ANESTHESIOLOGY

The Journal of

THE AMERICAN SOCIETY OF ANESTHESIOLOGISTS, INC.

HEMODYNAMIC STUDIES DURING THIOPENTAL SODIUM AND NITROUS OXIDE ANESTHESIA IN HUMANS [*][†][‡][§]

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SINCE the introduction of thiopental sodium (pentothal® sodium) into clinical anesthesia by Lundy (1) in 1934, much experimental work has been done with this drug. Prime and Gray (2) reported greatly decreased cardiac output using heart-lung preparations in dogs. They attempted to measure cardiac output in humans by means of the modified Fick principle and reported an increase in cardiac output in persons under thiopental sodium and nitrous oxide anesthesia; no mention was made of the depth of anesthesia. Johnson (3), in 1951, reported a decrease in cardiac output in humans under narkotal anesthesia; both the Fick and the Hamilton dye-dilution techniques were used. He concluded that the autonomic tone of the heart and blood vessels was decreased and that peripheral pooling of blood occurred. Li, Reynolds, Rheinlander and Etsten (4), in 1954, reported similar results in human beings during thiopental sodium anesthesia and indicated the depth of anesthesia as determined by the electro-encephalograph. They noted a 25 per cent decrease in the cardiac index during light levels of anesthesia, with a decrease in estimated intrathoracic blood volume of 23 per cent and a decrease of 35 per cent in the stroke volume. Greishheimer and co-workers (5), in 1953, using the cuvette oximeter

and determining cardiac output in dogs by the dye-dilution method, found that cardiac output increased with duration of anesthesia and had no relation to the depth of anesthesia. Reynolds and Veal (6), in 1938, observed that repeated intravenous administration of small amounts of thiopental sodium sufficient to maintain light anesthesia produced signs of circulatory failure in dogs after variable periods.

Gruber (7), working with animals, reported a decrease in blood pressure after the rapid administration of thiopental sodium, with a rapid return to normal. After slow injection of the same drug, an increase in blood pressure was observed. Changes in cardiac rate varied in different animals. Dilatation of the heart was evident in dogs.

In 1952, Patrick and Faulconer (8) observed the effects of thiopental sodium on ventilation and concluded that the arterial oxygen concentration remained high during such anesthesia but that the carbon dioxide tension was increased. Since the latter correlated so well with the pH of arterial blood, they concluded that the primary alterations in the acid-base balance were due to alterations in ventilation, with consequent respiratory acidosis.

Betlach (9), and Volpitto and Marangoni (10), reported no significant changes in the electrocardiographic patterns in patients under thiopental sodium anesthesia, while Kohn and Lederer (11) maintained that ectopic beats were frequent during this form of anesthesia and that defects in interventricular conduction were observed. Gruber and associates (12, 13) observed various arrhythmias associated with thiopental sodium anesthesia and concluded that they were produced by unduly increased blood pressure during anesthesia. Rosner, Newman and Burstein (14) noted frequent sinus tachycardia in humans anesthetized with thiopental sodium combined with a muscle relaxant and attributed this to hypoxia after improper ventilation.

The present study was undertaken in an attempt to correlate changes in various hemodynamic variables with different levels of anesthesia determined by the electro-encephalogram as described by Kiersey and associates (15) and to relate these changes to the duration of anesthesia.

METHODS

Each patient received an intravenous injection of 10 mg. of morphine sulfate and 0.4 mg. of atropine sulfate, with the exception of 1 patient, who received 7.5 mg. of morphine and 0.4 mg. of atropine. These injections were given one-half hour before the observations were begun. Each patient also received 1½ grains of pentobarbital sodium (nembutal®) about 1 hour prior to the observations.

With the patient on the operating table, an earpiece oximeter (16) was connected, as were electrocardiographic and electroencephalographic leads. A Cournand number 4 or 5 F. catheter was advanced into the region of the superior vena cava in the central venous circu-

lation via a 15 gauge or 16 gauge needle introduced into a vein in the arm. This catheter was utilized for injection of dye and was connected to a strain-gauge manometer (17) for recording the central venous pressure. A specially ground 20 gauge needle (18) was then introduced into the radial artery and was connected in series to a cuvette oximeter (19–21) and a strain-gauge manometer (fig. 1). All the leads were assembled into a cable that led to a mobile recording assembly outside the operating room where arterial and venous blood pressures, arterial oxygen saturation, electrocardiographic lead II, respirations and dye-dilution curves were monitored and recorded photokymographically (fig. 2).

