

increasing level of tobacco smoke exposure. In addition, systolic and diastolic blood pressure and BMI were independent predictors of the aortic elasticity indices. In multivariable models, cotinine level ( $P = .020$ ) and systolic blood pressure ( $P < .001$ ) were inversely associated with AC and directly related to SI (cotinine level,  $P = .005$ ; systolic blood pressure,  $P = .0003$ ).

**CONCLUSIONS:** These data suggest that passive smoking is associated with decreased aortic elasticity in children, indicating early arterial changes.

## RHEUMATIC FEVER IN THE NEW MILLENNIUM

Submitted by Alyaa Kotby

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**INTRODUCTION:** During the past 2 decades the presentation of rheumatic fever (RF) has changed markedly from that of an acute florid illness to a more subtle subacute form that is frequently missed.

**OBJECTIVE:** With this work we attempted to describe the changing face of RF with its different patterns and time of presentation, particularly subclinical carditis.

**METHODS:** This work included 1732 patients with RF followed up in the pediatric department and pediatric cardiology clinic at Pediatric Hospital, Ain Shams University. Every patient was subjected to a thorough clinical examination, measurement of erythrocyte sedimentation rate and antistreptolysin O titer and C-reactive protein levels, a chest radiograph, electrocardiography, and echocardiography Doppler. Echocardiography was performed at the time of admission and repeated after 2, 4, and 6 weeks and 1 year after the attack. Diagnosis of RF was based on the revised Jones criteria.

**RESULTS:** Age at the first attack was  $<5$  years for 10% of the patients, 5 to 10 years for 51%, 11 to 15 for 36%, and  $>15$  for 3%; the male/female ratio was 1:1.34. Major clinical RF manifestations were carditis (60%), polyarthritis (56%), chorea (15%), erythema marginatum (0.12%), and subcutaneous nodules (0.12%). Seventy-two percent had carditis, after we combined clinical and echocardiographic criteria of cardiac affection 6 weeks after the attack. Pure arthritis was present in 41% of the patients, arthritis and carditis in 29%, and arthritis and subclinical carditis in 30%. One year after the initial attack the number of patients with echocardiographic features of valve affection remained the same. Pure chorea was present in 55% of the patients, chorea and carditis in 30%, and chorea and subclinical carditis in 25%. One year after the initial attack, 70% of the patients with chorea had echocardiographic features of valve affection. Chronicity of chorea is common with multiple relapses.

**CONCLUSIONS:** RF is not uncommon in children  $<5$  years of age. Subclinical carditis should be anticipated and looked for at the right time in susceptible patients, particularly those with rheumatic arthritis and chorea. Multi-center studies should be carried out for the addition of the echocardiographic features of carditis to Jones' minor criteria for the diagnosis of RF. Diagnosis of carditis requires a high index of suspicion in at least 1 of 3 cases.

## EXPERIMENTAL RESEARCH OF SIMVASTATIN IN REVERSING PULMONARY VASCULAR REMODELING IN VIVO AND IN VITRO

Submitted by Hanmin Liu

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**INTRODUCTION:** Simvastatin was predicted to be a potential inhibitor to pulmonary vascular remodeling. This novel reversion induced by simvastatin has remained an uncertain mechanism.

**OBJECTIVE:** Our goal was to explore the role of simvastatin as a potential inhibitor of pulmonary vascular remodeling.

**METHODS:** We established a neointimal pulmonary hypertensive rat model receiving monocrotaline after pneumonectomy. Simvastatin was administered after the operation. Hemodynamic and vascular remodeling corresponding indices were detected. *GATA-6*, a gene transcription factor, was evaluated in vivo. Proliferation and the cellular cycle were assessed in cultured vascular smooth muscle cells (VSMCs).  $\alpha$ -SM-actin, F-actin, and paxillin were detected to evaluate the phenotype changes.

**RESULTS:** Neointimal changes developed in 88.5% of right lung intraacinar arteries after pneumonectomy and monocrotaline administration. Mean pulmonary artery pressure, the right ventricle/(left ventricle + S) ratio, and media wall thickness significantly increased in rats that had pneumonectomy and were treated with monocrotaline but decreased significantly in simvastatin-treated rats. The expression of *GATA-6* markedly decreased in these rats and was significantly upregulated after receiving simvastatin. In vitro, the proliferation was significantly downregulated in VSMCs with simvastatin compared to that with platelet-derived growth factor.  $\alpha$ -SM-actin increased significantly, and F-actin or paxillin was downregulated in simvastatin-treated rats.

**CONCLUSIONS:** Our data indicate that simvastatin is most likely a pulmonary vascular remodeling inhibitor, which may reverse the proliferation of VSMCs and phenotype changes. Simvastatin can also upregulate *GATA-6* expression in lung tissue.

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