"Coronary Suction" as a Source of Air Embolism:

An Experimental Study Using the Kay-Cross Oxygenator *

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THE EXTENDED duration of clinical cardio-pulmonary substitution required for the repair of some cardiac abnormalities and the unpredictable amount of intracardiac blood requiring aspiration make return of "coronary suction" blood to the circulation desirable during extracorporeal circulation.

Alterations which have been shown to occur in the blood by its course through the pump-oxygenator circuit, are expected to be magnified when blood is drawn through the coronary return aspirator, where turbulent mixing of gas with blood takes place. Some of the known blood changes which occur, are formed element destruction, foaming, and the formation of gas or particulate emboli. Gas emboli have a particularly lethal effect upon the central nervous system,[2, 6] and therefore the returning coronary sinus drainage should be bubble-free, or the bubbles should be dissipated during the transit of blood through the oxygenator.

Some oxygenator systems employ an antifoam-coated sponge in the coronary suction reservoir to aid in the dissipation of gas bubbles and the prevention of foaming. Antifoam itself has been reported to be toxic and has appeared embolically in the central nervous system.[7, 13] For this reason, it would seem desirable to eliminate this substance from the coronary suction systems, as has been done in those systems which rely upon a bubble trap only, to clear the returning arterial blood of any residual microbubbles. Therefore, it was considered important to know if antifoam was necessary to obtain bubble-free coronary return, or whether this could be accomplished adequately with a bubble trap.

This study was prompted by the occasional observation of small gas bubbles in the coronary suction line returning blood from a "bubble-trap" reservoir to the venous reservoir, during clinical perfusion. Bubbles were seen most frequently when the vacuum was increased momentarily in order to return large volumes of aspirated blood adequately. Review of the literature showed little investigations into the problem of intracardiac aspiration and its deleterious effects.

The purpose of these experiments was to evaluate the efficacy of the bubble trap only, as compared to the use of an antifoam coated sponge in the prevention of gas emboli introduced by coronary suction. Cerebral vascular permeability changes were studied, using the sodium fluoroscein technic, which has been employed in the evaluation of oxygenators. In addition, the factors of vacuum change and drying effect of short periods of air aspiration only were evaluated.

Sodium Fluoroscein Detection of Air Embolism

The cerebral vessels normally are impervious to certain acid dyes, including sodium fluoroscein, trypan blue and Congo
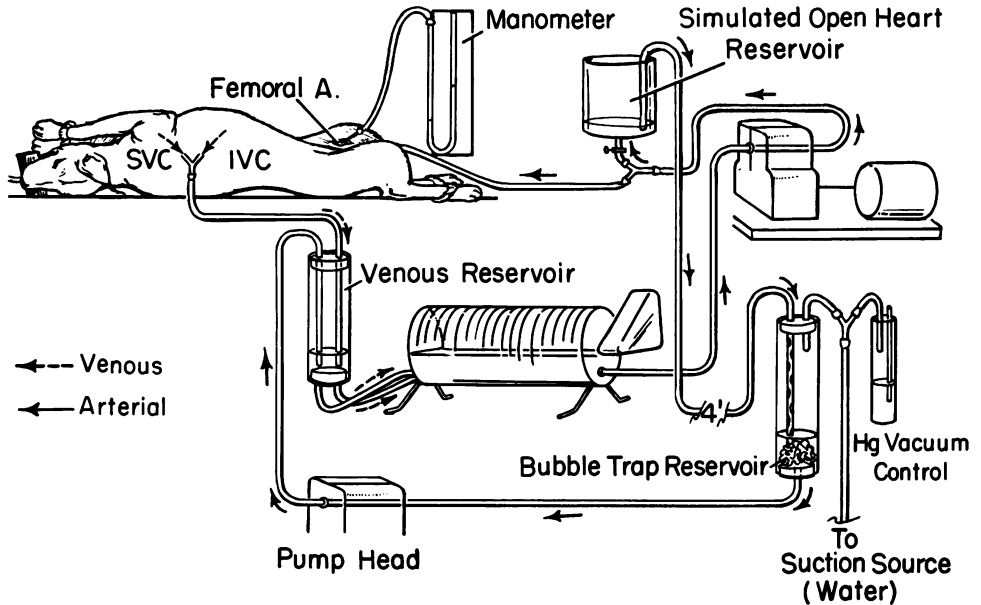
FIG. 1. Diagram of experimental pump oxygenator and coronary suction circuits.