Recordings of pressure and saturation were made with the patient breathing room air and subsequently 100 per cent oxygen prior to the
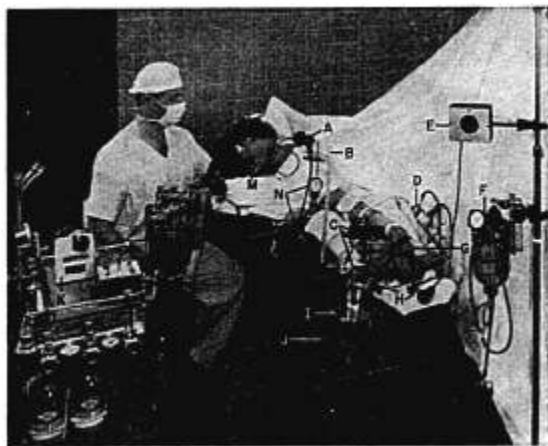

FIG. 1. Assembled apparatus for recording hemodynamic variables during operation. A. Strain-gauge manometer for recording respiration as pressure fluctuations in the anesthesia face-mask assembly. B. Buret for measuring rate of blood flow through the cuvette oximeter during recording of dye-dilution curves from indwelling needle in the radial artery connected to a strain-gauge manometer-cuvette oximeter assembly C. for recording radial arterial pressure, arterial oxygen saturation and arterial dye-dilution curves. D. Strain-gauge manometer and cardiac catheter for recording central venous pressure. E. Intercommunication speaker and microphone assembly for communication with the panel operator at the recording cart. F. Wash-bottle assembly containing heparinized saline under pressure for in vivo calibration and for flushing the manometer systems. G. ECG electrode on the right hand. H. Signal markers for signaling the injection of dye, the rate of blood flow through the cuvette oximeter and other important events. I. Discard basin to collect discarded saline and blood from the cuvette during sampling of blood. J. Junction box and cable leading to the recording cart outside the operating room. K. Beckman oxygen analyzer to measure the oxygen concentration in the anesthetic mixture. L. Junction box for electro-encephalographic leads. M. Earpiece oximeter. N. Tycos manometer and hand-bulb assembly for inflation of the pressure capsule in the earpiece oximeter.

induction of anesthesia; samples of arterial blood were withdrawn during these periods for determination of pH and for Van Slyke analysis of oxygen saturation to check the accuracy of the calibration of the earpiece and cuvette oximeters. With the patient breathing 100 per cent oxygen prior to induction of anesthesia, an arterial dye-dilution curve was recorded. Induction was begun by means of a 2.5 per cent solution of thiopental sodium given intravenously, followed by inhalation of a mixture of equal quantities of nitrous oxide and oxygen given at a rate of 8 liters per minute and maintained throughout the period of anesthesia.
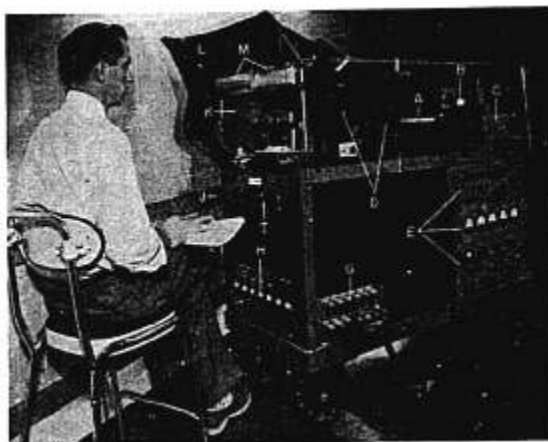

FIG. 2. Photokymographic assembly outside the operating room. A. Photokymographic camera. B. Galvanometer light source. C. Gearbox to adjust speed of camera. D. Mirrors for dividing and reflecting beams of light onto the photographic paper in the camera and onto the visual monitoring scale in front of the panel operator. E. Power supply and galvanometer light intensity controls. F. Cable connecting recording camera to transducers in the operating room. G. Junction box. H. Strain-gauge and oximeter power-supply controls. I. Oximeter control panels. J. Intercommunication switch. K. Galvanometers, for oximeters and strain gauges. L. Cloth hood to shield stray light and to facilitate reading of the visual scales. M. Visual monitoring scales.

