and widely investigated by them. It has also been used to some extent in civilian resuscitation. Gordon, Fainer and Ivy[7] found it would produce a tidal volume of 185 cc. with a minute volume of 4625 cc., when the line pressure was 14.7 mm. Hg. A "suck and blow" resuscitator, operating with pressures of plus 14 and minus 9 mm. Hg., gave a tidal volume of 216 cc. and a minute volume of 4536 cc. The tank type of respiratory, commonly employed in this hospital for respiratory paralysis in poliomyelitis, and the "portable" chest respirators (such as the Monaghan) which we have also employed, belong fundamentally in the same group of intermittent positive pressure breathing devices.

Artificial respiration by electrical stimulation of the phrenic nerve was first performed in 1793[8]. Resuscitation of dogs who had suffered respiratory arrest from excessive chloroform was reported in 1855. Several instances of its use in humans ahd accumulated by 1866.[9] The inhibition of spontaneous respiratory efforts by an afferent vagal reflex was reported in 1936.[10] Aside from these scattered instances, electrophrenic respiration was not used to any extent prior to the reports of Sarnoff and his colleagues which began to appear in 1948[11] The method consists of percutaneous stimulation of the phrenic nerve as it lies in the neck on the anterior surface of the scalenus anterior muscle just behind the sternocleidomastoid muscle. The "indifferent" electrode is customarily placed behind the shoulder on the same side. Any suitable frequency of impulse may be used which will cause a sustained contraction of the diaphragm. Frequencies below 10 per second will tend to make it flutter. If the stimulus is gradually built up to a maximum the respiratory pattern will be more nearly normal and more comfortable for the patient, should he be perceiving sensory stimuli from his respiratory apparatus.

Briefly, the investigations of Sarnoff and his colleagues have shown:

1. that the ventilation varies with the peak voltage applied, within reasonable limits[12],

2. that the spontaneous minute volume and blood gas concentrations can be duplicated with submaximal stimulation of one phrenic nerve, and hyperventilation alkalosis can be produced with stimulation of one phrenic nerve[12],

3. that spontaneous respiration is suppressed if adequate ventilation is provided by electrophrenic respiration, apparently by the Hering-Breuer (vagal) mechanism[13,14],

4. that approximately normal ventilatory ratios between the two lungs are maintained during electrophrenic respiration applied to one phrenic nerve. It has been observed fluoroscopically that the mediastinum shifts toward the stimulated side, while the diaphragm on the unstimulated side moves up[15],

5. that the deleterious effects of positive pressure breathing in patients without peripheral sympathetic reflexes, or in shock, do not occur with electrophrenic respiration and may be alleviated by it[16],

6. that it is relatively easy to learn to administer electrophrenic respiration.

(II) Influence of Respiration on Circulation

A. Normal Respiration

With quiet normal respiration there are only minor fluctuations in blood pressures in the systemic and pulmonary circulations. With a slight increase in the depth of respiration these changes increase.[17] The changes in intrapleural pressure are imposed upon the exterior of the heart and affect particularly its diastolic pressure. With inspiration the pressure gradient between the peripheral veins and the right auricle increases. This leads to an increased venous return to the right heart and an increase in diastolic volume and in stroke volume. The increase in the pulmonary vascular bed more than compensates for the increase in right ventricular output, so that the pulmonary venous flow to the left heart is decreased and a fall in left heart stroke volume and in systemic blood pressure occurs. The decrease of intrathoracic pressure on the thoracic aorta is also thought to contribute to the lowering of systemic pressure. In expiration the opposite changes occur. Because of the simultaneous changes occurring in the capacity of the pulmonary vascular bed and output of the right heart, the respiratory fluctuations are only partially reflected by the left heart. Lauson suggests that the lungs may be thought of as a buffer between the right ventricle and the left ventricle to aid in maintaining a constant output from the left ventricle.

The work of Thompson and his associates[18,19] demonstrated that the various maneuvers of artificial respiration will promote the flow of blood in the presence of cardiac standstill as long as the blood is kept fluid. With a suck-and-blow resuscitator, the pre-asphyxial levels of oxygen saturation in the arterial blood was obtained in about 15 minutes. The venous oxygen content did not rise in any instance.

