

## HIGH BLOOD PRESSURE, SMOKING AND CARDIOVASCULAR RISK

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### Introduction

Smoking and high blood pressure have been identified as two major cardiovascular risk factors, accounting for a great proportion of total and cardiovascular mortality worldwide. Indeed, according to the latest estimations of the World Health Organization, more than 5 million deaths are attributable to the smoking habit and no less than 7 millions to high blood pressure (1). The prevalence of smoking habit is estimated around 30% of adult population all over the world (47% in men and 12% in women) (2) and high blood pressure ( $\geq 140/90$  mmHg) is found in no less than 30% of adult population in most countries, either developed or developing (3). The coexistence of both risk factors in individuals at community level is approximately 5%.

For decades it has been postulated that smokers shown lower blood pressure levels than non-smokers. Nevertheless, in recent years several epidemiological and clinical studies have demonstrated the opposite, showing significant differences in 24-h blood pressure monitoring (Figure 1) (4). Trends in prevalence of smoking and high blood pressure are relatively stable, with a slight increase related to the ageing process of the population in the case of high blood pressure and the little effect of the smoking prevention and control programmes, particularly on the youth.

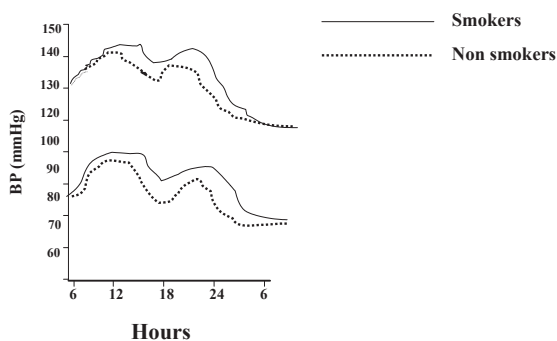


Figure 1. 24-hours blood pressure monitoring curves in smokers and non-smokers

### How smoking and high BP act in the cardiovascular risk

Both risk factors increase the final cardiovascular risk when acting independently, but their interaction in the same individual contributes to rise that risk more than 4 fold (Figure 2) (5). This is mainly due to the interactions of smoking with blood pressure and other cardiovascular risk factors, as is shown in Table 1 (6).

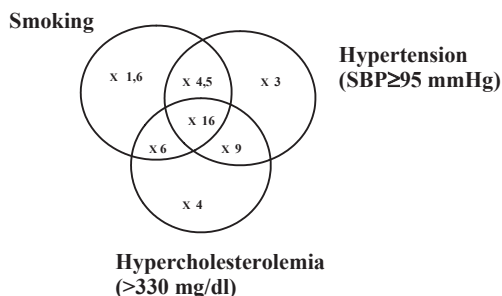


Figure 2. CHD risk according to the combination of smoking, arterial hypertension and hypercholesterolemia

### Table 1. Interactions between smoking and other cardiovascular risk factors

1. BLOOD PRESSURE
  - a. Rises with smoking
  - b. As a rule, smokers have lower BP figures
  - c. Hypertensive smokers:
    - . Are harder to achieve optimal BP control
    - . Have a worse prognosis
    - . Are more likely to have atherosclerotic renovascular hypertension
    - . Are more likely to develop malignant hypertension
2. SERUM LIPIDS
  - a. Increased
    - . Total Cholesterol
    - . LDL-Cholesterol
    - . Free fatty acids
    - . Triglycerides
  - b. Decreased
    - . HDL-Cholesterol
3. OBESITY
 

As a rule, smokers have lower body weight
4. HEMATORRHEOLOGY
  - a. Increased
    - . Fibrinogen
    - . Blood viscosity
    - . Leukocyte count
    - . Hematocrit
    - . Platelet aggregation
  - b. Decreased
    - . Platelet survival
    - . Bleeding time
    - . Erythrocyte distensibility
5. ORAL CONTRACEPTIVES
 

Substantial increase of risk of MI, stroke and thromboembolic events
6. HORMONAL CHANGES
 

Increased plasma estradiol (men) and vasopresin and worsen glucose tolerance

High blood pressure decisively increases the risk of cerebrovascular disease, coronary heart disease and renal failure, while smoking dramatically increases the risk of coronary heart disease, peripheral vascular disease and, in a less extent, cerebrovascular disease. The underlying mechanism in both cases is mainly the acceleration of atherosclerosis jointly with vascular and endothelial dysfunction. Nicotine, carbon monoxide, oxidants and cadmium are the main tobacco or tobacco smoke compounds that intervene in the physiopathological pathways leading to the organ damage. Some of them are connected with the physiological mechanisms of blood pressure regulation, which could explain the influence of smoking in blood pressure raising (Table 2) (6, 7)

In view of those evidences, smoking cessation is an essential component of the comprehensive management of hypertensive patients. Unfortunately, several studies have shown the limited involvement of physicians in antismoking initiatives, even in patients with smoking-related chronic conditions (8), and the maigre results of clinical interventions in terms of long-term smoking cessation in hypertensive population (9). Therefore, more intensive efforts are needed to achieve better implication of physicians and other health professionals in smoking cessation at clinical level and in smoking prevention and control at community level (10).