Arterial and venous pressure, arterial oxygen saturation and arterial dye-dilution curves were recorded at various levels of anesthesia as determined by the electro-encephalogram (figs. 3 and 4). Cardiac output was calculated from these recordings by the conventional Hamilton method (22–25) (figs. 5 and 6). Stroke volume, central blood volume and peripheral resistance also were calculated, and samples of arterial blood were withdrawn prior to each dye-dilution curve for determination of pH.

RESULTS

Thirteen patients were studied, 12 of whom were women. Their ages ranged from 35 to 71 years, with a mean of 53. Eleven women underwent operations on the breast, whereas the twelfth underwent a vaginal plastic procedure; the man had an operation for removal of a hydrocele (table 1). All these patients were considered clinically to have a normal cardiovascular status. Factors such as positional changes, the open thorax and use of muscle relaxants were intentionally avoided.
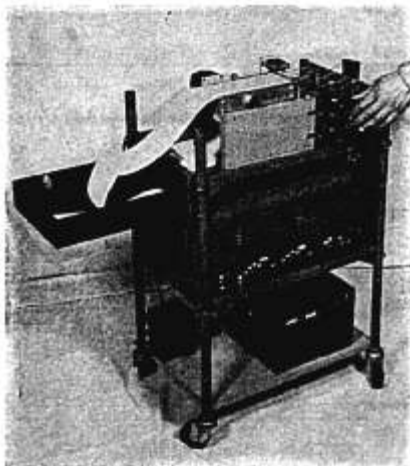

FIG. 3. Single-channel electro-encephalograph used during thiopental sodium and nitrous oxide anesthesia. This recording assembly was located outside the operating room and connected to the patient via a junction box and a shielded cable 20 feet in length.

Cardiac Index.—The mean cardiac index of all patients breathing 100 per cent oxygen prior to induction of anesthesia was 3.6 liters per minute per square meter of body surface, with a range from 2.7 to 5.0 liters. The cardiac index in all patients tested was smaller at electro-encephalographic levels 1 and 2 of thiopental sodium and nitrous oxide anesthesia than it had been when the patients were awake and breathing 100 per cent oxygen; the mean decrease was 24 per cent. When anesthesia was deepened to levels 3, 4 and 5 (burst-suppression levels) (15), the cardiac index showed a further significant decrease of 24 per cent of the values at levels 1 and 2 (p < .001). However, on return to levels 1 and 2 after deep anesthesia, the cardiac index

showed no significant systematic return to higher values over an average period of 25 minutes (fig. 7 and table 2).

Stroke Index.—The mean stroke index of all patients breathing 100 per cent oxygen prior to induction of anesthesia was 42 cc. per cardiac beat per square meter of body surface, with a range from 33 to 54 cc. A significant decrease in stroke index was found at anesthesia levels 1 and 2 (p < .001), with a further significant decrease in stroke index when anesthesia was deepened to burst-suppression
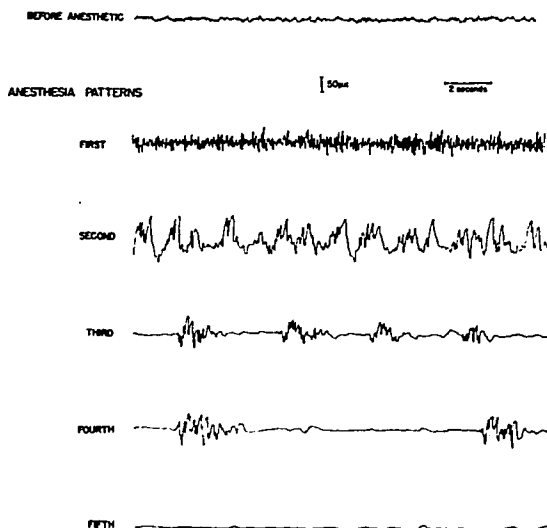

FIG. 4. Electro-encephalographic patterns associated with the various levels of thiopental sodium anesthesia. (Reproduced from Kiersey and associates (15), with permission of the editor of the British Journal of Anaesthesia.)

levels as determined by the electro-encephalogram (p < .02). On return to lighter levels of anesthesia, an increase in stroke index was observed that was on the border of statistical significance (p < .05). The mean decrease in stroke index at light levels of anesthesia was 27 per cent, while a further mean decrease of 24 per cent of the values at levels 1 and 2 was observed when anesthesia was deepened to levels 3, 4 and 5. On return to light levels after deep anesthesia had been attained, a mean increase of 18 per cent was observed (fig. 8 and table 3).