B. Positive Pressure Respiration

Positive pressure breathing has been divided into continuous and intermittent positive pressure breathing. In continuous positive pressure breathing inspiration is passive and expiration active. With intermittent positive pressure breathing both inspiration and expiration tend to be passive, but varying amounts of active expiration may be required, depending upon how low the pressure falls in the expiratory phase. With intermittent positive pressure breathing roughly 50% of the mean mask pressure is transmitted to the right heart[20] and the mean mask pressure with the Pneumatic Balance Resuscitator is about 40% of the line pressure.

The major physiological defect in intermittent positive pressure breathing is that the intrathoracic pressure is increased in inspiration, leading to a decrease in filling of the right heart and a decrease in its output. For three or four beats the left heart filling is increased and its output is increased. If the inspiratory portion of the cycle is too great, or if the intrathoracic pressure in the expiratory phase stays too high or drops too slowly, the opportunity for filling of the right heart and the pulmonary vascular bed will be poor and the increase in output of the left ventricle during the inspiratory phase correspondingly reduced.[21] While the ultimate effect of intermittent positive pressure breathing will vary with the pressures and the type of cycle employed,

the use of a pressure of about 20 cm. water at ground level caused reductions in cardiac output from 15 to 25% in normal individuals.[22,23,20] Barach[24] states that with voluntary hyperventilation there is a decrease in stroke volume but it is accompanied by an increase in pulse rate, while with passive hyperventilation the pulse rate fails to compensate.

Maloney and Whittenberger[25] commenting on the body (tank) respirator, point out" .... close scrutiny of the mechanics of the respirator indicates that it produces respiration by pressure relationships which are mechanically and physiologically identical with those produced by applying positive pressure by mask to the patient's face, except for the negligible air density difference. During the time of no air flow at the peak of inspiration the pressure within the alveolus is greater than the pressure outside the chest wall by an amount equal to the positive or negative pressure applied." They state that in subjects with circulatory instability due to hemorrhage, barbiturate poisoning, medullary paralysis, etc. the body respirator may produce severe circulatory depression. This, they suggest, can be eliminated by the use of a positive as well as a negative intratrank pressure phase, counting on the rigidity of the chest wall to render intrathoracic pressure changes less than those occurring in the abdomen and limbs. However, with "suck-and-blow" resuscitators, Motley et al[21] stated that it was of no apparent advantage to include negative pressure in the mask cycle.

Table II shows the effect of pressure breathing on the circulation in individuals with an essentially normal circulatory system. It may be noted that the subject compensates in intermittent positive pressure breathing with a sufficient elevation of peripheral venous pressure to maintain the effective right heart filling pressure. It is the individual with a peripheral vascular collapse, or whose peripheral vessels can respond no further to a decreasing blood volume or left heart output, who fails to maintain this right heart filling gradient during inspiration. This sets up a vicious cycle leading to rapid deterioration with intermittent or continuous positive pressure breathing.

Table III summarizes the findings of various groups on the effects of pressure breathing on cardiac output. The data found related to intermittent positive pressure breathing are scant and no figures were found for its effects on subjects in shock. The data from Beecher demonstrate nicely the progressive deterioration of a dog in shock on continuous positive pressure breathing.

C. Electrophrenic Respiration

The effects of electrophrenic respiration on the circulation are similar to the effects of normal respiration. Sarnoff, Maloney, and Whittenberger[16] compared the effects of intermittent positive pressure breathing by manual compression of a bag and electrophrenic respiration on dogs with the entire sympathetic nervous output blocked and in one human with spinal anesthesia up to T3. They found that if the subject was kept in the horizontal or Trendelenburg position, death did not occur from circulatory collapse alone but it did occur if the circulation was subjected to the added embarrassment of intermittent positive pressure breathing. They point out also that the effects of intermittent positive pressure breathing on the circulation grow worse as one attempts to increase the tidal volume and that the rate is limited. They concluded that "it is clear that when the sympathetic pathways are intact permitting of full compensatory vascular activity, intermittent positive pressure breathing has little or no depressor effect. The same type of intermittent positive pressure breathing applied in the presence of sympathetic blockade, depresses the circulation." Finally, they demonstrated that the adverse effects of intermittent positive pressure breathing under these conditions were alleviated by electrophrenic respiration.

