

Volume 5 Issue 1

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Coronary artery disease (CAD) are closely linked to hypertension. They constitute the main cause of disability, morbidity and mortality in hypertensive patients. On the other hand epidemiologic studies revealed that hypertension is a major risk factor for CAD. Furthermore, CAD are predicated to be the main techniques, drugs and therapeutic interventions that are introduced in the management of coronary patients. For these reasons, the EHS organized a working group on CAD with the objective of writing a practical manual targeted to the internists and cardiologists and contains the Guidelines.

The book is expected to be available before the end of this year, it consists of four sections covering the diagnosis and management of the different coronary syndromes, including values and limitations of the commonly used diagnostic procedures, drugs and interventions in the treatment of coronary patients, special resuscitation in his everyday handling of the coronary patients, stress has been given to practical questions such as clinical evaluation of chest pain, how to predict the need for hospitalization, the role of ECG-Stress testing, the values and limitation of coronary angiography and other imaging techniques, management of acute coronary syndromes with a detailed chapter on acute myocardial infarction.

This is the second book produce by the EHS, the first book was published two years ago consisting of a brief review and guidelines for the management of hypertension in Egypt, The success of this first book, which became an important source of information for many of the practicing physicians was an important factor behind the idea of writing a new practical manual about CAD these two books fit within the educational goals of the EHS and are available at no cost to all Egyptian physicians. The production and printing of both books was through the generous support of the Egyptian drug industry, namely, Hoechst Marion Roussel And Merck Sharp & Dhome-Egypt.

These two books were the result of collaboration of a number of Egyptian experts from different Universities and will help the Egyptian practitioner to catch with the rapid developments in the fields of hypertension and coronary Artery Disease.

M.Mohsen Ibrahim, MD
Prof. & Chairman Department of Cardiovascular Medicine -Cairo University
president of the Egyptian Hypertension ,Society

THE PRESIDENTS MESSAGE

THE PRESIDENTS MESSAGE CORONARY ARTERY DISEASE EHS GUIDELINES

Coronary artery disease (CAD) are closely linked to hypertension.They constitute the main cause of disability,morbidity and mortality in hypertensive patients. On the other hand epidemiologic studies revealed that hypertension is a major risk factor for CAD .Furthermore,CAD are predicated to be the main techniques,drugs and therapeutic interventions that are introduced in the management of coronary patients. for these reasons, the EHS organized a working group on CAD with the objective of writing a practical manual.targeted to the internists and cardiologists and contains the Guidelines.

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Editorial

HYPERTENSION IN THE ELDERLY
OMAR AWAAD

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Ain shams University

There are two patterns of hypertension in the elderly combined systolic and diastolic hypertension and isolated systolic hypertension (ISH)> 65y their prevalence change with age as evidenced in framingham study ,where the types of HPT between age 70-79 were (20% ISH & 50% borderline ISH). However this should not be admixed with pseudohypertension [White coat effect or artifactually elevated with modest lowering utilities .in this situation measurements is better assessed by finger recording.

The retrieved data ,from the so many clinical trials conducted on elderly hypertensives[AUSTRALIAN.EWPHE,WARRENDER,SHEP,STOP-HT,MRC.STONE,..etc.]cleared that risks of morbidity&morbidity is high specially in those without treatment for 2 years also mortality from CHD & stroke is much higher than in normal population.in such a category the systolic pressure [SBP] is more predictor of cardiovascular[CV] risk even more in ISH where there is 1% increase in the rate of mortality with each 1mm increase in SBP in the very old>85y; lowest mortality was achieved at BP range of [140- 169/70-99].Moreover,in general,higher BP predisposes to dementia (Alzheimer) the basic pathophysiological mechanism of hypertension in the elderly is the loss of distensibility & elasticity in large arteries with an increase in pulse wave velocity & an early return of plus wave reflection in systoli with or without a decrease in diastolic pressure[DBP]. the cardiac output,intravascular filling, renal blood flow & PRA Decrease while PVR, LV wall thickness and mass increase .if an abrupt increase in SBP & DBP occurs , one should suspect atherosclerosis &/or renovascular disease.

It is worth noting that postural hypotension [A fall in SP of 20mm Hg after 1 min quite standing] is common in elderly hypertensive. in SHEP the incidence was 10 % at 1 min & 12% At 5 min 17% at either. the prevalence is higher if rising from a supine position ,I.e the higher the basal SBP the greater the postural fall observed the most common mechanism, for this is ;venous pooling in the legs,autonomic insufficiency,reduction in baroreceptor sensitivity, splanchnic blood of after eating &/or shifts in the threshold of cerebral autoregulation.

