presumptuous. The never-ending progression of new drugs and techniques changes anaesthetic practice almost overnight. Over the years, since the use of analgesics became widespread, one can trace an evolution that seems to point towards greater dependence upon nitrous oxide and less upon the analgesic. There is always the possibility that new methods will enable us to measure the effects of the analgesics during anaesthesia more accurately than hitherto. We will then have a more exact knowledge of what part the analgesic really plays, whether one drug is preferable to another, or even whether these drugs ought to be discarded altogether.

Summary

The ability to use nitrous-oxide–oxygen as a safe general anaesthetic was increased with the introduction of thiopentone. The advent of curare further widened the scope of nitrous oxide anaesthesia. Too much reliance upon thiopentone supplementation led to frequent complications, while too much reliance on curare to maintain immobility led to patient-awareness during an otherwise inadequate anaesthetic.

Pethidine was originally used to increase the depth of nitrous-oxide–oxygen anaesthesia. Investigations of pethidine as a supplement to nitrous oxide revealed many advantages associated with its use. Other narcotic analgesics such as levorphanol, alphaprodine, and ketobemidone were soon investigated as supplements.

Throughout these investigations the great shortcoming has been the lack of any clear-cut criterion for the assessment of analgesia during general anaesthesia. Such clinically measurable values as the mg./minute thiopentone requirements, the reaction time, and the duration of post-operative analgesia have been used both to measure and to compare the efficacy of analgesic supplements. They give valuable but incomplete information in this direction.

Many narcotic analgesics have been used successfully as supplements to nitrous oxide anaesthesia. Only when more precise methods for measuring the effects of these supplements during anaesthesia are found, however, will we have an idea of their proper role in anaesthesia, whether one drug is preferable to another, or whether they need be used at all.

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REFERENCES

Auld, W. (1952). Anaesthesia, 7, 161.
Blazeby, R. H. (1951). Ibid., 6, 168.
Boisson, P. (1952). Acta anaesth. belg., 3, 7.
Brodie, B B., Mark, L. C., Papper, E. M., Lief, P. A., Bernstein, E., and Rovenstine, E. H. (1950). J. Pharmacol., 98, 85.
Brotman, M., and Cullen, S. C. (1949). Anesthesiology, 10, 696.
—— —— and Wilkins, D. S. (1950). Ibid., 11, 527.
Brown, A. K. (1952). British Medical Journal, 2, 1331.
—— (1953). Anaesthesia, 8, 26.
—— (1954). British Medical Journal, 2, 967.
Crawford, J. H., and Binning, R. (1953). Ibid., 1, 272.
Cullen, S. C. (1950). Ibid., 2, 276.
Duguid, A. M. E., and Heathcote, R. St. A. (1940). Quart. J. Pharm., 13, 318.
Eisleb, O., and Schaumann, O. (1939). Dtsch. med. Wschr., 65, 967.
Foldes, F. F., and Swerdlow, M. (1956). British Medical Journal, 1, 686.
—— —— Lipschitz, E., Weber, G., and Pirk, L. A. (1955). Canad. Anaesth. Soc. J., 2, 362.
Franks, E. H. (1954). Brit. J. Anaesth., 26, 418.
Griffiths, H. F. (1950). British Medical Journal, 2, 763.
Gross, E. G., Brotman, M. A., Nagyfy, S. F., Sawtelle, W. W., and Zager, L. L. (1949). Fed. Proc., 8, 297.
—— Holland, H. L., and Schueler, F. W. (1948). J. appl. Physiol., 1, 298.
Hamilton, W. K., and Cullen, S. C. (1955). Anesthesiology, 16, 22.
Hardy, J. D., Wolff, H. G., and Goodell, H. (1940). J. clin. Invest., 19, 649.
Johnson, P. D. (1951). British Medical Journal, 2, 705.
Johnstone, M. (1951). Ibid., 2, 943.
Lancaster, F. M., and Levin, J. (1956). Ibid., 1, 381.
Lee, R. E., and Hasegawa, A. K. (1950). J. Pharmacol., 98, 20.
—— and Pfeiffer, C. C. (1951). J. appl. Physiol., 4, 193.
Loeschcke, H. H., Sweel, A., Kough, R. H., and Lambertsen, C. J. (1953). J. Pharmacol., 108. 376.
McDermott, T. F., and Papper, E. M. (1950). N.Y. St. J. Med., 50, 1721.
Meals, W. G. (1948). Anesthesiology, 9, 644.
Murphy, M. (1951). Irish J. med. Sci., p. 535.
Mushin, W. W. (1951). Proc. roy. Soc. Med., 44, 840.
—— (1955). Joseph Clover Lecture, Royal College of Surgeons. Personal communication.
—— and Rendell-Baker, L. (1949). British Medical Journal, 2, 472.
—— —— (1950). Brit. J. Anaesth., 22, 235.
Neff, W., Mayer, E. C., and DeLaLuz Perales, M. (1947). Calif. Med., 66, 67.
—— —— and Thompson, R. (1950). British Medical Journal, 1, 1400.
Pearce, C. (1951). Brit. J. Anaesth., 23, 205.
Prescott, F., Ranson, S. G., Thorp, R. H., and Wilson, A. (1949). Lancet, 1, 340.
Randall, H. S., Belton, M. K., and Leigh, M. D. (1952). Canad. med. J., 67, 311.
Ruben, H., and Andreassen, A. K. (1951). Brit. J. Anaesth., 23, 33.
Ruddell, J. S. (1950). Lancet, 1, 953.
Severinghaus, J. W. (1953) Univ. of Pennsylvania, Seminar: " Recent Advances in Pharmacology."
Siker, E. S., Foldes, F. F., Pahk, N. M., and Swerdlow, M. (1954). Brit. J. Anaesth., 26, 405.
Swerdlow, M., Foldes, F. F., and Siker, E. S. (1955). Amer. J. med. Sci., 230, 237.
Thompson, R. C., and Neff, W. B. (1950). Cur. Res. Anesth., 29, 77.
Verne, D. (1952). J. oral. Surg., 10, 152.
Wilkins, D. S., Cullen, S. C., and Brotman, M. (1951). Anesthesiology, 12, 145.
Wolfers, P. (1953). British J. Anaesth., 25, 244.