(III) Use of Electrophrenic Respiration in the University Hospitals

Table II.

Effect of Pressure Breathing on Circulation in Man with Essentially Normal Circulatory System

Type of Respiration	Phase	Intra-Pulmonic Pressure	Peripheral Venous Pressure	Right Auricular Pressure	Vein to Right Auricle Gradient	Source
Normal	Inspiration	-1.5	7	-4	11	Best & Taylor (29)
	Expiration	+3.0	8	-3	11	Best & Taylor (29)
Continuous Positive Pressure Breathing 20 cm. H_2O	Inspiration	20	23.6	19.9	3.7	Barach (23)
	Expiration	25*	23.6	25.4	-1.8	Table V
Intermittent Positive Pressure Breathing 20 cm. to 0 cm.	Inspiration	20	15	+4	11	Adapted from Barach (23)
	Expiration	0	8	-3	11	

All pressures in cm. H_2O with reference to atmospheric.
Numbers in parentheses refer to bibliography.
* Estimated.

Table III.

Effects of Pressure Breathing on Cardiac Output

Source	Subject	Type of Positive Pressure Breathing	Pressure Used	* % Changes in Cardiac Output	Remarks
Humphreys (30) 1937	Dog	Intermittent	6.5-2	-46	Average of 9 experiments. Range +8 to -79%
		Continuous	20	-18.4 to -74.3	
Beecher (3) 1943	Dog	Continuous	10	-9	Normal dog
		Continuous	10	-19	Hemorrhagic shock
		Continuous	10	-47	After 10 minutes with shocked dog
Molomut (22) 1946	Man	Continuous	20	-12 to -24	At ground level
		Continuous	20	-9 to +24	At 46,000 feet
Motley (20) 1946	Man	Continuous	20	-14	At ground level
		Continuous	20	-10	At 25,000 feet
Barach (24) 1947	Man	Continuous	20	-26	
		Intermittent	20-0	-15	

* From that with spontaneous respiration.
Pressures in cm. of H_2O with reference to atmospheric.
Numbers in parentheses refer to bibliography.

The experiences at the University Hospitals with electrophrenic respiration have been small. At the time of writing the method has been used in two moribund patients.

Case No. 1, ... A 4-year old girl who fell from a moving automobile at 10:00 a.m. on 9/22/50. She arrived at the hospital in coma with a systolic blood pressure of about 55 mm. Hg. X-rays revealed a skull fracture from the vertex to the foramen magnum. The skull was trephined. Shortly after her arrival at the hospital spontaneous respiration ceased and she was maintained on intermittent positive pressure breathing by manual compression of a rebreathing bag by the anesthesiologist. At 1:30 p.m. on 9/23/50 electrophrenic stimulation was begun with a manually interrupted stimulator. At 4:00 p.m. she was changed to an automatically cycling electrical stimulator. The respirations appeared to be deep and smooth, but measurements of tidal or minute volumes were not made. She had a rather severe acidosis with a CO_2CP of 24-28 which may have been on a respiratory basis.

On 9/24/50 a loose connection necessitated resumption of intermittent positive pressure breathing for about two hours. At the end of this time she was thought to have expired, no heart sounds being audible. Electrophrenic respiration was then resumed, partly to see how long the phrenic nerve would respond after circulatory arrest. However, within five minutes the heart sounds were audible again and her blood pressure a few hours later was 64/30.

At 3:00 p.m. on 9/26/50 she was tracheotomized so that the endotracheal tube inserted on 9/22/50 could be removed. She seemed to tolerate the procedure well and electrophrenic respiration was resumed after surgery. However, about one hour later she deteriorated rapidly and expired at 4:20 p.m. Autopsy showed marked cerebral edema and pulmonary edema.

