References

- Fitzpatrick MM, Walters MDS, Trompeter RS, Dillon MJ, Barratt TM (1993) Atypical (non-diarrhea associated) hemolytic-uremic syndrome in childhood. J Pediatr 122: 532-537
- Churg J, Goldstein MH, Bernstein J (1989) Thrombotic microangiopathy including hemolytic uremic syndrome, thrombotic thrombocytopenic purpura, and postpartum renal failure In: Tisher CC, Brenner BM (eds) Renal pathology, vol 2. Lippincott, Philadelphia, pp 1081-1113
- Bailey JW, Sada E, Brass C, Bennett JE (1985) Diagnosis of systemic candidiasis by latex agglutination for serum antigen. J Clin Microbiol 21: 749-752
- Obayashi T, Tamura H, Tanaka S, Ohki M, Takahashi S, Kawai T (1986) Endotoxin-inactivating activity in normal pathological human blood samples. Infect Immun 53: 294–297
- Karmali MA, Petric M, Lim C (1985) The association between idiopathic hemolytic uremic syndrome and infection by verotoxinproducing *Escherichia coli*. J Infect Dis 151: 775
- Bartizal K, Abruzzo G, Trainor C, Krupa D, Nollstadt K, Schmatz D, Schwartz R, Hammond M, Blakovec J, Vanmiddlesworth F (1992) In vitro antifungal activities and in vitro efficacies of 1,3beta-D-glucan synthesis inhibitors L-671, 329, L-646, 991, tetra-

- hydroechinocandin B, and L-687, 781, a papulacandin. Antimicrob Agents Chemother 36: 1648-1657
- Medoff G, Kobayashi GS (1987) Fungal diseases. In: Feigin RD, Cherry JD (eds) Textbook of pediatric infectious disease, 2nd edn, vol 2. Saunders, Philadelphia, pp 1925–1949
- Filler SG, Ibe BO, Luckett PM, Raj JU, Edwards JE Jr (1991) Candida albicans stimulates endothelial cell eicosanoid production. J Infect Dis 164: 928–935
- Ghannoum MA, Filler SG, Ibrahim AS, Fu Y, Edwards JE Jr (1992) Modulation of interactions of *Candida albicans* and endothelial cells by fluconazole amphotericin B. Antimicrob Agents Chemother 36: 2239–2244
- Kustimur S (1992) The role of eicosanoids in the kidney damage induced by *Candida albicans*. Prostaglandins Leukot Essent Fatty Acids 47: 83–84
- Fitzpatrick MM, Shah V, Trompeter RS, Dillon MJ, Barratt TM (1992) Interleukin-8 and polymorphoneutrophil leukocyte activation in hemolytic uremic syndrome of childhood. Kidney Int 42: 951–956
- 12. Suranyi MG, Guasch A, Hall BM, Myers BD (1993) Elevated levels of tumor necrosis factor-alpha in the nephrotic syndrome in humans. Kidney Dis 21: 251–259

Literature abstract

Crit Care Med (1994) 22: 1747-1753

Dopamine suppresses pituitary function in infants and children

Greet Van den Berghe, Francis de Zegher, and Peter Lauwers

Objectives: Dopamine, a natural catecholamine with hypophysiotropic properties, is used as a first choice drug for inotropic and vasoactive support in pediatric intensive care. In infants and children, the pituitary gland plays a crucial role as a regulator of growth, metabolism, maturation and, possibly, immune function. We evaluated the effect of dopamine infusions (5 μ g/kg/min iv) on the dynamics of prolactin, growth hormone, and thyrotropin secretion and on the thyroid axis in critically ill infants and children.

 ${\it Design:}$ Prospective, randomized, controlled, open-labeled, clinical study.

Setting: Intensive care unit of a university hospital over a 9-month period.

Patients and Methods: The study population consisted of infants and children recovering from cardiovascular surgery. The group was stratified into two age groups (infants aged 12 to 90 days [n=18] and children aged 0.3 to 6.7 yrs [n=15]) and was studied dynamically (blood sampling every 20 mins for 3 hrs) on two consecutive days, after randomization for dopamine withdrawal on the first or the second day. Serum prolactin, growth hormone, insulin-like growth factor-1, thyro-

tropin, thyroxine (T₄), triiodothyronine (T₃), and reverse triiodothyronine (reverse T₃) concentrations were measured.

Measurements and Main Results: In the newborns, dopamine was found to suppress prolactin, growth hormone, and thyrotropin secretion consistently, rebound releases starting within 20 mins after dopamine withdrawal. One day later, prolactin concentrations were ten times higher, pulsatile growth hormone secretion was augmented, thyrotropin was unchanged, but T₃ was increased by 30% and the T₃/reverse T₃ ratio was inverted.

In the children, dopamine suppressed prolactin and thyrotropin (but not growth hormone) secretion, rebound releases starting within 20 mins after dopamine withdrawal. One day later, prolactin concentrations were at least twice as high, thyrotropin was increased ten-fold, T_4 was augmented by 14%, T_3 by 30% and the T_3 /reverse T_3 ratio had doubled. Neither in newborns nor in children did dopamine withdrawal appear to affect the low serum insulin-like growth factor-1 concentrations.

Conclusions: The data indicate that dopamine infusion induces or aggravates partial hypopituitarism and the euthyroid sick syndrome in critically ill infants and children.