EXPERIENCES WITH CARDIAC CATHETERIZATION USING HALOTHANE–COMPRESSED–AIR ANESTHESIA

MARTIN LLOYD NORTON, M.D., YUKIO KUBOTA, M.D.

CARDIAC catheterization has presented many problems in anesthesiologic management. It is the purpose of this paper to 1) outline the requirements for cardiac catheterization imposed upon the anesthesiologist; 2) describe technique of management; 3) demonstrate some of the cardiopulmonary effects of fluothane when utilized as recommended; 4) describe a method of monitoring patients; 5) suggest further utilization of cardiac catheterization techniques in anesthesiologic research; and 6) summarize our experience with catheterization techniques in children with congenital heart disease.

The criteria for the ideal anesthetic management of patients for cardiac catheterization (table 1) and related procedures are stringent. Further complications are imposed by the necessity of managing the patient in darkness and also by the fact that these patients are invariably greater than normal anesthetic risks by virtue of their basic pathology.

The reported experience to date has not suggested compliance with the described criteria. Most of the available inhalation agents are explosive. Chloroform for cardiac catheterization must be further evaluated. Nitrous oxide has proven to be impotent without heavy premedication and/or supplement with other agents [1] such as barbiturates, narcotics or tranquilizers. Trichlorethylene [2] has similarly proven to require heavy premedication or supplementation. The justification for endotracheal intubation [3] has been questioned. The heavy "premedicant schema" [4] exposes patients to the risks of respiratory and cardiovascular instability.[5–8] Agents given intramusculuarly [9] or rectally [10] are difficult to control and frequently delay awakening. Intravenous injection of these same agents especially via the intracardiac catheter does not appreciably improve controllability and imposes the additional hazard of *p*H and hydrostatic pressure changes. The effect of directly exposing the endocardium to injected agents requires investigation. The intravenous route is also impractical in the management of the very young patient due to the technical problems of inserting and maintain the needle, *in situ*.

Objections to inhalation techniques include: 1) the anesthetic might interfere with blood gas analysis; 2) high concentrations of oxygen might change arterial and mixed venous saturation values; 3) above normal oxygen percentages might change pulmonary hydrostatic pressures, resistances and or flow patterns; and 4) the anesthetist would be exposed to excessive radiation hazards involved in holding a face mask.

We are presenting here in our approach to the problem which we believe meets the objections outlined without deviating from the desired criteria.

TABLE 1

CRITERIA IMPOSED FOR ANESTHETIC MANAGEMENT OF CARDIAC CATHETERIZATION PROCEDURES

1. Nonexplosive agents.
2. High potency, providing rapid smooth induction of anesthesia.
3. A readily obtainable "steady state" in light planes of anesthesia.
4. Agent and/or its effects should not interfere with blood gas analyses.
5. Agent and/or its effects should not alter the cardiopulmonary hemodynamics.
6. Rapid and smooth emergence from anesthesia.
7. Constancy of blood oxygen concentration.
8. Controllability of airway.
9. Avoidance of narcotics and adjuvants that may interfere with cardiorespiratory physiology.

This study was received from the University Hospital, 303 East 20th St., New York, New York, and accepted for publication April 4, 1960. Dr. Norton is Assistant Professor of Clinical Anesthesiology, New York University Post-Graduate Medical School, and Dr. Kubota is Fellow in Anesthesiology, University Hospital, New York.

Method

This investigation included 38 patients, anesthetized a total of 54 times. Their ages ranged from three months to 14 years. Weight distribution was from nine to 132 pounds. Physical status according to the American Society of Anesthesiologists classification ranged from category II to VII, with the majority in III. The diagnoses of the patients are noted in table 2.

Eighteen patients served as their own controls. In these patients (ages 4 to 14 years), part or all of the catheterization procedure was completed with the patient awake, and the procedure repeated 20 to 30 minutes after the induction of anesthesia and the establishment of a "steady state" with no other time interval intervening. Twelve patients were operated upon and confirmatory data obtained at operation. Thirteen patients were subsequently anesthetized one or more times for the same catheterization procedures utilizing other anesthetic techniques and agents. All patients were observed in the recovery room regardless of level of consciousness.