The development of increasing amounts of edema in the neck during the last 24 hours of her life made it increasingly difficult to stimulate the phrenic nerve without also stimulating the brachial plexus. She also had developed some superficial necrosis of the skin of the motor points, particularly on the left side, where an acid electrode solution had been employed during the first 12 hours.

Comment: The experience on 9/24/50 would seem to indicate that intermittent positive pressure breathing was depressing the circulation in this patient towards extinction. The rapid and sustained improvement after resumption of electrophrenic respiration appeared to be a verification of the experiment reported by Sarnoff et al[16]. The cause of her sudden exodus after the apparently well-tolerated tracheotomy was not known

Case No. 2, - a 15-year-old boy with a diffuse brain tumor whose respirations ceased a few hours after attempted needle biopsy of the brain. He was maintained on electrophrenic respiration for a little more than 28 hours, at the end of which time he was returned to intermittent positive pressure respiration during surgery. Following this the phrenic nerve would respond only once or twice in succession and then required a period of rest of two or three minutes. At this time his blood pressure had been unobtainable for about 5 hours but the heart sounds were still audible. He was then fitted with a Monaghan chest respirator but death followed shortly. Autopsy showed marked pulmonary edema, small brain hemorrhages and considerable autolysis of the brain, although it had been "fixed" shortly after death.

Comment: It was thought that the failure of the phrenic nerves to respond to stimulation in the terminal stages was due to hypoxia, secondary to failure of the peripheral circulation or failure of gaseous exchange in the lungs or both.

(IV) Potential Uses of Electrophrenic Respiration in this Hospital.

A. Traumatic Brain Lesions

Cases such as Case No. 1 cited above, but perhaps with less damage and a better prognosis would seem to be well adapted for the use of electrophrenic respiration and the accessibility of the patient for nursing procedures, etc. are also points of advantage over the tank type or chest type respirator.

B. Postoperative Respiratory Failure with Circulatory Collapse

It would seem likely that cases of this type might do better on electrophrenic respiration than intermittent positive pressure breathing, though the recent use of Arterenol[26] has largely met the problem of maintaining the systemic blood pressure in these cases.

C. Asphyxia Neonatorum

Once a clear airway has been obtained, it would seem likely that expansion of the lungs could be obtained as well with electrophrenic respiration as by any of the other methods available. It is true that it suppresses spontaneous respiratory activity but this disadvantage (if it is one) would be offset by the salutory effects on the circulation with resulting better oxygenation of the vital centers, so that spontaneous respiratory activities might be more apt to occur than after a corresponding period of intermittent positive pressure breathing. Sarnoff cites one instance in which electrophrenic respiration was administered to a newborn infant for 10 minutes with subsequent spontaneous respiration.

D. Poliomyelitis

Where the phrenic nerve is sufficiently intact to conduct the impulse, electrophrenic respiration may be used. It has been reported[27] to be satisfactory in those patients with non-functioning respiratory centers, where the patient can breathe well when reminded to do so but breathes infrequently and poorly otherwise. It is also reported to be useful in suppressing irregular and inefficient respirations which, in some patients, prevent satisfactory ventilation with the tank type respirator. Usually, after the patient has been ventilated for a while with the electrophrenic respirator, his drive for spontaneous respiration is reduced to the point where the tank type respirator can function without interference by spontaneous respiratory efforts.

E. First Aid

Drownings, electrocutions and similar accidents are few and far between in this hospital and, as mentioned previously, should generally be treated by a "push-and-pull" method of manual artificial respiration as a first aid method. When some apparatus is to be substituted, the electrophrenic respirator should be at least as good as the "suck-and-blow" resuscitators and much easier of application than such equipment as the Drinker respirator.

Respiratory failure with cyanide poisoning or with the newer "nerve" war gases would not be likely to respond to electrophrenic respiration. It might respond to manual or other methods.

Caution: The Sanborn Electrophrenic Respirator is not accepted by the Council on Physical Medicine and Rehabilitation for use in the presence of explosive gases. While it is probably safe so to use it, it has not met the requirements of the Council. An all-electronic spark-free instrument has recently been described by Severinghaus[28].