Intra-arterial Blood Pressure.—A significant decrease of the mean pressure in the radial artery was found in all patients being induced to electro-encephalographic levels 1 and 2 of anesthesia (p < .001). This mean decrease was 19 per cent, with a range from 5 to 42 per cent. When the depth of anesthesia was increased to levels 3, 4 and 5, no further significant systematic change in mean arterial pressure was observed (p < .2). No further significant systematic change in pressure (p < .3) was observed in an average of 25 minutes when the patients were allowed to return to lighter levels of anesthesia (fig. 9).

A highly significant difference (p < .001) in the decrease of systolic arterial pressure was observed, depending on whether induction of
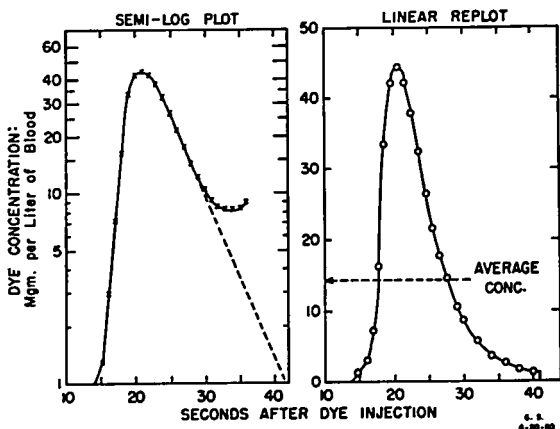

FIG. 5. Typical semilogarithmic plot and linear replot utilized to calculate cardiac output from an arterial dye-dilution curve recorded at operation. The dashed line in the left-hand figure represents the extrapolation of the curve to eliminate the effects of recirculation and to obtain the initial passage time of the dye. The figure on the right is a linear replot, using the values obtained from the extrapolated curve. Cardiac output was calculated by using the formula $(60 \times I)/CT$ (Hamilton), where $I =$ amount of dye injected in mg. $C =$ average concentration of dye in mg. (obtained by planimetry) and $T =$ time in seconds for the initial passage of dye.

anesthesia was rapid or slow (less than 2 cc. of solution per minute). The mean decrease in the systolic arterial pressure was 23 per cent in those patients receiving rapid induction compared to a mean decrease of 8 per cent in those patients in whom induction was slow (table 4). In all patients, the decrease in blood pressure occurred within 90 seconds after the onset of induction; some return toward preanesthetic levels was noted in all patients within 3 minutes. The decrease in
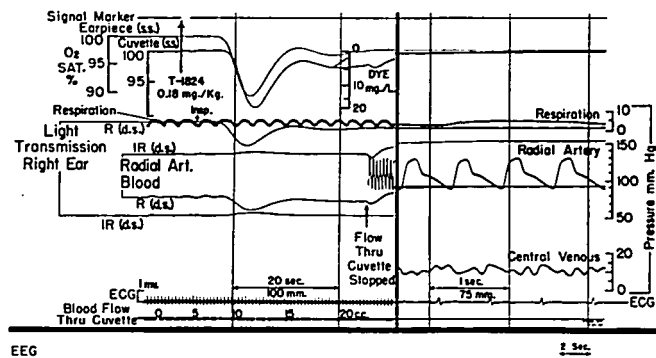
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Fig. 6. Photokymographic record showing dye-dilution curves and other variables recorded during anesthesia and operation. The break in the signal-marker line indicates the injection of Evans blue (T–1824) through the catheter into the central venous circulation; this is followed by a delay representing the most rapid transit time of dye from the site of injection to the recording site. This delay is followed by a rapid downward deflection, representing increasing concentration of dye, succeeded by a slower upward deflection, or decreasing concentration, as the dye is partially cleared from the heart, lungs and arterial system proximal to the sampling site. A smaller secondary deflection represents systemic recirculation of the dye. At the vertical black line, the speed of the camera was shifted from 5 mm. per second to 75 mm. per second. This faster speed facilitates study of the details of the contours of the arterial and venous pressure pulses. At the bottom is a typical electro-encephalographic tracing taken during thiopental sodium and nitrous oxide anesthesia and operation.