For the purposes of this study we have used the following techniques and procedures. Premedication included only secobarbital, and scopolamine or atropine as indicated. In some cases belladonna preparations were omitted. Our objective was light sedation. Attention to omission of belladonna drugs was of particular importance in those patients in whom we intended to use acetylcholine as a diagnostic–prognostic test for pulmonary hypertension.

Induction and maintenance of anesthesia was accomplished by inhalation techniques only. The vehicle for vaporization of halothane was compressed air with the oxygen metered to give an oxygen content between 21.5 and 25 per cent. The Stephen-Slater nonrebreathing mask, or valve, was used throughout the study with an average 10-liter flow via pressure regulated valves through a Mark I Fluotec calibrated in tenths of a per cent.

All patients had a plastic oropharyngeal airway inserted soon after induction of anesthesia. No patients required tracheal intubation. Evaluation of a "steady state" and depth of anesthesia was by clinical observation and by analysis of electroencephalogram patterns. Our objective was EEG level 1 to 2.

TABLE 2

Diagnostic Categories

Diagnosis	No. Included
Tetralogy of Fallot	6
Ebstein's malformation*	1
Taussig-Bing syndrome**	2
Pulmonary stenosis	12
Aortic stenosis	4
Anomalous pulmonary drainage	5
Normal heart	2
Interventricular septal defect	8
Interatrial septal defect	5
Coaretation of pulmonary arteries	2
Patent ductus arteriosus	2
Pulmonary hypertension	5
Transposition of great vessels, total	2

* Ebstein's Malformation—displacement of tricuspid valve into right ventricle, hypoplastic right ventricle, large dilated right atrium, associated septal defect.

** Taussig-Bing Syndrome—complete transposition of aorta and partial transposition of pulmonary artery.

Monitoring of the patients was by intra and extracardiac electrocardiography, blood gas analyses (pH, CO_2, and O_2 content), intra and extracardiac phonocardiography, direct fluoroscopic visualization of cardiac and respiratory activity, intracardiac and great vessel pressures via Statham Strain-Gauge and dye-dilution hemodynamic studies as well as the aforementioned electroencephalography.

To evaluate the exposure of the anesthetist's hand to roentgen radiation a Cambridge dosimeter of the gold-leaf type, registering in milliroentgens, was taped to the anesthetist's glove or hand holding the face mask. We did not monitor angiocardiography and cinecardiography because during momentary X-ray exposures the anesthetist's hand was removed from the field.

We have found it possible to complete the procedures of cardiac catheterization, angiocardiography, cinecardiography, and dye dilution studies in as little time as one and one-half hours, though our longest time was four hours, including special studies. Average time for the procedure is two and one-fourth hours.

Results

The results of monitoring roentgen exposure (table 3) indicated an exposure range of 10

TABLE 3

ROENTGEN EXPOSURE TO THE ANESTHETIST'S HAND (CAMBRIDGE GOLD-LEAF DOSIMETER)

	KV	MA	Exposure Time (min.)	mR
1.	75	2.5	20	23
2.	75	2.75	22	35
3.	75	2.0	14	12
4.	75	2.0	12	10
5.	75	2.75	18	22
6.	75	2.5	8	23
7.	75	2.75	10	14
8.	75	2.5	12	18
9.	75	3.0	10	20
10.	75	2.5	9	15
11.	78	2.5	20	16
12.	78	2.5	12	18
13.	78	2.5	18	39
14.	80	2.5	20	31
15.	75	2.5	14	10
16.	70	2.5	13	18
17.	75	2.5	21	32
18.	75	2.5	22	36
19.	80	3.0	8	15
20.	70	2.75	12	21
21.	75	2.75	10	12
22.	78	2.5	14	49
23.	78	2.5	14	18
24.	78	2.5	8	10

to 50 mR, averaging 15.1 mR exposure to the anesthetist's hand.

No significant variations in oxygen or carbon dioxide content were found in the patients serving as their own controls (table 4). The comparison between arteriovenous determinations using local anesthesia and halothane-compressed air anesthetic management (table 5) was a maximum of 0.4 volumes per cent, with an average of 0.19 volumes present. Also the magnitude of the difference varied in the same direction regardless of the anesthetic technique used.