References

1. Wiggin, S. C., P. Saunders, and G. A. Small.
Resuscitation.
New Eng. J.Med., 241:370-378; 413-420, (Sept.), '49.

2. Cordier, D. G.
Methods of Artificial Respiration.
Brit.Med.J.2:381-383 (Sept.), '43.

3. Gordon, A. S., F. Raymon, M. Sadove and A. C. Ivy.
Manual Artificial Respiration. Comparison of effectiveness of various methods on apneic normal adults.
J.Am.M.Assn.,144:1447-1452, (Dec.),'50.

4. Asmussen, E. and M. Nielsen:

Efficacy of Artificial Respiration. J.Applied Physiol.,3:95-102 (Aug.), '50.

5. Schwerma, H., A. C. Ivy, Burkhardt, W. L. and Thometz, A. F. Resuscitation from Obstructive Asphyzia. Am.J.Physiol. 156:145-148 (Feb.),'49.
6. Blood, F. R., and D'Amour, F. E. Efficiency of Various Types of Artificial Respiration at High Altitudes. Am.J.Physiol.156:52-61 (Jan.) '49.
7. Gordon, A. S.; Fainer, D.C. and Ivy, A. C. Artificial Respiration. A new method and a comparative study of different methods in adults. J.A.M.A. 144:1455-1464 (Dec.) '50.
8. Editorial: From "Animal Magnetism" to Electrophrenic Respiration. New Eng.J.Med.,242:340-341 (March), '50.
9. Sansom, A. E. Chloroform: Its Action and Administration. Pp.279--, Phila., Lindsay and Blakiston, 1866.
10. Brigenti, A. Arch.Fisiol. 36:36-47, '36. Cited in ref. 8 and 14.
11. Sarnoff, S.; Hardenbergh, E. and Whittenberger. Electrophrenic Respiration. Science 108:484 (Oct.), '48.
12. Sarnoff, S. J., Hardenbergh, E., and Whittenberger, J. L. Electrophrenic Respiration. Am.J.Physiol.,155:1-9 (Oct), '48.
13. Sarnoff, S. J., Whittenberger, J. L. and Hardenbergh, E. Electrophrenic Respiration. III. Mechanism of the inhibition of spontaneous respiration. Am.J.Physiol.,155:203-207 (Nov.),'48.
14. Chatfield, P. O. and Sarnoff, S. J. Role of Pulmonary Proprioceptive Reflexes in Suppression of Spontaneous Breathing during Electrophrenic Respiration. Am.J.Physiol.163:118-124, (Oct.),'50.
15. Sarnoff, S. J., Gaensler, E. A. and Maloney, J. V., Jr. Electrophrenic Respiration. IV. The effectiveness of contralateral ventilation during activity of one phrenic nerve. J. Thorac. Surg., 19:929-937 (June), '50.
16. Sarnoff, S. J., Maloney, J. V., Jr. and Whittenberger, J. L. Electrophrenic Respiration. V. Effect on the circulation of electrophrenic respiration and positive pressure breathing during the respiratory paralysis of high spinal anesthesia. Ann. Surg.,132:921-929 (Nov.), '50.
17. Lauson, H. D., Bloomfield, R. A., and A. Cournand. The Influence of the Respiration on the Circulation in Man. With special reference to pressures in the right auricle, right ventricle, femoral artery and peripheral veins. Am.J.Med.,1:315-336, (Oct.), '46.
18. Thompson, S. A., Quimby, E. H., and Smith, B. C. The Effect of Pulmonary Resuscitative Procedures Upon the Circulation as Demonstrated by the Use of Radioactive Sodium. Surg., Gyn. and Obst., 83:387-391, '46.
19. Thompson, S. A. and Rockey, E. E. The Effect of Mechanical Artificial Respiration Upon Maintenance of the Circulation. Surg., Gyn. and Obst., 84:1059-1064, '47.
20. Motley, H. L., A. Cournand, Eckman, M. and Richards, D. W.,Jr. Physiologic Studies on Man with the Pneumatic Balance Resuscitator, "Burns' Model". J.Av.Med.,17:431-461, '46.
21. Motley, H. L., Cournand, A., Werko, L., Dresdale, D. T., Himmelstein, A. and Richards, D. W., Jr. Intermittent Positive Pressure Breathing. A means of artificial respiration in man. J.A.M.A. 137:370-382 (May),'48.
22. Molomut, N. and Allen, S. C. Effect of Pressure Breathing on Circulation at High Altitude as Measured by the Ballistocardiograph. J.Av.Med.,17:357-363, '46.
23. Barach, A. L., Eckman, M., Ginsberg, E., Rumsey, C. C., Jr., Korr, J., Eckman, I. and Besson, G. Studies on Positive Pressure Respiration. I. General aspects and types of pressure breathing.