NEONATAL BLOOD PRESSURE IN RELATION TO MATURITY, MODE OF DELIVERY, AND CONDITION AT BIRTH

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This paper compares the average systolic blood pressure at birth of groups of infants differing in various respects. It also describes the course of the pressure during the first few months of life.

Method and Material

The systolic blood pressure was estimated by palpating the brachial artery below an inflatable cuff. The cuff was 2.5 cm. wide, as recommended by Robinow et al. (1939). The artery was palpated in the antecubital fossa—this is more reliable than palpation of the radial artery. The average of two or three readings, taken with the child quiet, was recorded. Pressures were measured within an hour of birth and often much sooner. Further measurements were made on the first, third, and ninth days after delivery, at 3 and 6 weeks, and at 3 and 6 months.

Most of the neonatal pressures were measured by one observer, and the later pressures by the other, but there were frequent opportunities for cross-checking. There was always good agreement.

Initially, all cases in which one of us could record the pressure within an hour of delivery were included. Later only abnormal cases were studied. For analysis the infants were divided into groups as described in the results. The groups are mutually exclusive.

Results

Unless otherwise stated, the figures quoted are the mean of all the readings in a group, and significance has been calculated by the t test.

Normal Deliveries

No Anoxia.—There were 54 normal pregnancies and spontaneous deliveries in the series. The mean systolic pressure rose slowly from 69 mm. Hg at birth to 93 mm. Hg at 6 months; the rise was most rapid during the first month