Oxygen saturation was measured in the right ventricle or the pulmonary artery. The pulmonary artery site was used preferentially. There were insignificant variations in mixed venous oxygen saturation, between local and halothane anesthesia (table 6).

Fieldman and associates [10] reported a reversal of the usual inferior-superior vena cava oxygen saturation ratios when progressing from the awake to the anesthetized states. Our results (table 7) indicate that this is neither predictable nor invariable, either as to existance of a change, magnitude, or direction.

TABLE 4

COMPARISON OF OXYGEN AND CARBON DIOXIDE SATURATION (VOLS. %) AND *p*H DURING LOCAL AND HALOTHANE-AIR ANESTHESIA

Pt.	Site	Local			Halothane-Air		
		O_2	CO_2	*p*H	O_2	CO_2	*p*H
1.	FA	17.0	28.4	7.40	16.9	27.8	7.40
2.	FA	16.3	36.1	7.45	16.0	37.5	7.45
3.	FA	15.8	36.7		16.2	41.8	
4.	FA	16.1	41.1		15.9	42.0	
5.	PA	12.5	37.0		12.5	38.8	
6.	BA	15.5	40.1	7.41	15.4	39.6	7.40
7.	PA	11.9	39.7		12.5	42.0	
8.	MPA	12.3	40.6		12.1	38.9	
	BA	17.0	37.5		17.1	37.5	
9.	FA	17.3	35.8	7.49	17.3	43.2	7.43
	PA	15.5	40.9		15.4	43.2	
10.	BA	16.9	37.9	7.41	16.5	41.0	7.41
	RV	14.5	40.6		14.4	41.8	
11.	RBA	17.4	39.2		18.0	45.1	
12.	LBA	14.9	41.4		14.8	42.1	
13.	PA	17.3	41.8	7.43	17.5	39.8	7.47

The differences in oxygen saturation show no specific trend. Pulmonary intravascular

TABLE 5

COMPARISON OF ARTERIOVENOUS OXYGEN DIFFERENCES DURING LOCAL AND HALOTHANE-AIR ANESTHESIA (VOLS. %)

Awake			Halothane-Air			Difference
FA-BA	PA	A-V	FA-BA	PA	A-V	
15.8	17.2	1.4	16.2	14.5	1.7	0.3
16.2	12.8	3.4	15.9	12.7	3.2	0.2
16.3	11.1	5.2	16.0	11.0	5.0	0.2
16.8	15.8	1.0	17.0	15.8	1.2	0.2
16.6	12.5	4.1	16.8	12.5	4.3	0.2
15.5	11.9	3.6	15.4	11.5	3.9	0.3
15.7	12.4	3.3	15.7	12.4	3.3	0.0
17.0	13.0	4.0	16.9	13.0	3.9	0.1
17.3	15.5	1.8	17.3	15.4	1.9	0.1
16.9	15.5	1.4	16.8	15.8	1.0	0.4
17.0	12.3	4.7	17.1	12.1	5.0	0.3
17.2	15.1	2.1	17.4	15.1	2.3	0.2
15.0	12.5	3.5	15.0	12.5	3.5	0.0
17.4	13.5	3.9	18.2	14.1	4.1	0.2
14.9	11.9	3.0	13.8	11.0	2.8	0.2

FA = Femoral Artery; BA = Brachial Artery; PA = Pulmonary Artery; A-V = Arteriovenous Difference.

TABLE 6

DIFFERENCES IN MIXED VENOUS OXYGEN SATURATION AS MEASURED IN THE PULMONARY ARTERY OR RIGHT VENTRICLE DURING LOCAL AND HALOTHANE-AIR ANESTHESIA (VOLS. %)

