

non-blocked extremities did not change. Temperature increased significantly in the left hand. There were no changes in the other extremities.

COMMENT

Several recent publications discuss the many clinical and laboratory findings associated with cryofibrinogenemia.¹⁻⁴ While a complete discussion of this entity is beyond the scope of this report, the peripheral vasospastic element is of particular interest. Ritzmann,¹ based on the work of Gladner,⁵ suggests that the underlying systemic disease leading to cryofibrinogenemia increases the production of vasoactive peptides formed during the conversion from fibrinogen to fibrin. These, in turn, might predispose to the vasospasm noted. In addition, sympathetic overactivity probably makes the vasospasm more severe. The plethysmographic data presented are consistent with a dual mechanism. If sympathetic overactivity alone was responsible for the vasospasm, a much greater response in blood flow after block would have occurred, assuming the presence of a normal peripheral vascular system. In this patient biopsies did not reveal any primary vascular disease. The stellate

block, of course, would not effect the production of vasoactive peptides. However, the fact that blood flow did increase markedly, suggests that permanent sympathectomy should be beneficial. Indeed, several of Ritzmann's patients did benefit from sympathectomy. Surgical sympathectomy in the patient discussed here has been postponed at present because of complicating factors in the treatment of the as yet undiagnosed underlying condition.

REFERENCES

1. Ritzmann, S. E., Levin, W. C., Ivers, J. B., and Koch, J. L.: I. Cryoproteinemias. 2. Primary Cryofibrinogenemia—its association with cryopathy and telangiectasis, *Texas Rep. Biol. Med.* 21: 567, 1963.
2. Ritzmann, S. E., and Levin, W. C.: Cryopathies: A review, *Arch. Intern. Med.* 107: 754, 1961.
3. Jager, B. V.: Cryofibrinogenemia, *New Eng. J. Med.* 266: 579, 1962.
4. Ritzmann, S. E., Hamby, C., Cooper, R., and Levin, W. C.: I. Cryoproteinemias. 1. The characterization and assay of cryofibrinogen, *Texas Rep. Biol. Med.* 21: 262, 1963.
5. Gladner, J. A.: The molecular biology of the thrombin-fibrinogen interaction, *Proc. of the IX International Congress of Hematology*, Mexico City, Sept. 9-14, 1962 (abstract).

Spontaneous Bilateral Pneumothorax in a Newborn Infant

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The anesthesiologist frequently assumes the responsibility of resuscitating the depressed newborn infant. Respiratory difficulty of the newborn infant can be classified into two groups: (1) failure of the central nervous system to function with hypopnea or apnea, and (2) failure of the lungs or chest structures adequately to expand.¹ Awareness of the different causes of neonatal respiratory difficulty will facilitate the exact diagnosis, expedite the specific therapy, and enhance the prognosis.

The following is the report of a case of spontaneous bilateral pneumothorax in a newborn

infant delivered precipitously and without maternal anesthesia. Although the diagnosis was not established for three hours, good resuscitative measures and subsequent specific treatment led to prompt recovery.

CASE REPORT

A 28 year old para 2-0-0-2 in good general health, normal vital signs, and normal laboratory findings was admitted in active labor. She was given medication of meperidine 50 mg. + Largon 20 mg., injected intravenously. One and one-half hours later, after 6½ hours of first stage and 15 minutes of second stage labor, membranes ruptured spontaneously and delivery of a 4,000-gr. male infant followed. The infant gave a short weak cry and became progressively cyanotic. Suctioning of the mouth via a DeLee trap yielded thick mucus. Oxygen was blown over the infant's

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