(Fig. 1, Table I). The results were analysed to determine what effect drugs given to the mother during labour had on the neonatal blood pressure ; the administration of morphine, pethidine, and barbiturates was without influence upon the values obtained.
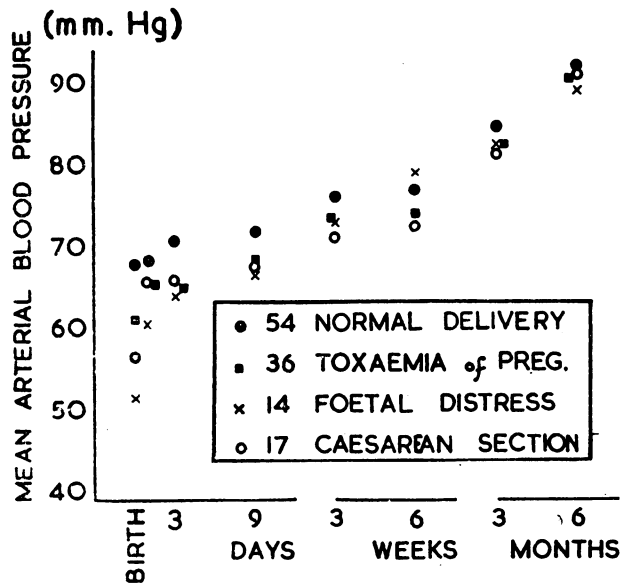

FIG. 1.—Mean arterial blood pressures at birth and during the first few months of life. (Normal infants.)

TABLE I.—*Normals* (54 *Babies*)

Age	Mean Systolic Pressure (mm. Hg)	S.D.
Birth　..　..　..	69	6
1 day　..　..　..	70	5
3 days　..　..	72	6
9 ,,　..　..	73	6
3 weeks　..　..	77	5
6 ,,　..　..	78	3
3 months　..　..	86	5
6 ,,　..　..	93	5

TABLE II.—*Neonatal Asphyxia* (14 *Babies*)

Age	Mean Systolic Pressure (mm. Hg)	S.D.	P
Birth　..　..	53	3	0·001
1 day　..　..	62	4	0·001
3 days　..　..	65	4	0·001
9 ,,　..　..	68	5	0·01
3 weeks　..　..	74	5	—
6 ,,　..　..	80	7	—
3 months ..　..	83	6	—
6 ,,　..　..	90	4	—

Foetal Anoxia.—There were 14 cases of anoxia which were classified as "white asphyxia." In each case one of us was present at the delivery and measured the blood pressure as soon as possible. All these cases were either of spontaneous or of forceps delivery. Table II shows that the mean systolic pressure—53 mm. Hg at birth—is lower than in normal babies at birth, the difference, P>0.001, being highly significant. It is also shown that this pressure remained significantly, lower than normal until discharge, but was within the normal range by 6 weeks. One of the infants included in this series, with a mean systolic pressure of 50 mm. Hg, died soon after birth.

Abnormal Deliveries

Breech.—The blood pressure was measured in six breech deliveries only ; one of these was also premature, and died on the second day. The mean systolic pressure at birth fell in the lower range of the value for normal deliveries, but was normal within three to five days.

Forceps.—In this group there were cases of both " low " and " medium," but none of " high," forceps delivery.

Taking the whole group, the mean systolic pressure at birth was 65 mm. Hg, which is not significantly lower than that of babies born spontaneously. The systolic pressure in these cases remained slightly but not significantly lower than normal until the third week.

Caesarean Section.—In these cases the mean systolic pressure at birth was 58 mm. Hg, significantly lower than in normals. In most of them the infant was, to all appearances, in good condition and there was no correlation between the blood pressure and the time taken for respiration to be established. The pressure rose during the first day, but tended to remain lower than in spontaneous deliveries for three months ; the difference, however, was not significant after three weeks. The caesarean sections were done for a variety of reasons, the majority being elective.