II. Effects on respiration and circulation at sea level.
J.Av.Med.,17:290-320, '46.

24. Barach A. L., Fenn, W. O., Ferris, E. B., and Schmidt, C. F.
The Physiology of Pressure Breathing. A brief review of its present status.
J.Av.Med.,18:73-87, (Feb.), '47.

25. Maloney, J. V., Jr., and Whittenberger, J. L.
Circulatory Effects of the Body Respirator.
Am.J.Med.,8:393 (March), '50. (Abstract)

26. Littman, A., Grossman, M. I., Gunnar, R. M., Isaacs, J. H., Hirschmann, J. H. and Foley, E. F.
Electrocardiographic Effects of Arterenol and Isopropylarterenol in Man, with a Note on the Auricular T Wave.
J.Applied Physiol.,3:235-242 (Nov.), '50.

27. Sarnoff, S. J., Maloney, J. V., Jr., Sarnoff, L. C., Ferris, B. J. and Whittenberger, J. L.
Electrophrenic Respiration in Acute Bulbar Poliomyelitis. Its use in management of respiratory irregularities.
J.A.M.A. 143:1383-1390 (Aug.), '50.

28. Severinghaus, J. W.
Electrophrenic Respirator.
Anesthesiology, 12:123-128 (Jan.), '51.

29. Best, C. H. and N. B. Taylor.
The Physiological Basis of Medical Practice.
Williams and Wilkins, Baltimore, pp. xiii and 1169, Ed. 4.

30. Humphreys, G. H., Moore, R. L., Maier, H. C. and Apgar, V.
Studies of the Cardiac Output of Anesthetized Dogs during Continuous and Intermittent Inflation of the Lungs.
J.Thoracic Surg., 7:438-451, 1937-'38.

31. Beecher, H. K., Bennett, H. S., and Bassett, D. L.
Circulatory Effects of Increased Pressure in the Airway.
Anesthesiology 4:612-618 (Nov.),'43.

II. MEDICAL SCHOOL NEWS

Coming Events

May 7-11 Continuation Course in Electrocardiography for General Physicians
May 8 George E. Fahr Lecture; "Certain Mechanical Peculiarities of the Heart," George E. Burch, Tulane University School of Medicine; Medical Science Amphitheater.
May 14 Minnesota Multiphasic Personality Inventory
May 16 Special Lecture; "Investigations on the Assimilation of Amino-Acids by Bacteria," Ernest F. Gale, Cambridge University, England; Museum of Natural History Auditorium; 8:00 p.m.
May 16 Journal Lancet Lecture; "Brain Mechanism and Behavior," Heinrich Kluver, University of Chicago; Medical Science Amphitheater; 8:00 p.m.
May 17 Student Day and Medical Six O' Clock Dinner

Dr. Burch Will Present First George E. Fahr Lecture

Dr. George E. Burch, Professor and Head of the Department of Medicine, Tulane University of Louisiana, will give the first George E. Fahr Lecture on Tuesday evening, May 8. The Lectureship, which was made possible by gifts from former students, patients, and friends of Dr. Fahr, will become an annual event and will bring to our campus each year an outstanding lecturer in the field of cardiovascular disease. Dr. Burch, who is well known for his many contributions in this field, has selected for the subject of the Fahr Lecture, "Certain Mechanical Peculiarities of the Heart." All interested physicians and the public are invited.