TABLE 1

Patients Studied During Thiopental Sodium
and Nitrous Oxide Anesthesia

| Patient	Age, Years	Sex	Weight, Pounds	Body Surface Area, Square Meters	Type of Operation
1	35	F	131	1.63	Radical mastectomy
2	71	M	125	1.68	Excision of spermatocele
3	61	F	160	1.72	Radical mastectomy
4	40	F	117	1.52	Vaginal plastic
5	65	F	198	1.94	Radical mastectomy
6	55	F	170	1.88	Simple mastectomy
7	54	F	138	1.64	Radical mastectomy
8	65	F	145	1.80	Radical mastectomy
9	48	F	120	1.54	Simple mastectomy
10	37	F	118	1.56	Biopsy of breast and excision of tumor
11	43	F	120	1.59	Biopsy of breast and excision of tumor
12	62	F	100	1.42	Radical mastectomy
13	51	F	125	1.56	Radical mastectomy
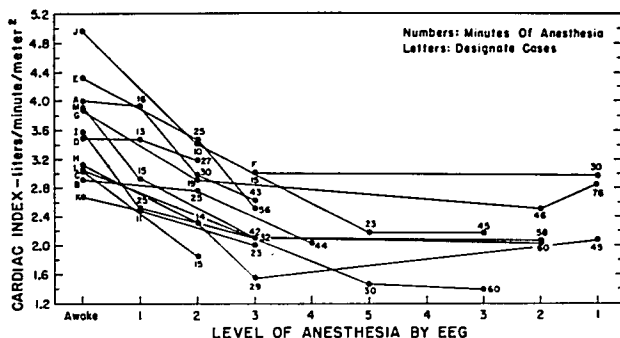
FIG. 7. Changes in cardiac index during thiopental sodium and nitrous oxide anesthesia. Note the decrease in cardiac index during induction to light anesthesia (levels 1 and 2), followed by a further decrease when anesthesia was deepened to burst-suppression depths (levels 3, 4 and 5). After deep anesthesia, the return to lighter levels was not accompanied by a significant systematic increase in cardiac index.

pressure was greatest in those patients who had the highest mean pressures prior to induction of anesthesia.

Cardiac Rate.—Significant systematic changes in cardiac rate were not observed in patients during induction of anesthesia, during deep anesthesia or during emergence from deep anesthesia. However, the rate in 1 patient increased from 84 to 150 beats per minute when she

TABLE 2

CARDIAC INDEX * AT DIFFERENT ELECTRO-ENCEPHALOGRAPHIC LEVELS
OF THIOPENTAL SODIUM AND NITROUS OXIDE ANESTHESIA

Patient	Awake	Depth of Anesthesia		
		Level 1,2 →	Level 3,4,5 →	Level 1,2
1	4.0	3.0	2.6	—
2	2.9	2.8	2.0	—
3	3.0	1.9	—	—
4	3.5	3.5	—	—
5	4.3	3.5	2.5	—
6	—	—	3.0	3.0
7	3.9	2.9	2.5	2.9
8	3.1	2.3	1.6	2.1
9	3.6	2.5	2.1	2.1
10	5.0	3.4	2.2	2.2
11	2.7	2.5	2.0	—
12	3.1	1.5	—	1.4
13	3.9	3.0	2.1	2.1

* Expressed as liters per minute per square meter of body surface.

TABLE 3

STROKE INDEX * AT DIFFERENT ELECTRO-ENCEPHALOGRAPHIC LEVELS
OF THIOPENTAL SODIUM AND NITROUS OXIDE ANESTHESIA

Patient	Awake	Depth of Anesthesia		
		Level 1,2 →	Level 3,4,5 →	Level 1,2
1	54	28	22	—
2	42	25	30	—
3	33	22	—	—
4	39	38	—	—
5	51	41	16	—
6	—	—	37	41
7	42	30	32	29
8	39	30	25	32
9	40	33	24	24
10	49	38	20	26
11	38	36	27	—
12	34	18	—	—
13	48	36	18	21

* Expressed as cubic centimeters per cardiac beat per square meter of body surface.

entered deep levels of anesthesia. No correlation could be made in
this group of patients between cardiac rate and cardiac index.

