thesia which was accompanied by maternal hypotension resulted in fetal acidosis. This acidosis had both a metabolic component and a respiratory component. The fetal deterioration was arrested and often corrected by administration of ephedrine sulfate to the mother.

An Improved Method for the Recognition of Atypical Plasma Cholinesterase. J. CRISPIN SMITH, PH.D., and FRANCIS F. FOLDES, *M.D., Division of Anesthesiology, Montefiore Hospital and Medical Center, Bronx, N. Y.* Determination of the dibucaine number (D.N.) has been recommended (Kalow, W., and Genest, K.: Canad. J. Biochem. *35:* 399, 1957) for detection of the various genotypes of human plasma cholinesterase (PChE). The D.N. allows a clear-cut differentiation between normal (NN) and atypcial (DD) homozygotes. The distinction between the NN and the heterozygote (ND), however, may be doubtful on occasions. Measurement of the relative rates of hydrolysis of benzoylcholine and acetylcholine (Rubinstein, H. M., and Dietz, A. A.: J. Lab. Clin. Med. *61:* 979, 1963) also makes it possible to distinguish the NN from the DD genotype. The detection of the ND genotype, however, has no advantages over the dibucaine test. In attempting to develop a test that would make possible the unequivocal differentiation of the NN, ND, and DD genotypes we searched for a compound which, in contrast to hitherto-examined substrates (Davies, R. O., Marton, A. V., and Kalow, W.: Canad. J. Biochem. *38:* 545, 1960), is hydrolyzed as fast or faster by the DD than by the NN enzyme. Tetracaine fulfilled this requirement. Because the relative hydrolysis rate of procaine by the DD genotype is the lowest of all substrates examined (Foldes, F. F., Foldes, V. M., Smith, J. C., and Zsigmond, E. K.: ANESTHESIOLOGY *24:* 208, 1963) determination of the ratios of the hydrolysis rates of procaine and tetracaine seemed suitable for a clear-cut differentiation between the NN, ND and DD genotypes. The method developed on this premise consists of the ultraviolet spectrophotometric determination of the hydrolysis of 5×10^{-5} M. solutions of procaine at 290 $m\mu$ and tetracaine at 313 $m\mu$ by a modification of Kalow's (J. Pharmacol. Exp. Therap. *104:* 122, 1952) method. The mean rates and standard errors of the hydrolysis of procaine by NN (23 subjects), ND (24) and DD (18) plasmas were 1.06 ± 0.07, 0.73 ± 0.06 and 0.16 ± 0.02 μmoles/ml. plasma/hour, respectively. The corresponding values for tetracaine were 0.29 ± 0.02, 0.37 ± 0.02 and 0.38 ± 0.03. The ratio of the hydrolysis rates of procaine and tetracaine multiplied by 100 (P/T ratio) revealed a highly significant difference between the NN, ND and DD groups. The means, and standard errors and ranges of the P/T ratios for the NN, ND and DD genotypes were 366 ± 8 (320–464), 196 ± 6 (152–239), and 40 ± 3 (16–59), respectively. There was no overlap of the P/T ratios of the three groups. The simple method described allows unambiguous identification of the three genotypes and for this reason is preferable to the method based on the determination of the D.N.

Hypoxemia in Shock and Myocardial Infarction: A Clinical Review. JAN D. SMITH, M.D., JEAN J. PENNINCKX, M.D., and PETER SAFAR, M.D., *Department of Anesthesiology, University of Pittsburgh School of Medicine, Pittsburgh, Penna.* Patients admitted to the Intensive Care Unit during a 12-month period (1966/67), whose respiratory care was guided by arterial blood gas determinations, were reviewed. Pa_{O_2}, Pa_{CO_2}, *p*Ha and bicarbonate were determined (a) during spontaneous breathing of room air (when possible); (b) during spontaneous breathing of 100 per cent oxygen ($F_IO_2 = 1$) for 20 minutes; (c) during IPPB/$F_IO_2 = 1$ (assisted respiration); and (d) during IPPV/$F_IO_2 = 1$ (controlled ventilation). Positive-pressure ventilation was with large tidal volumes (approximately 15 ml./kg.), to determine the reversibility of the shunt effect ($D_{A_a}O_2$ with $F_IO_2 = 1$). In most patients sampling was via an arterial catheter left in place for periods as long as a week. In two patients V_D/V_T was calculated from the Bohr equation. *Results:* I. *Cardiogenic Shock without Pulmonary Edema* (13 patients). Three/13 survived. Measurements were made within one hour after onset of shock. *p*Ha and bicarbonate indicated meta-