During his visit to our campus, Dr. Burch will also participate in a continuation course for physicians in Electrocardiography. The course will be presented at the Center for Continuation Study May 7-11 and will emphasize active interpretation of electrocardiograms under the supervision of an instructor. Dr. Harry E. Ungerleider, Medical Director of the Equitable Life Assurance Society, New York City, will also participate as a visiting faculty member in this course. He will discuss "Myocardial Infarction" and "Prognostic Aspects of the Electrocardiogram."

Alumni and Foundation Banquet

All alumni, faculty, and friends of the Medical School and Foundation members are reminded of the joint meeting of the Minnesota Medical Foundation and the Minnesota Medical Alumni Association at 6:00 p.m., Monday, April 30, in Rochester, Minnesota. The dinner and the meeting will be in the University Club Cafe in the Kahler Hotel. Dr. Theodore C. Blegen, Dean of the Graduate School, will give the principal address on the subject, "University Research Tradition: A Centennial Perspective."

Faculty News

Doctors Ivan D. Baronofsky and F. John Lewis spoke to a state wide meeting of public health nurses held in Bismarck, North Dakota, April 24 and 25. The meeting was sponsored by the North Dakota State Department of Health. Dr. Lewis presented a paper on "Cancer of the Gastro-intestinal Tract." Dr. Baronofsky's subject was "Cancer of the Breast."

Dr. Arnold J. Kremen, Cancer Coordinator of the University of Minnesota Medical School, and Dr. Arthur H. Wells, President of the Minnesota Division of the American Cancer Society, spent the period from April 16-21 visiting seven of the major cities of North Dakota. They addressed North Dakota physicians speaking on the subjects of gastro-intestinal cancer and cancer of the head and neck. The meetings were sponsored by the North Dakota Cancer Society.

Dr. Owen H. Wangensteen will present papers on "The Ulcer Problem" and "Extensions of Operations for Cancer" on April 26 at the First District Medical Society Meeting at the Veterans Administration Hospital in Fargo, North Dakota.

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III.

UNIVERSITY OF MINNESOTA MEDICAL SCHOOL
WEEKLY CALENDAR OF EVENTS

Visitors Welcome

April 30 - May 5, 1951

Monday, April 30

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; M-109, U. H.

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

11:00 - 11:50 Physical Medicine Seminar; E-101, U. H.

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

12:15 - 1:20 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 - Public Health Seminar; 113 Medical Sciences.

4:30 - 5:30 Dermatological Seminar; M-436, U. H.

5:00 - 5:50 Clinical Medical Pathologic Conference; Todd Amphitheater, U. H.

5:00 - 6:00 Urology-Roentgenology Conference; C. D. Creevy, O. J. Baggenstoss, and Staffs; Powell Hall Amphitheater.

Minneapolis General Hospital

8:30 - 10:00 Pediatric Rounds; Dr. Lowry; 7th Floor Annex.

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

1:00 - 2:00 Staff Meeting; Classroom, 4th Floor.

1:30 - Pediatric Rounds; Dr. Ulstrom; 5th Floor Annex.

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.

1:00 - Metabolic Disease Rounds; N. E. Jacobson and G. V. Loomis; Bldg. I.

4:00 - Medical Surgical Conference; Conference Room, Bldg. I.

Tuesday, May 1

Medical School and University Hospitals

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

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

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

1:00 - 2:00 Physiology Seminar on Cardiac Metabolism; 129 Millard Hall.

3:15 - 4:20 Gynecology Chart Conference; J. L. McKelvey and Staff; Station 54, U.H.

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

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

4:00 - 5:00 Electrocardiographic Conference; EKG Laboratory, 6th Floor, U. H.

5:00 - 6:00 X-ray Conference; Presentation of Cases by General Hospital Staff; Drs. Lipschultz and Stansbury; Eustis Amphitheater, U. H.

Ancker Hospital

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

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

Veterans Administration Hospital

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

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

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

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

1:30 - Liver Rounds; Samuel Nesbitt.