red. This blood-brain barrier, which probably is the endothelial lining of the cerebral vessels,[1] is broken down in the presence of a number of influencing factors, allowing the dye substance to escape into the brain tissue. Broman has shown that air embolism will cause permeability changes in vessels, 10 to 20 minutes after the air injection. Solid emboli took much longer. Broman's experiments showed that pH and temperature changes were relatively unimportant factors in changing vascular permeability.

In recent well-controlled studies, Story, Hodges and co-workers [7, 15] have applied this technic in the detection of embolism associated with bubble, drum and screen oxygenators. In those studies, sodium fluoroscein was injected and the sliced brain examined for fluorescent areas under ultraviolet light, after completion of the test procedure.

Method

Mongrel dogs weighing from 10.2 to 18.7 kg. were used for these experiments. In all perfused animals, the 14-inch Kay-Cross

oxygenator with 59 flat discs rotating at 120 RPM was employed. One hundred per cent oxygen at five liters per minute was used to accomplish oxygenation. Two and five-tenths per cent pentothal sodium, intravenously, was used for anesthesia and the lungs were inflated with oxygen using an automatic positive pressure respirator. Vena caval cannulations were accomplished through a small right thoracotomy incision permitting access to the atrial appendage, cavae and vena azygos major, which was ligated. Gravity venous drainage emptied the cavae and oxygenated blood was returned to the femoral artery by a TM-1 Sigma Motor pump at flow rates sufficient to maintain relatively normal mean blood pressure, which was monitored continuously by a mercury manometer attached to a femoral artery cannula. Animals were heparinized, using 1.5 mg. Heparin per kg. of body weight. Cardiotomy was not done.

The perfusion and coronary sinus circuits are illustrated in Figure 1. They are similar to those used in clinical extracorporeal circulation, at this hospital. A simulated "open heart" was created by diverting a portion

of blood from the arterial perfusion line through a Y-connector into a Lucite reservoir, from which the blood was aspirated. The flow into this reservoir was regulated by a screw clamp, applied to the line leading to the reservoir. The "coronary suction" system consisted of a six-inch tip of a one-fourth-inch I.D. Mayon tubing, attached to a four-foot segment of three-eighths-inch Mayon tubing which led to a three-inch I.D. Mayon plastic "bubble trap" reservoir, containing a "tuffy" sponge (with or without antifoam). This reservoir was placed close to the floor to utilize the advantage of gravity. Another segment of three-eighths-inch I.D. Mayon tubing carried the blood through a T6S Sigma Motor pump (latex ventricle) to the venous reservoir, where its distal tip was submerged. The volume of blood diverted into the "open heart" reservoir was varied in different experiments, but was not changed during an individual experiment, unless perfusion conditions necessitated this. The perfusion flow and coronary suction flow were determined by direct measurement at the termination of the experiment, using residual oxygenator blood.

The vacuum used for aspiration was obtained by a water faucet attachment and regulated by the length of an air vent beneath a mercury column (Fig. 1).[11, 13] Except where indicated, the vacuum used was minus-one cm. Hg.

Total cardiopulmonary bypass was accomplished for an hour. The coronary suction system was started as soon as the perfusion was balanced and was continued during the remainder of the hour period. Occasionally it was necessary to change the flow rates momentarily because of blood pressure changes, or a dangerously low level of blood in the oxygenator.

Thirty minutes before the termination of the perfusion 25 mg. of 20 per cent sodium fluoroscein, per kilogram of body weight, was injected into the venous reservoir, and the animal was sacrificed at the completion

of the one-hour perfusion, using an overdose of intravenous Nembutal.

The brain was carefully removed and sectioned coronally. The cut surfaces were examined for areas of fluorescence, according to the technic outlined by Story and co-workers,[15] and the number of fluorescent areas counted. This technic differed somewhat from that of Story et al.,[15] in that in most instances seven cortical and three cerebellar sections were made as compared to four to six sections described in their study. This partially accounts for a larger total fluorescent count in our perfusion controls. Blood samples were obtained at the start and at the termination of the perfusion; oxygen content, carbon dioxide content, hemoglobin, pH, and plasma hemoglobin were determined.