bolic acidosis in 11/13 patients. Pa_{CO_2} was 17–35 mm. Hg. The lowest Pa_{O_2} values in patients with $F_IO_2 = 1$ ranged between 40 and 145 mm. Hg. IPPB or IPPV/O_2 increased Pa_{O_2}. II. *Cardiogenic Shock with Pulmonary Edema* (nine patients). Two/9 survived. All had metabolic acidosis with hypocarbia (lowest Pa_{CO_2} value 15 mm. Hg). Pa_{O_2} values during spontaneous breathing of 100 per cent oxygen were 50–140 mm. Hg. As expected, IPPB or IPPV/$F_IO_2 = 1$ cleared pulmonary edema in most cases, and increased Pa_{O_2} (Miller, W. F., and Sproule, B. J.: Dis. Chest *35:* 469, 1959). III. *Uncomplicated Myocardial Infarction* (13 patients). All survived. Only one patient had evidence of metabolic acidosis. Pa_{CO_2} was variable. During spontaneous breathing of air, Pa_{O_2} values were 44–95 mm. Hg (in 9/13 patients, below 70 mm. Hg). During spontaneous breathing of 100 per cent oxygen, Pa_{O_2} was 154 to 550 mm. Hg. In four patients IPPB/$F_IO_2 = 1$ changed Pa_{O_2} from 295 to 430; 360 to 320; 340 to 500; and 310 to 430 mm. Hg, respectively. IV. *Miscellaneous Shock States* (seven patients). Three had oligemic shock, three septic shock, and one shock with diabetic acidosis. Two patients with oligemic shock and one with septic shock died. Blood gas changes were similar to those seen in cardiogenic shock. The lowest Pa_{O_2} values occurred in septic shock, 30 to 60 mm. Hg with IPPV/$F_IO_2 = 1$. *Comment:* Histologic changes in the lungs of subjects with shock as reported by others, include intra-alveolar and interstitial edema, hemorrhage, fibrin deposits, emboli, and thrombi. The hypoxemia observed seems to be the result of a combination of the following factors: (1) increased V_D/V_T, known to occur in oligemic shock (Gerst, P. H., Rattenborg, C., and Holaday, D. A.: J. Clin. Invest. *38:* 524, 1959), vasodilatation, hypotension (Askrog, V. F., Pender, J. W., and Eckenhoff, J. E.: ANESTHESIOLOGY *25:* 744, 1964), and cardiogenic shock (McNicol, M. W. *et al.:* Brit. Med. J. *2:* 1270, 1965); (2) V/Q mismatching; (3) diffusion block (*e.g.*, interstitial edema); (4) increased Q_S/Q_T, perhaps due to alveolar collapse from pulmonary congestion, edema, obstruction or lack of deep breaths (MacKenzie, *et al.:* Lancet *2:* 825, 1964); and (5) decrease in Q_T without change in Q_S/Q_T (decreased $P_{\bar{v}}O_2$). Hypoxemia due to factors (1) to (3), apparently predominant in uncomplicated myocardial infarction, can be corrected by simple oxygen enrichment (*e.g.*, $F_IO_2 = 0.5$). Hypoxemia due to increase in Q_S/Q_T can be partially reversed by IPPV/$F_IO_2 = 1$. Hypoxemia due to decreased Q_T needs circulatory support. The effect of these measures on tissue oxygenation is unpredictable unless cardiac output and oxygen consumption are measured simultaneously. (Supported by U. S. Army Contract No. DA-49-193-MD-2160.)

The Circulatory Effects of the Addition of Nitrous Oxide to Halothane Anesthesia in Man. N. TY SMITH, M.D., E. I. EGER, II, M.D., CHARLES E. WHITCHER, M.D., R. K. STOELTING, M.D., and T. F. WHAYNE, M.D., *Department of Anesthesia, Stanford Medical School, Palo Alto, and University of California, San Francisco, Calif.* Reports describing the circulatory effects of adding nitrous oxide to halothane anesthesia have been contradictory. Some claim stimulation; others, including clinical reports, claim depression. We have investigated this problem in nine normal unpremedicated 21-year-old male volunteer subjects. *Method:* Anesthesia was induced and maintained with halothane-oxygen. Ventilation was controlled with a fixed-volume ventilator to maintain alveolar P_{CO_2} between 30 and 35 mm. Hg. After a stable level of halothane-oxygen anesthesia had been obtained (0.8, 1.0, 1.6, or 2.0 per cent alveolar halothane concentration), the diluent was changed either to nitrous oxide/oxygen 75/25 or to air. Immediately before and 15 minutes after the change, dye-dilution cardiac outputs and occlusion plethysmograph forearm blood flows were measured, and arterial blood was withdrawn for measurement of blood gases and catecholamines by the Weil-Malherbe method. Electrocardiogram, heart rate, direct brachial arterial pressure, right atrial pressure, and external carotid artery pulse were recorded continuously. Several other parameters were calculated from these measurements. *Results:* The following are the per cent changes and standard deviations in cardiovascular variables