"*Central Blood Volume.*"—All patients showed a significant de-
crease in the estimated "central blood volume" as calculated from
the Stewart (26) formula when a comparison was made between values
in patients who were awake and values in those undergoing levels 1
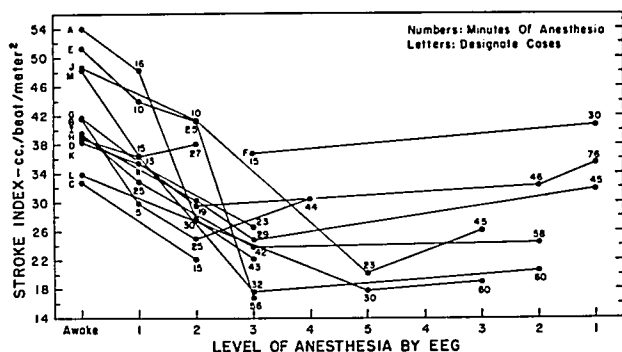
FIG. 8. Changes in stroke index during thiopental sodium and nitrous oxide anesthesia.
Note the decrease in stroke index during induction to light anesthesia (levels 1 and 2),
followed by a further decrease when anesthesia was deepened to burst-suppression depths
(levels 3, 4 and 5). After deep anesthesia, the return to lighter levels was accompanied by
a systematic increase in stroke index that was on the border of statistical significance.

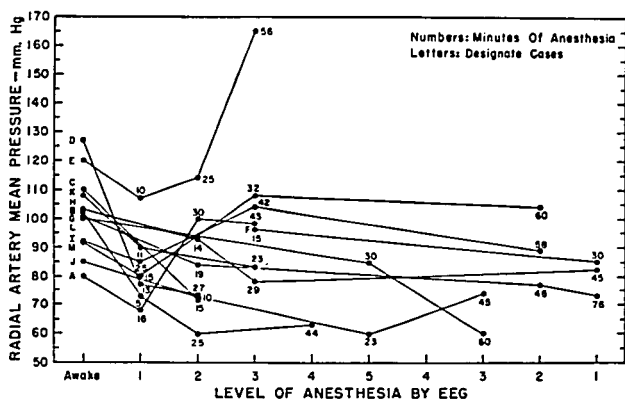FIG. 9. Changes in mean pressure in the radial artery during thiopental sodium and nitrous oxide anesthesia. Note the decrease in pressure in all patients during induction to light anesthesia. When anesthesia was deepened and subsequently lightened, systematic significant change in pressure was not observed, although wide variability was noted.

TABLE 4

COMPARISON OF DECREASES IN INTRA-ARTERIAL BLOOD PRESSURE DURING
RAPID AND SLOW INDUCTION OF THIOPENTAL SODIUM ANESTHESIA

| Induction | Patient | Arterial Blood Pressure, Millimeters of Mercury | | | | | |
| | | Systolic | | | Diastolic | | |
		Awake	During Induction	Decrease	Awake	During Induction	Decrease
Rapid*	1	112	75	37	56	56	0
	2	168	115	53	60	55	5
	3	156	135	21	80	72	8
	4	170	147	23	93	90	3
	5	175	143	32	72	65	7
	6	165	124	41	84	80	4
	7	125	94	31	80	76	4
Slow†	8	120	118	2	75	70	5
	9	170	162	8	70	70	0
	10	109	100	9	70	68	2
	11	170	155	15	88	90	−2
	12	100	85	15	56	54	2
	13	150	135	15	72	68	4

* More than 7 cc. of 2½ per cent solution of thiopental sodium per minute.
† Less than 2 cc. of 2½ per cent solution of thiopental sodium per minute.

and 2 of anesthesia (p < .01). A further significant decrease in the estimated "central blood volume" occurred in all patients when anesthesia was deepened to levels 3, 4 and 5 (p < .001). However, on emergence from deep anesthesia, a significant increase was not observed in the central blood volume (p < .3). These values paralleled those obtained for the cardiac index.

Peripheral Resistance.—The mean peripheral resistance in the patients studied while awake and breathing 100 per cent oxygen was 1,450 dynes/sec./cm.$^{-5}$, with a range from 870 to 2,020 dynes. No significant change took place in peripheral resistance during induction to light levels of anesthesia (p < .8). However, when the depth of anesthesia was increased to levels 3, 4 and 5, a significant increase in the peripheral resistance was noted (p < .01), with a mean increase of 670 dynes/sec./cm.$^{-5}$. On emergence from deeper levels of anesthesia, no significant systematic change in the peripheral resistance was observed (p < .1).