Patient	Awake	Halothane-Air	Difference
1.	17.2	14.5	2.7
2.	12.8	12.7	0.1
3.	11.8	11.9	0.1
4.	12.5	12.5	0.0
5.	12.9	12.3	0.6
6.	11.1	11.0	0.1
7.	15.8	15.8	0.0
8.	17.3	15.5	1.8
9.	12.5	12.5	0.0
10.	11.9	12.5	0.6
11.	12.9	13.7	0.8
12.	15.4	15.6	0.2
13.	12.4	12.4	0.0
14.	15.7	15.7	0.0
15.	12.3	12.1	0.2
16.	15.1	15.4	0.3

hydrostatic pressures and cardiovascular pressures remained relatively constant as well (table 8). Peripheral pressures as measured in the femoral or brachial arteries (table 9) failed to reveal appreciable degrees of hypotension with anesthesia, as compared to the pre-anesthesia levels. Contrary to reports of others, we did not encounter bradycardia or tachypnea while maintaining the desired level of anesthesia (EEG levels 1 to 2).

TABLE 7

COMPARISON OF IVC/SVC OXYGEN CONTENT DURING LOCAL AND HALOTHANE ANESTHESIA (VOLS. %)

	Awake		Difference	Halothane-Air			IVC Diff.	SVC Diff.
	IVC	SVC		IVC	SVC	Diff.		
1.	12.0	11.8	0.2	12.2	13.0	0.8	0.2	0.8
2.	9.8	14.6	4.8	10.7	14.9	4.2	0.9	0.3
3.	12.5	14.0	1.5	12.0	14.2	2.2	0.5	0.2
4.	13.0	13.8	0.8	12.8	12.3	0.5	0.3	1.5
5.	10.9	12.1	1.2	12.2	13.0	0.8	1.3	0.5
6.	13.0	11.3	1.7	11.9	13.3	2.6	1.1	2.0
7.	13.3	10.5	1.8	12.6	14.6	2.0	0.7	4.1
8.	12.8	12.4	0.4	14.6	15.8	1.2	1.8	3.4
9.	12.4	14.0	1.6	13.0	15.0	2.0	0.6	1.0
10.	13.1	13.6	0.5	12.4	12.3	0.1	0.7	1.3

TABLE 8

PRESSURE RECORDINGS UNDER LOCAL AND HALOTHANE-AIR ANESTHESIA (MM. HG)

Pt.	Site	Local-Awake	Halothane-Air	Difference
1.	MPA	25/15	25/15	0
2.	RV	25/0 M 18	25/2 M 18	M0
	RA	2/-2	2/-2	0
3.	RV low	84/0	84/0	0
	RV high	73/3	73/3	0
	PA	83/10 M 66	75/46 M 65	M1
	BA	57/43	57/43	0
4.	RA	8/1	8/1	0
	RV	59/0	59/0	0
	MPA	59/36	59/36	0
5.	PA	10/3	10/2	0/1
	RA	11/0	11/0	0
	RV	61/0	61/0	0
6.	RA	M3	M3	M0
	PA	14/5 M 11	14/5 M 11	0
	BA	90/58 M 80	90/58 M 80	0
7.	FA	121/72 M 86	95/54 M 85	M1
	LV	168/29 M 91	164/27 M 89	M2
	RV	89/4	89/4	0
	MPA	30/10	30/10	0
8.	RV	125/12 M 50	94/15 M 46	M4
9.	RA	2/-1	2/-1	0
	RV	42/3 M 17	42/3 M 17	0
	MPA	40/12	40/12	0
	LV	100/0 M 33	100/3 M 33	M0
	PV	5/2 M 3	4/0 M 3	M0
10.	MPA	28/12 M 23	34/15 M 23	M0
	RBA	85/58 M 80	98/78 M 85	M5
11.	RA	2/-1	2/-1	0
	RV	15/2 M 8	M8	M0
	PA	13/5	13/5	0

We have encountered a minimal number of complications, all but three of which responded to temporary cessation of catheter manipulation or dye introduction. Two patients developed cardiac arrhythmias (nodal tachycardia) during induction of anesthesia. Three patients required digitalization during the maintenance phase of the procedure, for marked supraventricular tachycardia following distention of the right atrium by the catheter in two cases, and inadvertent entrance into the coronary sinus in the third case. In the opinion of the cardiologists, as well as ourselves, none of the arrhythmias (excepting nodal tachycardia) could be directly attributed to anesthesia. The nodal tachycardias described, cleared spontaneously on attaining the "steady state" at a halothane maintenance concentration (as delivered from the apparatus) of 0.5 to 1.0 per cent, and did

