



This Week in the Journal

JANUARY 8, 2004

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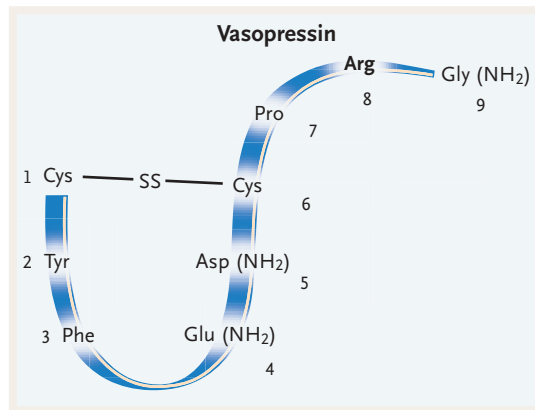
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ORIGINAL ARTICLE

Vasopressin for Out-of-Hospital Cardiopulmonary Resuscitation



Epinephrine is recommended for use in the resuscitation of patients with cardiac arrest. In this clinical trial, vasopressin, as compared with epinephrine, improved the rates of survival to hospital admission and discharge, but only among patients with asystolic cardiac arrest. There was no advantage to vasopressin therapy in patients with ventricular fibrillation or pulseless electrical activity.

If confirmed, these findings will further refine approaches to vasopressor therapy in patients with cardiac arrest and will lead to changes in the recommendations for cardiopulmonary resuscitation.

SEE P. 105; EDITORIAL, P. 179

ORIGINAL ARTICLE

Low-Dose Aspirin in Polycythemia Vera

Venous and arterial thromboses are major complications of polycythemia vera, but there is lingering uncertainty about the best means of thromboprophylaxis. This randomized trial showed that low-dose aspirin (100 mg daily) reduced the risk of thrombotic events, as compared with a placebo, without causing excessive bleeding.

Despite its limitations, this trial should evoke a reconsideration of the use of low-dose aspirin for thromboprophylaxis in patients with polycythemia vera who have no contraindications to aspirin therapy.

SEE P. 114; PERSPECTIVE, P. 99

ORIGINAL ARTICLE

Interferon Gamma-1b for Idiopathic Pulmonary Fibrosis

In 1999, we published the results of a preliminary study of long-term treatment with interferon gamma-1b and low-dose prednisolone in patients with idiopathic pulmonary fibrosis. These investigators report the results of a follow-on study of 330 patients with corticosteroid-unresponsive idiopathic pulmonary fibrosis who were randomly assigned to receive subcutaneous interferon gamma-1b or placebo. After a median of more than one year, interferon gamma-1b therapy did not improve progression-free survival, measures of lung function, gas exchange, or the quality of life.

Although a survival benefit of interferon gamma-1b therapy cannot be ruled out, the early promise of this treatment for idiopathic pulmonary fibrosis has not been fulfilled.

SEE P. 125; EDITORIAL, P. 181

ORIGINAL ARTICLE

Autoantibodies against the Folate Receptor and Neural-Tube Defects

The mechanism by which periconceptual folate supplementation reduces the incidence of neural-tube defects in the fetuses of women without clinical folate deficiency is unknown. After developing an assay for autoantibodies against the folate receptor, the authors found these autoantibodies in 9 of 12 women with a current or previous pregnancy complicated by a neural-tube defect, as compared with 2 of 20 women with an uncomplicated pregnancy. The autoantibodies blocked the binding of [³H]folic acid to folate receptors in vitro.

These data suggest that autoantibodies against the folate receptor might mediate at least some cases of neural-tube defect, although more data are needed before a cause-and-effect relation can be assumed.

SEE P. 134; PERSPECTIVE, P. 101

SPECIAL ARTICLE

Use of Operative Procedures in For-Profit and Not-for-Profit Health Plans

There is concern that in order to reduce expenditures, for-profit health plans may restrict patients' access to care, especially expensive operative procedures. This study of Medicare beneficiaries found the opposite.

Among beneficiaries enrolled in for-profit plans, the rates of use of some costly operative procedures were higher than among beneficiaries in not-for-profit plans; the rates of use of other procedures were similar.

The findings in this study were unexpected but have important implications for our understanding of the effects of financial incentives in health care plans.

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MECHANISMS OF DISEASE

Polycystic Kidney Disease

Polycystic kidney diseases are inherited renal disorders due mainly to mutations in genes that regulate the development and function of cells that line renal tubules. This review outlines the clinical importance of polycystic kidney diseases and discusses the cell biology and molecular mechanisms that cause the formation of hundreds of cystic lesions in the renal parenchyma.

SEE P. 151



CLINICAL IMPLICATIONS OF BASIC RESEARCH

Familial Cylindromatosis and the TNF- α Pathway

A recent study shows that the gene that is mutated in familial cylindromatosis represses tumor necrosis factor α (TNF- α) signaling — a finding that may have implications for preventing the disease.

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