Central Venous Pressure.—Unlike those values obtained when patients are under ether anesthesia (27), the changes in central venous pressure were equivocal during thiopental sodium and nitrous oxide anesthesia. Increases in central venous pressure on the border of statistical significance were observed during induction to light levels of anesthesia, while decreases also on the border of statistical significance were observed when anesthesia was deepened to levels 3, 4 and 5 (p values of .05 and .05, respectively). Significant systematic change in the central venous pressure was not observed on emergence from deep anesthesia.

Arterial Blood pH.—Determinations of the pH of arterial blood were made on a Cambridge research-model pH meter on all patients at different levels of anesthesia. The pH of arterial blood decreased in direct proportion to the depth of anesthesia determined electroencephalographically, unless respiration was assisted; it increased on emergence from anesthesia. At deep levels, values for pH as low as 7.25 were obtained. In those patients in whom apnea was produced, controlled respiration increased the pH (fig. 10).

Frequency of Respiration.—A significant increase in the frequency of respiration was noted in patients entering deep levels of anesthesia prior to the production of apnea (p < .01). In all other phases of the study, significant changes in the frequency of respiration were not observed.

Arterial Oxygen Saturation.—As already indicated, arterial oxygen saturation was continually monitored by use of an earpiece oximeter. At specific intervals during the study prior to each dye-dilution curve, samples of blood were withdrawn through a cuvette oximeter for determination of arterial oxygen saturation. Both oximeters were calibrated by Van Slyke analysis (28) of samples of blood drawn from many patients.

During induction, a mean decrease of 11 per cent in arterial oxygen saturation was noted in 3 patients in whom anesthesia was induced while they breathed room air. The saturation rapidly returned to values of more than 98 per cent as soon as the mixture of nitrous oxide and oxygen was added. No attempt was made to determine whether or not the saturation would return to preanesthesia levels if these patients were allowed to continue to breathe room air. However, in children undergoing diagnostic cardiac catheterization, similar decreases in arterial oxygen saturation have been observed, with return to the preanesthesia level within a relatively short period after administration of pentothal (29).
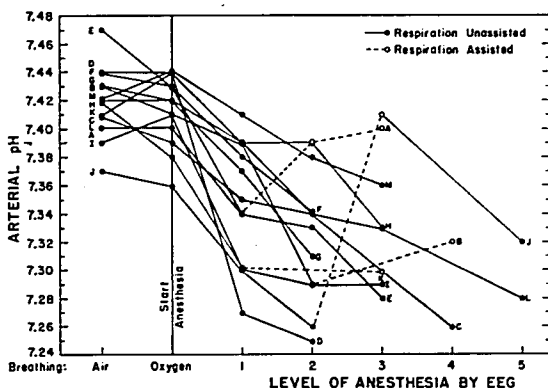

FIG. 10. Changes in pH of arterial blood during thiopental sodium and nitrous oxide anesthesia. Note that the pH decreased in proportion to the depth of anesthesia. When respiration was assisted, however, the values for pH did not decrease but either remained constant or returned toward normal.

The remaining 10 patients in this study underwent induction with thiopental sodium after they had breathed 100 per cent oxygen for a minimum of 8 minutes. Decrease in arterial oxygen saturation did not occur in any of these patients. After variable periods with the patients breathing a mixture of equal quantities of nitrous oxide and oxygen at a flow of 8 liters per minute, cuvette determinations on radial arterial blood likewise showed no decrease in oxygen saturation. At this rate of flow, the percentage of oxygen in the anesthetic mixture, as determined with a Beckman model A oxygen analyzer, was maintained at a very constant level.

Effects of Assisted and Controlled Respiration.—Anesthesia was sufficiently deep in 5 of the patients to produce a great decrease in ventilatory effort; therefore, respiration was manually assisted.

Values for the pH of arterial blood increased in all of the patients so assisted. The maintenance of positive pressure in the airway was associated with a decrease of pressure in the radial artery and an increase in the central venous pressure; these values immediately returned to original levels on release of the positive pressure.

Electrocardiographic Changes.—Twelve of the 13 patents experienced no electrocardiographic changes even at deep levels of anesthesia. A bigeminal pulse developed in 1 patient whose blood pressure increased suddenly from 154 millimeters of mercury systolic and 88 diastolic to 210 millimeters systolic and 127 diastolic as anesthesia was deepened to electro-encephalographic level 3. This disappeared as soon as anesthesia was permitted to return to levels 1 and 2.

