

CASE REPORTS

Stellate Ganglion Block in a Patient with Cryofibrinogenemia

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Cryofibrinogenemia (the precipitation of fibrinogen when heparinized plasma is cooled) is a laboratory finding associated with thromboembolic phenomena, hemorrhagic tendencies, diabetes mellitus, carcinoma, myocardial infarction, collagen disorders and peripheral vasospastic conditions. Although the effect of sympathetic block had been previously mentioned in isolated cases,¹ no plethysmographic or thermographic (thermistor-recorded skin temperature) data were presented. The following report presents the effects of stellate ganglion block on a patient with cryofibrinogenemia.

CASE REPORT

The patient was a 41 year old white woman whose symptoms began in her teens when she suffered from intermittent attacks of Raynaud's phenomena. A duodenal ulcer, diagnosed at age 15, eventually led to a partial gastric resection at age 29. She also had four miscarriages in her early 20's. In the 8 years prior to admission, she had frequent attacks of paroxysmal auricular tachycardia and three episodes of pericarditis. For the past two years the patient complained of constant pain in all extremities, but especially in her legs.

On physical examination, the patient was thin, apprehensive, and appeared chronically ill. Her

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TABLE 1. Plethysmographic Data

	Blood Flow* (ml./minute/100 ml. digit volume)	
	Pre-block	15 Minutes After Left Stellate Ganglion Block
Left 2nd finger	3.0 (normal 18-48)	19.2 (normal 48-90)
Right 2nd finger	1.4	1.9
Left 2nd toe	not detectable (normal 6-20)	not detectable

* Room temperature 24.5° C ± 0.5° C.

TABLE 2. Thermographic Data

	Skin Temperature* (° C.)	
	Pre-block	15 Minutes Post-block
Left 3rd finger	24.4	31.8
Left 4th finger	24.6	31.6
Right 3rd finger	25.2	25
Right 4th finger	24.8	24.6
Left 1st Toe	24.6	24.6

* Room temperature 24.5° C ± 0.5° C.

skin, primarily around her mouth and on the dorsum of her hands, revealed small irregularly shaped venous lakes, which blanched on pressure. The skin was atrophic and an ulcer was noted over the left heel. She had a hearing deficit of the left ear. The liver was palpable two finger breadths below the costal margin and the tip of the spleen could be felt. There were generalized weakness of all four extremities and severe hyperalgesia over her legs. All extremities were cold, cyanotic, and it was questioned if peripheral pulses could be felt.

A number of special studies were performed. Peripheral nerve conduction time was decreased. An increased cryocrit as high as 30 volumes per cent (normal is less than 1.5 volumes per cent) was noted. Electrophoresis proved this to be all fibrinogen. Blood viscosity was increased as was total fibrinogen to 503 mg./100 ml. Other laboratory tests for pernicious anemia were not diagnostic. Muscle biopsy did not reveal the nature of the underlying disease. A presumptive diagnosis of a connective tissue disorder of undetermined type with secondary cryofibrinogenemia was made. The peripheral neuropathy was thought to be due to the vasospastic component of her disease. Stellate ganglion block was, therefore, suggested and performed as a diagnostic and potential therapeutic procedure.

The results of the plethysmographic and thermographic data are presented in tables 1 and 2. Peripheral blood flow preblock ranged from less than 20 per cent of normal to nondetectable. After left stellate ganglion block, blood flow increased sixfold. However, even this increase represented a flow of less than 40 per cent of that predicted in the sympathectomized arm. Blood flow in the

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.

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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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