Abnormal Pregnancy

Pre-eclamptic Toxaemia.—The criterion for inclusion in this group was that two of the following signs must be present in the mother : oedema, albuminuria, or blood pressure of 140/90 mm. Hg or more. None of the babies in this group were included in any of the other groups, and
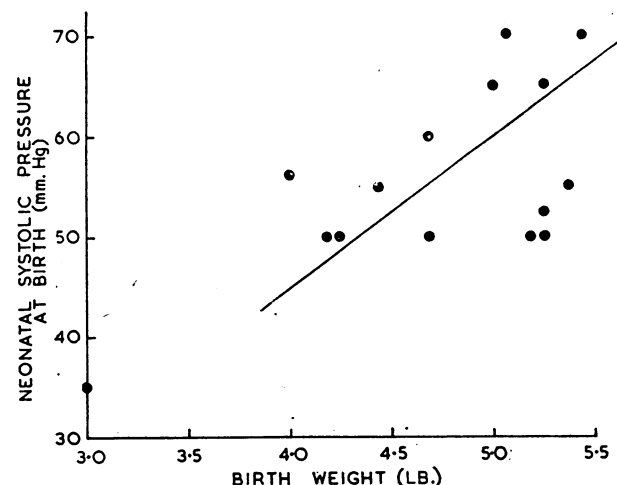

FIG. 2.—Neonatal systolic pressure in relation to birth weight. (Premature infants.)

the deliveries were both normal and abnormal. The mean systolic pressure of all babies born of toxaemic mothers was 62 mm. Hg, significantly lower than that found in the normal neonate. If, however, the method of delivery is taken into account and the blood pressure in each group — normal, caesarean section, and forceps — in the presence of pre-eclamptic toxaemia is compared with that in the corresponding groups without toxaemia discussed above, there is no significant difference. Pre-eclamptic toxaemia therefore does not influence the neonatal blood pressure, and the lower mean value obtained for the whole group is due to the inclusion of babies born by caesarean section.
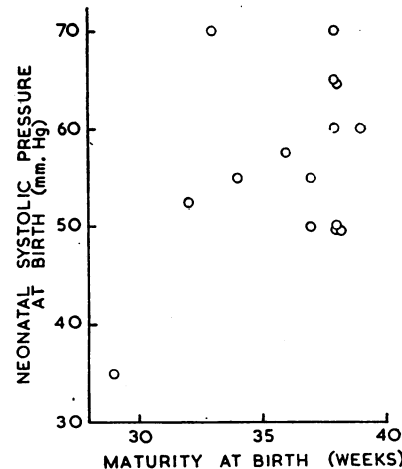

FIG. 3.—Neonatal systolic pressure in relation to maturity. (Premature infants.)

Premature Infants

This group contains all infants under 5½ lb. (2.5 kg.) birth weight, irrespective of the expected date of delivery. The systolic pressures that were recorded ranged from 35 to 70 mm. Hg, with a mean of 54. The lower end of the range was recorded in two infants who survived only three days, and it should also be noted that one infant with a pressure of 50 mm. Hg died on the first day. As shown in Figs. 2 and 3 the systolic pressure at birth was related to birth weight but not to maturity.

The premature infants attained values not significantly different from those of full-time infants by the third month, and the highest pressure recorded in any infant, 105 mm. Hg, at 6 months was in a prematurely born infant with a pressure of 50 mm. Hg at birth.

Twins

There were four sets of twins in this series. Where one was anoxic it had the lower blood pressure ; for instance, in one set of twins the first baby born did not breathe for five minutes and had a blood pressure 25 mm. Hg below its sibling, who breathed spontaneously at once.

Discussion

The method of palpation which has been used to measure the mean systolic pressure of the newborn is simple and reliable and gives readily reproducible values suitable for comparison. Woodbury, Hamilton, and Robinow (1938) found that there was a close correlation in infants between the mean systolic pressure measured by an intra-arterial needle in the umbilical artery and by palpation of the brachial artery only when a 2.5-cm. cuff was used. The method also compares well with the flush method, recently assessed by Reinhold and Pym (1955), Sullivan and Kobayashi (1955), and Forfar and Kibel (1956), but is somewhat easier.

The systolic pressures in this series are higher than those stated by most previous investigators, but are similar to those found by Robinow et al. (1939), whose values ranged from 60 to 80 mm. Hg, and by Schaffer (1955), who used an impedance plethysmograph to record pulsation distal to an occlusion cuff 4 cm. wide ; his values for systolic pressure ranged from 52 to 102 mm. Hg, but nowhere does he state at what time after birth the measurements were made.