COMMENT

The data presented are in accord with those of Li and associates and of Johnson. The progressive decrease in cardiac index with increasing depth of anesthesia as determined by the electro-encephalogram suggests that deep levels of thiopental sodium and nitrous oxide anesthesia are best avoided. This is especially evident when one notes the lag in the increase in the cardiac index on emergence from deep anesthesia. It appears that anesthesia beyond electro-encephalographic levels 1 and 2 would be especially dangerous for the cardiac patient, if such patients respond similarly. However, this study does not take into account the metabolic changes with subsequent decreased oxygen demand of the body under this form of anesthesia. An argument may be advanced that the effects of deep anesthesia induced by thiopental sodium may depress the metabolic functions of the body to such a degree that the low values for blood flow noted in this study are still sufficient to care for the body's needs.

The decrease in cardiac index was paralleled by a decrease in the estimated "central blood volume." At light levels of anesthesia, no significant change in peripheral resistance was observed, while the blood pressure decreased significantly in all patients. This would be compatible with vasodilatation, pooling of blood in the periphery and a decrease in venous return to the heart. This is borne out further by the decrease in stroke volume observed at these and deeper levels of anesthesia.

However, other factors may come into play at deeper levels of anesthesia. Since the central venous pressure did not increase but actually decreased somewhat during deep anesthesia, it is unlikely that myocardial insufficiency is one of these factors, although a transient increase in venous pressure was observed in those patients undergoing rapid induction. It may be possible that the retention of carbon dioxide and changes in pH of arterial blood at deeper levels of anesthesia affect the blood pressure and peripheral resistance so as to decrease further the cardiac output.

As shown by Patrick and Faulconer, respiratory acidosis develops during thiopental sodium anesthesia. The decrease of pH of arterial blood was directly proportional to the depth of anesthesia in the patients studied, unless respiration was assisted; at deep levels, the pH decreased to dangerously low values. From these data, it appears advisable to assist respiration at all levels of thiopental sodium and nitrous oxide anesthesia to keep the pH of arterial blood within normal limits.

The significantly smaller decrease in systolic blood pressure in those patients receiving slow induction with thiopental sodium as contrasted with the change in patients undergoing rapid induction suggests that the slow method is preferable. No excess of this anesthetic agent was used during slow induction as compared to the amounts used during the rapid technique. No comparison was made between these two groups of patients regarding changes in cardiac output during the phase of induction only.

The decrease in arterial oxygen saturation observed in those patients in whom anesthesia was induced while they breathed room air is in accord with the work of McClure and associates (30), Penrod and Hegnauer (31), and Johnson. However, the absence of a decrease in arterial oxygen saturation in patients in whom anesthesia was induced after they breathed 100 per cent oxygen for 8 minutes suggests that preliminary administration of 100 per cent oxygen may be advisable in the use of any agent that depresses respiration.

As stated previously, electrocardiographic changes were not observed in 12 of the 13 patients studied. The afore-mentioned bigeminal pulse that developed with a great increase in blood pressure suggests that this increase, not the state of anesthesia, was the cause of the arrhythmia. This is in accord with the work of Gruber's group.

SUMMARY AND CONCLUSIONS

Hemodynamic variables were studied at different electro-encephalographic levels of thiopental sodium and nitrous oxide anesthesia in 13 patients who had a clinically normal cardiovascular status.

Cardiac output decreased with increasing depth of anesthesia, and the return to preanesthetic values was slow.

A decrease in blood pressure was observed in all patients during induction of anesthesia. A significantly greater decrease in blood pressure occurred after rapid induction than was noted after slow induction.

Values for the pH of arterial blood decreased in proportion to the depth of anesthesia, unless respiration was assisted.

Changes in estimated values for "central blood volume" paralleled the decrease in cardiac index and stroke volume. Vasodilatation, pe-

ripheral pooling of blood and decreased venous return to the heart would be compatible causative mechanisms for these effects.

Central venous pressure did not increase with deep anesthesia; thus, myocardial sufficiency apparently was maintained.

Peripheral resistance was increased only after deep anesthesia was attained. This was compatible with the body's attempt to maintain blood pressure in the face of a low cardiac output.

A decrease in arterial oxygen saturation was noted when induction was attempted while the patients breathed room air. This decrease was not observed when patients breathed 100 per cent oxygen.

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NOTICE OF THE ANNUAL MEETING

THE AMERICAN SOCIETY OF ANESTHESIOLOGISTS, INC.

OCTOBER 30 TO NOVEMBER 3, 1955

BOSTON, MASSACHUSETTS