Oiled heparinized syringes were used for blood collections and the samples were capped and iced until the determinations were made. The oxygen and carbon dioxide content of whole blood were determined, using the method of Van Slyke and Neill.[16] Hemoglobin was determined, using the Coleman Jr. spectrophotometer and oxygen capacity calculated on the basis of one Gm. of hemoglobin combining with 1.34 cc. of oxygen. Plasma pCO_2 was estimated, using the nomograms of Van Slyke and Sendroy.[17] The Cambridge glass electrode pH meter was used to determine blood pH. Red cell destruction was measured by the determination of plasma hemoglobin concentration as described by Flink and Watson.[5]

The following groups of experiments were conducted (Experiments in Groups II through VII were staged so that not all experiments in one group were done consecutively.):

Group I (Normal Controls): Four normal control animals received sodium fluorescein 12.5 mg. per kg. intravenously and were sacrificed 30 minutes later. In no instance was cerebral fluorescence visible.

TABLE 1. *Coronary Suction Test Results*

Dog No.	Weight (kg.)	Perfusion Rate (cc./min.)	Perfusion Rate (cc./kg./min.)	Coronary Suction Flow Rate (cc./min.)	Mean BP Range (mm. Hg)	Initial Art. pH	Initial Art. O₂ Sat. (Vol. %)	Initial Ven. O₂ Sat. (Vol. %)	Final Art. pH

Dog No.	Weight (kg.)	Perfusion Rate (cc./min.)	Perfusion Rate (cc./kg./min.)	Coronary Suction Flow Rate (cc./min.)	Mean BP Range (mm. Hg)	Initial Art. pH	Initial Art. O_2 Sat. (Vol. %)	Initial Ven. O_2 Sat. (Vol. %)	Final Art. pH
GROUP III—Perfusion Controls:									
94	13.2	800	60.5	—	80–120	7.50	95	56.5	7.49
97	12	1,200	100	—	80–130	7.36	98.3	56	7.37
99	13.4	1,600	119	—	60–100	7.45	100.5	38	7.32
118	11.9	1,600	143	—	80–100	7.43	100.5	—	7.33
103	11.2	800	71.5	—	60–80	7.43	99.3	41	7.38
108	13	685	52.5	—	60–100	7.29	90	62	7.23
126	13	900	69	—	80–110	—	95.5	59.7	—
GROUP IV—Perfusion with Coronary Suction and Bubble Trap, without Antifoam:									
102	12.2	1,600	131	350	50–90	—	—	—	7.25
105	12	375	31.2	500	60–80	7.36	105.5	30	7.47
109	15.4	980	63.5	800	60–90	7.38	89.8	52	7.28
112	14	520	37	1,300	80–90	7.32	100.2	24.6	7.34
115	12.3	720	58.5	1,100	60–90	7.30	85.3	53.5	7.34
116	14.2	400	28.2	1,600	70–100	7.44	97	43	7.28
127	14.3	1,000	70	1,000	80–100	—	92.5	51.5	—
GROUP V—Perfusion with Coronary Suction and Bubble Trap, with Antifoam:									
95	13.4	800	60	350	70–120	7.42	100.5	41	7.30
104	13.2	500	37.8	700	60–100	—	—	—	7.35
106	13	1,300	100	300	80–120	7.43	99.5	50	7.37
111	13.5	800	59	900	70–100	7.32	84	43	7.39
113	14	400	28.6	1,350	70–95	7.52	97.5	24	7.35
114	14.3	400	28	1,300	90–100	7.40	116.8	61	7.41
119	12.8	500	39	1,400	80–90	7.40	92.7	49.5	7.29
120	15.4	600	39	1,400	50–80	—	—	—	—
GROUP VI—Perfusion with Coronary Suction and Bubble Trap, with and without Antifoam, Using Greater Vacuum:									
128	13.3	700	52.7	950	80–100	7.45	93.3	51	—
130	14.2	2,080	146	1,000	85–100	—	—	—	—
131	12	1,280	106	1,350	80–110	—	—	—	—
132	15.2	1,700	112	1,450	90–110	7.33	95.3	45.5	7.47
133	17.6	900	51	850	80–120	7.32	97.8	61.5	7.46
140	12.5	1,300	104	1,300	90–110	7.23	86.6	—	7.46
141	11.2	1,320	118	900	80–120	—	—	—	—
142	13.6	1,600	118	1,500	90–100	—	—	—	—
174	11.2	1,000	89	800	80–100	7.40	91.5	55.6	7.48
177	12.9	950	74	920	70–120	7.40	89.5	54.6	7.30
178	15.2	1,320	87	1,400	70–100	7.35	100	39.5	7.53
GROUP VII—Perfusion with Coronary Suction, Used Intermittently, to Determine Drying Effect:									
129	14.3	440	30.8	1,000	80–110	—	98.5	—	—
142(A)	12.7	1,440	113	1,000	80–120	—	—	—	—
172	10.7	1,000	85	1,000	90–120	7.32	92	88	7.41
173	10.2	1,000	98	1,060	90–110	7.27	96.8	49	7.44
GROUP VIII—Microbubble Air Embolism Control:									
121	18.7	1,400	75	—	80–100	—	—	—	—