TABLE 9

COMPARISON OF ARTERIAL PRESSURES BEFORE AND DURING ANESTHESIA (HALOTHANE-AIR) STATHAM STRAIN GAUGE

Site	Pre-anesthesia	During Anesthesia
BA	57/43	57/43
FA	121/72 M 86	94/54 M 72
FA	133/98 M 110	100/60 M 94
FA	72/50 M 60	74/48 M 60
BA	90/58 M 80	90/58 M 80
FA	50/40 M 41	62/40 M 44
FA	88/48	88/48
FA	90/32 M 80	92/40 M 80
BA	85/58	98/78 M 80
FA	95/6	100/0

BA = Brachial Artery; FA = Femoral Artery.

not recur during the remainder of the procedure. The incidence and nature of arrhythmias noted were no different than those we have observed with other techniques of anesthetic management.

Only four subjects given halothane-air anesthesia were not fully recovered upon arrival in the recovery room. Seven of the 13 patients anesthetized with other agents and techniques were asleep 30 minutes to three hours after arrival in the recovery room.

DISCUSSION

Use of halothane-compressed air anesthesia for the procedures of cardiac catheterization, angiocardiography, cinecardiography, phonocardiography, and dye dilution studies has proven useful in our hands, under the conditions presented.

The average roentgen exposure to the anesthetist's hand of 15.1 mR is within safe radiation limits [11] although somewhat more than reported by Inglis.[12] Constant monitoring of exposure hazards should be employed.

Failure of the agent, at delivery concentrations described, to interfere with gas analyses is infered from comparative values during the awake and anesthetized states (tables 4–8).

Our data suggest that blood oxygen content does not necessarily decrease with fluothane anesthesia and oxygen consumption is not directly related to changes in cardiac output, *per se*,[13] but rather also to other factors, for examples, the level of anesthetic plane, the degree of sedation established preanesthtically, the functional stress imposed upon the tissues, body temperature, the hemodynamics of the primary pathology and degree of arterial hypotension.

The changes noted previously in the inferior-superior vena cava oxygen saturation ratios when progressing from the awake to the anesthetized state suggest other variables to be of much greater import. Factors such as changes in tissue oxygen consumption with increasing anesthetic depth, arteriovenous shunts in the liver, variations in site of catheter placement (proximity to renal outflow), cerebral arteriovenous shunts, respiratory changes affecting oxygen content, and hemodynamic alterations produced by the primary pathology must all be considered. In view of this, it is doubtful that these values, *per se*, can be considered reliable as a guide to anesthetic effects on the cardiopulmonary circulation, or in the diagnosis of specific cardiac anomalies.

The two major objections to inhalational anesthesia for cardiac catheterization procedures are: 1) inhalational techniques may vary the oxygen saturation levels and ratios from the awake state; and 2) variations in oxygen saturation of the blood may change the pulmonary vascular resistance in such a way as to reduce the pulmonary intravascular hydrostatic pressures and thus effect a reversal of a right-to-left shunt or change the magnitude of the shunt. The differences we have found in oxygen saturation between the awake and anesthetized states have shown no specific trend. Neither have we noted any marked changes in related cardiopulmonary intravascular pressures. Patient to patient variances are a reflection of depth of anesthesia and changes in metabolic need. In the two cases where the pulmonary arterial oxygen saturation notably decreased there was a severe degree of pulmonic valve stenosis. We believed the decreased oxygen saturation under anesthesia to be related to mechanical obstruction by the catheter during induction and stabilization of anesthesia. Since these two cases we have removed the catheter from the pulmonary artery in cases of pulmonic stenosis, and reinserted it again after the desired level of anesthesia was attained. Variations in arterial CO_2 tension were independent of variations in oxygen tension. Also, simultaneous *p*H values remained relatively constant. This would appear to indicate that at a relatively constant

level of light plane anesthesia, no effect was produced upon respiratory function.

Our work included varying the levels of anesthesia from electroencephalographic levels 1 to 3.[11] We have found this guide to be useful and accurate as a clinical adjunct only in these upper planes of anesthesia. We recommend its use in the darkened fluoroscopy room as routine monitoring to aid in the clinical evaluation, of the patient.

