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ASSOCIATION for EUROPEAN CARDIOVASCULAR PATHOLOGY

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final programme

UNIVERSITY OF PADUA

A.N.M.C.O.

E.S.C.-WG17

FACULTY OF MEDICINE, UNIVERSITY OF PADUA

G.S.I.P.C.

S.I.A.P.E.C.-I.A.P.

S.I.C.

Congress Venue

Abbazia di Praglia
Via Abbazia, 7 - Bresseo di Teolo,
Padua, Italy

Ministero della Salute

E.C.M.

For Italian Participants Only
The Meeting has been registered for Continuing Medical Education of the Italian Ministry of Health and obtained **16 credit hours**. Number of participants is limited to 50.

P03 **Parental cigarette smoke as a risk factor for early coronary artery disease and sudden unexpected perinatal and infant death.**

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Cigarette smoke is the most important risk factor for fetal and infant sudden death (SIDS). The risk of stillbirth or death during the infant's first year of life is directly related to the amount the mother smoked. There is also an association between smoke and other pregnancy problems, such as low birth weight and miscarriage. The pathogenic mechanism of smoke is referable to different factors. The combustion products of nicotine, in addition to their diffuse toxicity, are heterogeneous and cause specific lesions of the autonomic nervous system. Gaseous combustion products, such as carbon oxide, lead to atherosclerotic plaques in the cardiovascular district and in the sino-atrial and atrio-ventricular arteries. Consequently, these combustion products cause an oxygenation deficit of the common myocardium, as well as developmental abnormalities of the conducting tissue, laying the morphological substrate for arrhythmias. We recorded a high incidence of preatherosclerotic lesions in the coronaries of fetuses with smoker mothers. If the maternal smoking habit persists, juvenile plaques can be observed in the infant. These early atherosclerotic lesions can be attributed to a direct action of the combustion products of nicotine on the smooth muscle cells of the tunica media of the arterial walls and/or on the neurons, interfering with homeostasis and cell differentiation, as well as to an indirect action of hypoxemia induced by arterio- and arteriolosclerosis. Our study population included 22 stillborns and 49 infants dying suddenly and unexpectedly. All cases died *sine causa* between the 32nd week of gestation and one year of age. Samples of the myocardium and the major coronary arteries were stained with Hematoxylin-eosin and Azan. The cardiac conduction system was removed in two blocks: the first included the sino-atrial node and the *crista terminalis*, the second contained the atrio-ventricular node, His bundle down to the bifurcation and bundle branches. These two blocks were cut serially at intervals of 40-mm (levels) and stained alternately with Hematoxylin-eosin and Azan. In 55% of fetuses and in 67% of the infants, multifocal coronary early atherosclerotic lesions of varying entity were detected. The alterations ranged from focal plaques with mild myointimal thickening to juvenile soft plaques in infants reducing the arterial lumen. In 45 % of stillborns and in 75% of infants with coronary lesions the parents were smokers. A significant correlation was observed between early atherosclerotic lesions and the risk factor considered. The reduction in the coronary lumen can be such as to cause alterations in cardiac blood supply. The harmful effects of cigarette smoking are not confined to the coronaries but also affect the small and medium-caliber arteries, -including the sino-atrial and atrio-ventricular arteries. Analysis of our series suggests that parental cigarette smoking has the highest significance among the risk factors considered in the pathogenesis of sudden fetal and infant death, while the newborn's position in the crib, which has been assigned a fundamental importance in recent years, is not equally supported by anatomo-pathologic data.