* Number of cortical sections/number of cerebellar sections.
** Number of fluorescent areas/number of fluorescent areas greater than 3 mm. in diameter.

TABLE 1—*Continued*

Final Art. O$_2$ Sat. (Vol. %)	Final Ven. O$_2$ Sat. (Vol. %)	Plasma Hgb. Increase (mg. %)	Final Oxygenator Blood Volume Change (cc.)	Anti-foam (Gm.)	No. Brain* Sections Examined	No.** Fluorescent Areas	Final Art. pCO$_2$ (mm. Hg)	Vacuum (cm. Hg)	No. Periods of Coronary Suction Occlusion
95	50.7	—	0	—	6/3	16/0	19.2	—	
97.3	49.5	32	0	—	7/3	9/0	19.7	—	
96.2	37.6	32	0	—	7/3	29/0	22.6	—	
97.7	49	13	0	—	7/3	5/0	22.3	—	
98	43.8	—	0	—	6/3	9/0	25.2	—	
90.3	44.3	13	−300	—	6/3	4/0	24.6	—	
92.2	48	—	0	—	7/3	10/0	—	—	
91.6	46.7	—	0	—	7/3	6/0	31.5	1	
101.5	40.5	—	0	—	7/3	1/0	13.5	1	
84.9	46.7	34	−400	—	7/3	5/0	22.9	1	
96	43.8	27	−300	—	7/3	11/0	19.5	1	
87.3	35	44	−200	—	7/3	15/0	22.3	1	
90	25.6	32	0	—	7/3	15/0	25	1	
90.5	—	—	−350	—	7/3	1/0	—	1	
92	44.3	26	−100	0.25	7/3	4/0	24.9	1	
102	54	—	0	0.4	7/3	103/4	14.3	1	
93.8	38	39	−100	0.2	7/3	4/0	25.2	1	
102.5	46	17	0	0.2	7/3	9/0	16.1	1	
96.3	33.4	—	0	0.2	7/0	52/0	15.3	1	
102.4	26.5	75	−100	0.5	8/3	16/0	22.3	1	
89.5	27	50	−250	0.7	7/3	14/0	19.4	1	
—	—	—	−200	1.2	7/3	34/0	—	1	
—	—	—	−350	—	7/3	41/0	—	2–4	
75	33.5	—	−350	—	7/3	42/0	—	2	
95	42	—	−350	0.55	7/3	49/0	—	2	
88.3	40.5	25	−350	1.0	7/3	40/0	19.8	2	
94.5	—	15	−400	—	7/3	19/0	16.7	2	
96.8	48	189	−50	2.0	7/3	114/10	17.5	2	
—	—	—	−400	0.5	7/3	152/5	—	1–6	
—	—	—	−400	—	7/3	6/0	—	2	
98.7	55	59	−300	—	7/3	1/0	15.8	3–4	
86.6	47.5	71	−400	—	7/3	110/25	25.9	0–4	
94.5	58.7	59	−450	—	7/3	50/0	15.3	1–6	
84.5	—	40	−250	—	7/3	6/1	—	1	4
—	—	—	−250	—	7/3	31/0	—	1	3
98.8	58	84	−100	—	7/3	100 (confluent)	12	1	2
96.3	53.6	126	−100	—	7/3	6/0	14	1	1
—	—	—	−150	—	7/3	45/3	—		