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, May 2

Medical School and University Hospitals

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

Wednesday, May 2 (Cont.)

Medical School and University Hospitals (Cont.)

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

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

12:00 - 1:00 Radioisotope Seminar; 113 Medical Sciences.

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

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

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

8:00 - 10:00 Dermatological-Pathology Conference; Review of Histopathology Section; Robert 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

8:30 - Pediatric Rounds; Dr. Lowry; 7th Floor Annex.

9:00 - Pediatric Allergy Rounds; Dr. Nelson; 4th Floor Annex.

11:00 - 12:00 Pediatric Rounds;Franklin Top; 7th Floor Annex.

12:15 - Staff Meeting; 4th Floor Annex.

1:30 - Pediatric Rounds; Dr. Huenekens and Dr. Ulstrom; 5th Floor Annex.

Veterans Administration Hospital

8:30 - 10:00 Orthopedic-Roentgenologic Conference; Edward T. Evans and Bernard O'Loughlin; Conference Room, Bldg. I.

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

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

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, May 3

Medical School and University Hospitals

9:00 - 9:50 Medicine Case Presentation; C. J. Watson and Staff; M-109, U. H.

10:00 - 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.

12:00 - Physiological Chemistry Seminar; The Chemistry of Vision; T. Hoshiko; 214 Millard Hall.

1:00 p.m. Motion Picture; The Heart; Todd Amphitheater, U. H.

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

5:00 - Bacteriology Seminar; The Mode of Action of Penicillin and Other Works of Dr. Ernest F. Gale; Glen Peterson; 214 Millard Hall.

5:00 - 6:00 Radiology Seminar; Present Status of Steroid Therapy; Robert Huseby; Eustis Amphitheater, U. H.

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

Minneapolis General Hospital

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

11:30 - Pathology Conference; Main Classroom.

1:00 - 2:00 EKG and X-ray Conference; Classroom, 4th Floor Annex.

2:00 - Psychiatry Rounds; Dr. Benton; 4th Floor Annex.

Veterans Administration Hospital

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

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

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

2:15 - Chest Rounds; William Stead.

Friday, May 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.

Friday, May 4 (Cont.)

Medical School and University Hospitals (Cont.)

10:00 - 11:50 Medicine Ward Rounds; C. J. Watson and Staff; E-221, 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; Reflexes Arising During Anesthesia for Intrathoracic Surgery; Frederick Van Bergen and Joseph J. Buckley; 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.

4:00 - 5:00 Dermatology Seminar; W-312, U. H.

4:00 - 5:00 Vascular Rounds; Davitt Felder and staff members from the departments of Medicine, Surgery, Physical Medicine, and Dermatology; Eustis Amphitheater, U. H.

5:00 - Urology Seminar; Eustis Amphitheater, U. H.

Ancker Hospital

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

Minneapolis General Hospital

8:30 - Pediatric Rounds; Dr. Lowry; 7th Floor Annex.

10:00 - Pediatric Rounds; Franklin Top; 7th Floor Annex.

1:30 - Pediatric Rounds; Dr. Ulstrom; 5th Floor Annex.

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. Myers; Ward 62, Day Room.

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

Saturday, May 5

Medical School and University Hospitals

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

9:00 - 9:50 Medicine Case Presentation; C. J. Watson and Staff; E-221, U. H.

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

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

10:00 - 11:30 Surgery Conference; O. H. Wangensteen and Staff; Todd Amphitheater, U. H.

10:00 - 11:50 Medicine Ward Rounds; C. J. Watson and Staff; E-221, U. H.

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

11:00 - 12:00 Anatomy Seminar; Ultrastructure of the Nuclear Membrane in Nerve Cells, J. F. Hartmann; Megaloblastic Anemia in Monkeys; Response to Therapy, R. D. Sundberg; 226 Institute of Anatomy.

Ancker Hospital

8:30 - 9:30 Surgery Conference; Auditorium.

Minneapolis General Hospital

11:00 - 12:00 Pediatric Clinic; Dr. Thomas and Dr. Good; Classroom, 7th Floor Annex.

Veterans Administration Hospital

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

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