Therapeutic intervention to control hypertension in the elderly proved of value in 13 randomized controlled trials and 6 large high quality trials . the conclusion from such trials is that; the morbidity & mortality of the treated group where significantly better I.e only 18 elderly hypertensive and 15 ISH patients needed treatment for 5y to prevent CV events ,A number of explanation for this greater benefit has been proposed namely ;the elderly start with much higher risk and smoke less [better response to antihypertensive].Also being more recent ,the drugs assessed in most of such trials were those of greater cardioprotection. however treatment with low-dose diuretics or B-blockers was still claimed by some to be of benefit whether in diastolic or ISh .However ,the disclaimers to the general agreement of such trials argue that they may not accurately reflect what can be accomplished in clinical practice and that benefits may be lessened in the very old (over age 80) and had been only shown in those using diuretics or calcium channel blockers [CCBs],but not B-blockers.

Yet at this juncture, one has to admit that only a small minority of elderly hypertensive are being treated, though treatment of this category seems the judicious decision as; millions of people over the age 65 have hypertension [predominantly or purely systolic], their risk are significant, and the benefits of their treatment have been documented so therapy of such patients should be gentle & gradual due to their increased risk and be more cautious in the very elderly >80y for fear of increased mortality.

The need for lifestyle modification is one of the therapeutic objective, as enough data is available to document its efficacy, so non drug therapy should be applied before or instead of drug therapy, dietary Na should be moderately restricted (100-120 mmol/day) though its reduction is sometimes difficult in the elderly.

Among drug treatment recommendations that have been stated is; consider treatment for patients up to age 80 with SBP >160/90 mm Hg; the choice of therapy should be individualized according to the presence of concomitant conditions, first line therapy should be low dose of diuretics, CCB and ACE Inhibitors can be good alternatives and B-blockers are not the first choice except in and ischaemics, other recommendations suggest that; long action CCBs are an appropriate alternative to diuretics and that home BP recording may be particularly useful.

The golden therapeutic rule is; start with a lower dose better with (once-daily) smoothly working preparation and watch out for drug interactions.

It is worth noting that the factors that might contribute to the increased risk of pharmacological treatment in the elderly are; the decrease in baroreceptor activity [orthostatic hypotension], the impairment in cerebral autoregulation [cerebral ischaemia with small falls in BP], the decrease in intravascular volume [volume depletion, hyponatraemia,], muscular weakness], the decrease in existence of polypharmacy [drug accumulation], the existence of polypharmacy [drug interaction], and the CNS changes [depression & confusion] and the CNS changes [depression & confusion].

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ABSTRACTS OF WORLD LITERATURE HYPERTENSION AND ITS TREATMENT IN THE NINDS rt-PA STROKE TRIAL

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Background : We examined the frequency, course,and treatment of hypertension in the NINDS rt-PA stroke trial.

Method: blood pressure (BP) was measured at the time of admission ,at randomization ,and then 36 times during the first 24 hours after randomization .patients with a systolic BP of >185 mm Hg and a defined as hypertensive before randomization, and those with a systolic BP of >105 mm Hg or a diastolic BP of > 105 mm Hg within the first 24 hours after randomization were defined as hypertensive after randomization. standardized clinical assessments were conducted at 24 hours and at 3 months .post association of antihypertensive therapy with clinical outcomes
Result:of the 424 patients,121(19%) had hypertension on admission and 372 (60%) had hypertension in the 24 hours after randomization .the use of antihypertensive therapy before randomization (tPA 9%,placebo 29%) was similar between placebo and tPA-treated patients NO. adverse Effects of pre randomization antihypertensive therapy on 3-month favorable outcome were detected for either the placebo or tPA-treated Groups. for either the placebo or tPA-treated groups .for placebo patients with hypertension in the 24 hours after randomization ,clinical outcome measures were similar for those patients who did and did not receive antihypertensive therapy after randomization ($p>0.26$); antihypertensive therapy was not associated with declines in BP ($p=0.44$) or with abrupt declines ($P=0.14$), those tPA patients who were hypertensive after randomization and received antihypertensive therapy were less likely to have a favorable outcome at 3 months ($P<0.01$) than those who were hypertensive and did not receive antihypertensive therapy
Conclusion :the frequency of hypertension and the use of antihypertensive therapy were similar between the tPA and placebo group in the NINDS rt-PA stroke Trial .in the placebo group, antihypertensive therapy was not associated with less favorable outcomes at 3 months; postrandomization antihypertensive therapy was associated with less favorable outcomes for the tPA patients who were hypertensive, however ,because of the nonrandomized post hoc comparisons leading to type 1 errors,the significance of this observation is unclear .careful attention to BP and gentle management remain warranted for stroke patients treated with tPA..

stroke 1998;29:1504-1509.