The low readings obtained before the simultaneous comparison of the cuff method with direct intra-arterial puncture by Woodbury et al. were probably due to the wide cuff used, for a width of at least 4 cm. was the standard size for babies under 1 year of age (White House Conference, 1932). Balard (1912) using a Pachon oscillometer, found values of 55/35, and Reis and Chaloupka (1923), using a 6-cm. cuff and palpation, found values of 43 mm. Hg (range 32–58) on the first day, rising to 78 mm. (52–94) by the tenth day.

The effect of the different conditions prevailing at birth upon the neonatal blood pressure was studied by Reis and Chaloupka and by Bowman (1933). The former included in their series three toxaemic pregnancies, and found that this condition had no effect on the systolic pressure of the newborn ; this is in agreement with our own findings and those of Brasch (1949). Reis and Chaloupka also concluded that the more trauma to which the head was exposed— that is, the higher the forceps applied—and the larger the head, the higher would the blood pressure be. In two caesarean sections no effect on the blood pressure was noted, but there is no record at what interval after delivery these recordings were made. Bowman found in " cerebral " babies that the pressure was higher than normal, and was lowered by lumbar puncture. We were uncertain what was meant by this term, but if those cases emitting a " cerebral " cry are indicated, then in our series a lower systolic pressure than normal was found.

Summary

Palpation of the brachial artery below a 2.5-cm. inflatable cuff was used to measure the neonatal systolic blood pressure. The normal mean systolic pressure at birth was found to be 69 mm. Hg, rising to 93 mm. Hg at 6 months. Foetal anoxia and abnormal forms of delivery such as caesarean section caused a significant lowering of systolic pressure at birth ; the blood pressure of these babies recovered to normal levels within a few days. Drugs given to the mother before delivery and pre-eclamptic toxaemia had no effect on the neonatal systolic pressure at birth. The systolic pressure of premature babies was lower than that of full-term infants and was related to weight.

REFERENCES

Balard, P. (1912). C.R. Soc. Biol. Paris, 73, 483.
Bowman, J. E. (1933). Amer. J. Dis. Child., 46, 949.
Brasch, A. A. (1949). Arch. Dis. Childh., 24, 107.
Forfar, J. O., and Kibel, M. A. (1956). Ibid., 31, 126.
Reinhold, J., and Pym, M. (1955). Ibid., 30, 127.
Reis, R. A., and Chaloupka, A. J. (1923). Surg. Gynec. Obstet., 37, 206.
Robinow, M., Hamilton, W. F., Woodbury, R. A., and Volpitto, P. (1939). Amer. J. Dis. Child., 58, 102.
Schaffer, A. I (1955). Ibid., 89, 204.
Sullivan, M. P., and Kobayashi, M. (1955). Pediatrics, 15, 84.
White House Conference (1932). Growth and Development of the Child, Part II, p. 294. Century Co., New York.
Woodbury, R. A., Hamilton, W. F., and Robinow, M. (1938). Amer. J. Physiol., 122, 472.

CONGENITAL SPINAL PALSY

BY

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With the reduction of infant mortality and of deaths from intercurrent infections in early childhood, many children are surviving with severe congenital deformities of a degree which has not hitherto formed a major clinical or social problem. The medical profession and the educational world are now fully aware of the needs of the cerebral palsy group, but surprisingly little attention has been paid to those afflicted with paraplegia in minor or major degree, from interference with the spinal cord. These for the most part are infants with spina bifida, usually but not always with a meningeal hernia. There are some with compression lesions of the cord due to severe deformity of the vertebral column and a few with permanent damage from birth injury or infective myelitis.

This paper is concerned primarily with the spina bifida palsies (Fig. 1). In the general run of surgical practice there are two distinct groups. The one is occult, said to be very common and alternately blamed and exonerated as a cause of wet beds in the lives of otherwise innocent children. Careful examination of some of these occult cases exposes a neurological abnormality. In some, micturition symptoms arise at or after adolescence owing to the development of uterus or prostate.

The second group is cystic. Where the cyst is a simple meningocele there is sometimes no cord or nerve