Group II (Air Embolism Controls): Five animals received an injection of 0.6 to 0.9 cc. of air in the common carotid artery, 12.5 mg. of sodium fluoroscein intravenously, and were sacrificed 30 minutes later. Four of the five animals showed confluent four-plus staining.

Group III (Perfusion Controls): (No coronary suction.) Seven animals. In this group, the dog was perfused for an hour, without a coronary suction system in use. Sodium fluoroscein 25 mg. per kg. was injected 30 minutes before the termination of the perfusion and the animal was sacrificed at one hour.

Group IV (Perfusion with Bubble Trap Only, in the Coronary Suction System): Seven animals. These animals were perfused for an hour, during which the coronary suction system was used. The coronary suction reservoir acted as a bubble trap and no antifoam was used on the plastic sponge. The volume of coronary suction blood was varied.

Group V (Perfusion with Antifoam Sponge in the Bubble Trap): Eight animals. These experiments were identical to those in Group IV except that an antifoam A spray coated sponge was placed in the bubble trap. The amount of antifoam used was measured by weight.

Group VI (Increased Vacuum to "Bubble Trap" Reservoir): Eleven animals. In this group, the method was changed only with respect to the amount of vacuum applied to the bubble trap. Instead of the usual one cm. Hg vacuum, th's was increased from two to six cm. Hg, the greater vacuum representing the amount created by using unrestricted water suction. The vacuum was measured by a mercury U-tube.

Group VII (The Effect of Drying): Four animals. In this group of experiments the source of "intracardiac" blood was occluded intermittently, although air continued to be drawn through the aspirator tip and tubing from the vacuum source. Five-minute periods of occlusion were used, and the number of periods varied from one to four. At the termination of the five-minute period, blood was again aspirated through the suction system. It was believed that during these short intervals enough drying of surface blood might take place to result in particulate emboli, when the use of the tubing for aspiration of blood was resumed. The maximum vacuum used was one-cm. Hg and was not varied.

Group VIII (Air Embolism Control): One animal. This experiment resulted from the unexpected introduction of visible small bubbles into the arterial perfusion line from a small perforation in the tygon tubing on the negative-pressure side of the perfusion pump. This was an important control experiment, in that the sizes of the bubbles more closely simulated those of the bubbles introduced in the coronary suction than did the sizes of those introduced by injection of air into the carotid artery, in control group II. No coronary suction system was used in this experiment.

Results

Results of experiments are listed in Table 1. Preliminary experiments used in the development of the technic were eliminated. In the control perfusion experiments (Group III) the number of small fluorescent areas in brain sections varied from four to 29. Although this number was higher than in similar experiments by Story and his co-workers, these results can be explained on the basis that more sections were examined in this study and tubing and reservoirs were re-used, making the chance of persistent particles greater. Adequate arterial blood pressure and arterial oxygen saturation in all experiments eliminated these factors as a cause of fluorescent areas. The plasma hemoglobin increase did not exceed 32 mg. per cent.

Group IV (suction without antifoam) showed the range of fluorescent areas to be even less than in the perfusion control group. The number of fluorescent areas did not exceed 15, and in four experiments, six or fewer fluorescent areas were observed. Coronary suction flow rate varied from 350 to 1,600 cc. per minute. Most fluorescent areas were seen in those experiments with the greatest coronary suction-minute volume. On the basis of these results, one would conclude that under the conditions of these experiments, significant air emboli are not introduced by the coronary suction even in the absence of the antifoam sponge. Plasma hemoglobin levels were somewhat higher than in the control perfusions.