**Table 2. Cardiovascular effects of smoking**

1. CARDIAC EFFECTS
  - a. Coronary arteries
    - Atherosclerosis in native circulation
      - . MI (fatal and non-fatal)
      - . Recurrent infarctions
      - . Silent ischemia
    - Spasm
    - Restenosis after angioplasty
    - Atherosclerosis in bypass grafts
  - b. Sudden death
  - c. Unstable angina
  - d. Cardiomyopathy
  - e. Myocardial arteriolar hyperplasia
2. CEREBRAL EFFECTS
  - a. Atheroembolic brain infarcts
  - b. Subarachnoid hemorrhage
  - c. TIA
  - d. Recurrent carotid artery stenosis after endarterectomy
3. OTHER ARTERIAL PATHOLOGY
  - a. Aortic atherosclerosis
  - b. Iliofemoral atherosclerosis
  - c. Intermittent claudication
  - d. Lower limb ischemia and amputations
  - e. Recurrent atherosclerosis of the bypass grafts
  - f. Abdominal aortic aneurysm
  - g. Renal arteriolar hyperplasia
  - h. Failure of skin grafts
  - i. Uteroplacental arterial hyperplasia
  - j. Diabetic microangiopathy

**Smoking cessation strategies in hypertensive patients**

Smoking cessation strategies are currently very clearly defined after many years of worldwide experience. The available tools, either non-pharmacological or pharmacological, are deeply analysed in several qualified papers recently published (11-13). They include counseling/minimal intervention, nicotine replacement, and bupropion as first-line therapies; additionally, one can use specialized psychological intervention and other pharmacological agents (clonidine, antidepressants, etc.). These treatments should be implemented according to a stepped-based approach, like the one recommended many years ago for the treatment of hypertension (Table 3), with the aim of facilitating the involvement of physicians in smoking cessation in clinical settings.

Non-pharmacological strategies, namely counseling and minimal intervention (ask, advise, assess, assist, arrange) account for less than 5 minutes, so they can be regularly put into practice in every consultation of smoker patients. They are effective in approximately 15% on the average (14). Concerning pharmacological treatment, the

most widely used agents are nicotine replacement products (chewing-gum, patch, nasal spray, inhaler and tablet) and bupropion. Depending on the context and on the intensity of the intervention, their effectiveness is estimated around 20-25% (14). As a rule, the use of nicotine replacement in hypertensive patients does not need special caution, except in cases of severe and resistant hypertension and after a coronary event. In these cases nicotine replacement agents are contraindicated but, in general, they can be used following the same recommendations as for smokers as a whole. The only cautionary remark is avoiding their combination, which could supply a high dose of nicotine and, therefore, produce pressor effects (i.e. nicotine chewing-gum plus nicotine patch) (15-18).

Two final considerations are related to the harm reduction strategy and the frequent relapse. Concerning the later, during the following 12 months after the quit attempt around 70% of abstinentes totally or partially relapse. This is similar to the situation in hypertension control (more than 60-70% of hypertensives under treatment remain with their blood pressure figures uncontrolled). Physicians have to be aware that the definitive abstinence from smoking very often comes only after several unsuccessful attempts. Consequently, they must insist over and over again in their efforts, helping their smoker patients to give up smoking (19). In relation to the harm reduction strategy, it has been postulated in recent years with the aim of facilitating the integration of smoking cessation interventions in daily clinical practice, assuming that the reduction of risk is an optional objective when the complete abstinence is very difficult or even impossible (20). Needless to say that the full abstinence, like the full hypertension control, remains as the main goal of the physicians' intervention.

**Table 3. Algorithm for treating smoker-patients (stepped-based approach)**

STEP I	All smokers	Counseling/Minimal intervention
STEP II	Smokers who need pharmacological treatment	NRT or Bupropion
STEP III	Smokers who need more intensive pharmacological treatment	NRT + Bupropion or NRT combinations (e.g. tablet + patch)
STEP IV	Smokers who need other therapeutic approaches (comorbidity, intolerance, resistance)	Other pharmacological treatments (e.g. clonidine, antidepressants)  Specialized psychological treatment

NRT=Nicotine Replacement Therapy

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