All the above procedures were originally completed under halothane-compressed air anesthesia. However, since this series, we have preferred to use halothane and 100 per cent oxygen during cinecardiography and angiocardiography for the following reasons. As the techniques of contrast media injection involve a high-pressure injection method, we believe that this results in the introduction of a bolus into the cardiovascular system. This predisposes to the dangers of contrast media in the carotid or coronary arteries. It is preferable to introduce a maximal oxygen concentration into the system in the hope of "tiding over" the period of relative tissue hypoxia, and decreasing the degree of coronary vasospasm.

An observation yet to be adequately explained is that of the period of "breath-holding" following angiocardiography dye injection. We postulate this to be similar to the initial tachycardia, bradycardia, then again tachycardia sometimes noted. This "Bainbridge-like" reflex most likely originates in the stretch receptors of the atrium or aortico-carotid pressor receptor mechanism secondary to the high-pressure introduction of the contrast media.

Summary

We have presented our experiences in cardiac catheterization using halothane-compressed air anesthesia, predicated on the maintenance of light planes of anesthesia vigilantly monitored. Our observations have indicated no major interference by the described anesthetic technique with the diagnostic requirements of the procedure. We believe halothane-compressed air anesthesia is a useful and controllable technique. When administered as recommended, it does not interfere with determinations of oxygen saturation, carbon dioxide content, *p*H, intravascular pressures or other studies.

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REFERENCES

1. Taylor, C. and Stoelting, V. K.: Anesthetic management of small children undergoing cardiac catheterization and angiocardiography, Anesth. & Analg. **38**: 441, 1959.
2. Keats, A. S., Telford, J., Kurosu, Y. and Latson, J. R.: Providing steady state for cardiac catheterization under anesthesia, J .A. M. A. **166**: 215, 1958.
3. Eggers, G. W. N., Stoeckle, H. G. E. Jr. and Allen, C. R.: General anesthesia for cardiac catheterization, ANESTHESIOLOGY **20**: 817, 1959.
4. Lundy, J. S.: Amnesia-analgesia for management of children too young to cooperate, J. A. M. A. **169**: 1184, 1959.
5. Eckenhoff, J. E.: Editorial: Influence of opiates upon experimental results, ANESTHESIOLOGY **17**: 616, 1956.
6. Dripps, R. D. and Comroe, J. H. Jr.: Clinical studies on morphine 1. The immediate effect of morphine administration intravenously and intramuscularly upon the respiration of normal man, ANESTHESIOLOGY **6**: 462, 1945.
7. Eckenhoff, J. E., Helrich, M. and Hege, M. J. D.: Effects of narcotics upon the respiratory response to carbon dioxide in man, Surg. Forum of the American Coll. of Surg., 1954.
8. Drew, J. H., Dripps, R. D. and Comroe, J. H. Jr.: The effect of morphine upon the circulation of man and upon the circulatory and respiratory response to tilting, ANESTHESIOLOGY **7**: 44, 1946.
9. Keown, K. K., Fisher, S. M., Downing, D. F. and Hitchcock, P.: Anesthesia for cardiac catheterization in infants and children, ANESTHESIOLOGY **18**: 270, 1957.
10. Fieldman, E. J., Lundy, J. S., Dushane, J. W. and Wood, E. H.: Anesthesia for children undergoing diagnostic cardiac catheterization, ANESTHESIOLOGY **16**: 868, 1955.
11. National Bureau of Standards, Handbook on Radiation Protection Measurements, Publication Nos. 59, 60, and N.B.S. Technical News Bulletin **41**: 17, 1959.
12. Inglis, J. M.: Anesthesia for cardiac catheterization in children, Anaesthesia **9**: 25, 1954.
13. Severinghaus, J. W. and Cullen, S. C.: Depression of myocardium and body oxygen consumption with Fluothane, ANESTHESIOLOGY **19**: 165, 1958.
14. Gain, E. A. and Paletz, S. G.: An attempt to correlate the clinical signs of Fluothane anesthesia with the electroencephalographic levels, Canad. Anes. Soc. J. **4**: 289, 1957.