In Group V, antifoam in the amount of 0.2 to 1.2 Gm. was used on the plastic sponge in the bubble trap. The number of fluorescent areas observed was significantly

larger than in the previous group in three of these experiments. Perfusion pressures were slightly lower in these three animals but arterial oxygen saturation was adequate in all of those studied. Plasma hemoglobin values were comparable to and, at times, higher than those in the group without antifoam. Coronary suction rate varied from 300 to 1,400 cc. per minute. Again, as in the previous group, more fluorescent areas were observed in those animals with higher coronary suction rate. The antifoam sponge did not decrease the number of fluorescent areas and in three animals a significant increase was noted. Plasma hemoglobin likewise was not diminished in this group.

In Group VI, experiments similar to those in Groups IV and V were performed, but the vacuum was increased in these experiments. In seven experiments a significant increase in the number of fluorescent areas was noted. In those experiments in which the vacuum was not restricted (up to six cm. Hg) marked fluorescence appeared. The presence of antifoam did not significantly change these results. In experiment 174, exhaustion of available donor blood necessitated terminating the coronary suction phase of the experiment at 21 minutes, instead of the usual one hour.

In Group VII, testing the effect of drying, significant fluorescent areas were observed in two animals. In the other two, no significant change in the number of fluorescent areas, as compared with control experiments, was observed. Although the number of experiments was small they indicated that embolism had resulted from the re-use of coronary suction equipment in which blood films had been exposed to overflowing air for a short period of time. Plasma hemoglobin increases were significant in this group.

The single microbubble air embolism control experiment (Group VIII) showed significant increase in both large and small fluorescent areas.

Arterial oxygen saturation of the terminal samples has been greater than 90 per cent in all except seven experiments, where saturations varied from 75 to 89 per cent. In each of these seven animals there was a volume deficit of from 250 to 400 cc. in the oxygenator at the time the sample was taken. This indicates the necessity of maintenance of a full blood pool in the oxygenator to insure adequate oxygen saturation. There was no direct correlation between oxygen saturation and the number of fluorescent areas observed in sacrificed animals. In 22 experiments in which both initial and terminal venous oxygen saturation values were obtained the saturation remained the same or increased in nine, while there was a decrease in 13, varying from three per cent to 34 per cent. In ten of these 13, a pool volume deficit of from 100 to 400 cc. existed, when the sample was drawn.

In the 24 experiments, in which initial and terminal arterial PH values were obtained, there was an increase between initial and terminal values (average 0.09 units) in 12, and a decrease in 12 (average 0.10 units). A correlation between pH and flow rate was not observed.

Arterial pCO_2, in the terminal blood sample, was determined using the nomograms of Van Slyke and Sendroy. In no instance has the final pCO_2 been abnormally high. The pCO_2 varied between 12.0 and 31.5. This low pCO_2 reflects the oxygenator's capacity to adequately eliminate carbon dioxide from the venous blood. Hypocarbia has been blamed as a cause of neurological sequelae after cardiopulmonary bypass because of its effect of diminishing cerebral blood flow.[10] Cerebrovascular permeability apparently was not affected by this factor. Similarly low pCO_2 values in 15 prolonged two-hour animal perfusions done in this laboratory, using another type of disc oxygenator, did not result in evi-

dent neurologic sequelae in the 12 animals which survived these perfusions.[12]

Gross pathologic findings were not observed in any brain, regardless of the extent of fluorescence. Study of sections of some of the brains, showing more extensive fluorescence, likewise did not reveal evident microscopic lesions.

Discussion

The volume of blood aspirated from the open heart during extracorporeal circulation may vary considerably depending upon the type of defect encountered, the use of aortic or pulmonary artery occlusion, or cardioplegia, and the presence of increased bronchial or extracardiac collateral flow. In the presence of an unsuspected ductus arteriosus this volume may be overwhelming to the point that the planned procedure must be abandoned. Approximately 25 per cent of the perfusion flow, or an amount in excess of one liter per minute in adult patients, has been aspirated from the cardiac chambers in a series of 68 patients undergoing bypass reported by the Mayo Clinic group.[4]

Aspirated blood was found to be "toxic" and was discarded by Lillehei and others in their work with cross-circulation.[2] A high level of hemolysis was found to be present. These investigators doubted that the main toxic agent was plasma hemoglobin, but rather fibrinolysin or other lytic enzymes activated by the trauma of suction.

Utilization of the aspirated intracardiac blood provides for more operating time for the surgeon, ease in the operation of the pump oxygenator, and less danger and expense of an increasing number of blood units required.

The possible danger of air embolism introduced through the "coronary suction" system has been suspected by Maloney et al.,[9] who have incorporated a large settling chamber in the pump oxygenator to allow aspirated blood to sit for ten minutes before being returned to the circulation. One questions the efficacy of a small volume intra-arterial "plenum chamber" type of bubble trap in removing all bubbles, when considering the slow rate at which these minute bubbles rise.

Principles for an adequate coronary sinus suction system which have been presented by Donald et al.[4] include the use of low vacuum, large bore tubing to decrease velocity yet maintain a blood seal in the tube and a defoaming chamber containing an antifoam soaked sponge. DeWall [3, 4] additionally mentions the importance of a short aspirator tip, the availability of a second suction tip, utilization of the effect of gravity by placing the reservoir close to the floor, and maintaining the aspirator tip in a horizontal position to decrease the vacuum necessary.

Miller and others [8] depicted a cardiac blood collecting apparatus which decelerates the blood by passing it down the side of the bubble trap container, allowing air bubbles to dissipate. Further protection against air embolism was sought by the use of two concentric chambers through which the blood was forced to pass.

Senning [9] used a pulsator with pressure variation of atmospheric to minus-750 mm. Hg, applied to the suction reservoir to enlarge small bubbles so they could rise to the surface and burst.

It is apparent that most deleterious changes occur at points of turbulence, where hemolysis, minute bubble introduction, and foaming take place. These points are located at the suction tip, at the junction of the aspirator tip and at the suction tubing, and upon leaving the tubing into the reservoir, points at which the blood rapidly accelerates and decelerates. These velocity changes can be minimized by the use of a short suction tip of large caliber, low controlled vacuum of a high flow rate, and a large bore suction tubing (three-eighths-inch inside diameter) of as short a length as possible. Under these conditions

minimum resistance and velocity will result in maximum flow with less turbulence upon deceleration of the aspirated blood. By frequently breaking the blood column in the suction tubing by allowing the intermittent introduction of small columns of air, a lower vacuum will be required to keep the column moving. If the aspirator cannot remove the blood adequately a second aspirator should be employed rather than increasing the vacuum to excessive levels. The suction tube should be clamped off when it is not in use since the drying effect of aspirated air may be deleterious to the exposed blood film. The temptation to use the "coronary" aspiration tip in the pericardial sac and pleural space should be avoided since this blood may contain particles of lint, fibrin, tissue, or bone.

During clinical perfusions at this hospital, the volume of intracardiac blood aspirated has been retained in the bubble trap until the capacity of the trap is reached; it has been transferred then to the venous reservoir by a manually-controlled Sigma Motor pump. When the volume has been small, aspirated blood has not been returned to the oxygenator. With large volumes the pump has been run continuously and there has been less time for bubbles to rise at this phase.

Bubbles which remain in the blood entering the venous reservoir from the coronary sinus bubble trap may be dissipated within the oxygenator but in the rotating disc oxygenator may be swept into the arterial perfusion line in a matter of seconds and on to the patient. This problem may not exist in a screen type of oxygenator since all blood must pass through the filming phase before re-entering the arterial line.

Conclusions

1. As determined by the sodium fluoroscein injection technic, under the conditions of these controlled experiments, the antifoam sponge did not significantly decrease the number of air emboli introduced by way of the coronary suction and may itself be a source of embolization.

2. When the vacuum source was restricted to one-cm. Hg, the incidence of fluorescence in the coronary suction group was not significantly greater than in the control group. By increasing the vacuum to two-cm Hg, and then to six-cm Hg, significant jumps in the number of fluorescent areas occurred, indicating the need for low vacuum levels in suction systems.

3. Increased red blood cell destruction occurred with increased vacuum.

4. The flow of air through coronary suction equipment for short periods after initial use may cause drying of blood film and thereby make subsequent use of this equipment hazardous, from the standpoint of particulate embolization and red blood cell destruction.

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