ARTERIAL HYPERTENSION: THE NATIONAL PROGRAM OF ITS PREVENTION AND TREATMENT IN UKRAINE

Gorbos I, Smyrnova I,Svisclienko E,Sirenko Y
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Arterial hypertension (AH) is one of the commonest disease in Ukraine. About 5 million persons with arterial hypertension were registered in 1997 Annually ,In the course of people's primary visits to medical-prophylactic institutions almost 430000 patients with arterial hypertension were identified .According to the data of the epidemiological investigation ,there are nearly 13 million people with arterial hypertension in the country. and in half of them a border-line arterial hypertension is established.62.0% of all patients are aware of arterial hypertension presence, 23% receive treatment with an effective outcome in only 12.8% of them.

An unfavorable epidemiological situation with regard arterial hypertension and its complications that has been established in Ukraine can be Drastically improved provided that high arterial blood pressure has properly controlled, however, the population is inadequately instructed about arterial hypertension and possibilities for prevention of its complications, not all arterial hypertension patients are identified as yet. the mercury sphygmomanometer is lacking the arterial hypertension diagnosis and treatment leave much to be desired in Ukraine the production of modern antihypertensive drug, accessible and affordable, is still at low level and does not meet needs of the patients. there do not exist mechanism encouraging people to maintain and strengthen their health. there is no state policy aiming to formulate healthy life style. thus, arterial hypertension constitutes a national social challenge that requires state support and CO-ordinated efforts of different ministries and departments, in this connection the national and program for arterial hypertension prevention and treatment in Ukraine has been worked out .the program is focusing to reduce morbidity associated with arterial hypertension coronary heart disease and cerebrovascular diseases. presented in 26Th. Annual meeting of the Egyptian Society of Cardiology , Cairo, Egypt. February 1999.

SODIUM REDUCTION & WEIGHT LOSS IN THE TREATMENT OF HYPERTENSION IN OLDER PERSONS.
RANDOMIZED CONTROL TRIAL OF Nonpharmacologic INTERVENTIONS IN THE ELDERLY [TONE]
WELTON PK, APPEL LJ, ESPELAND MD, APPLGATE WB , ETTINGER WH, KOSTIS JB , KUMANYKA S, LACY CR, JOHNSON KC, FOLMAR S CULTER JA..
TONE Collaborative Research Group, New Orleans, USA

Context : Nonpharmacologic intervention are frequently recommended for treatment of hypertension in the elderly ,but there is a paucity of evidence from randomized controlled trials in support of this recommendation.

objective : to determine whether weight loss or reduced sodium intake is effective in the treatment of older persons with hypertension.

Design : Randomized controlled trial.

Participants : A total of 875 men and women aged 60 to 80 years with systolic blood pressure lower than 145 mm Hg and diastolic blood pressure lower than 85 mm Hg while receiving treatment with a single antihypertensive medication.

Setting : four academic health centers .

Intervention : The 585 obese participants were randomized to reduce sodium intake, weight loss , both or usual care, and the 390 nonobese participant were randomized to reduce sodium intake or usual care withdrawal of antihypertensive medication was attempted after 3 months of intervention .

main Outcome Measure :Diagnosis of high blood pressure at 1 or more follow-up visits, or treatment with antihypertensive medication , or a cardiovascular event during follow-up (range, 15-46 months; median, 29 months).

Results :The combined outcome measure was less frequent among those assigned Vs not assigned to reduced sodium intake (relative hazard ratio.0.69;95% confidence interval [C1],0.59-0.81;P<0.001) and , in obese participants ,among those assigned Vs not assigned to weight loss (relative hazard ratio, 0.70; 95% C1,0.57-0.87;P<.001) .relative to usual care, hazard ratio among the obese participants were 0.60 (95% C1, 0.45-0.80;P<.001> for reduced sodium intake alone, 0.64 (95% C1 , 0.49-0.85;

P=.002) for weight loss alone , and 0.47 (95% CI , 0.35-0.64; P<.001) for Reduced sodium intake and weight loss combined . the frequency of cardiovascular events during follow-up was similar in each of the treatment group

Conclusion : Reduced sodium intake and weight loss constitute a feasible, effective and save Nonpharmacologic therapy of hypertension in older persons.

JAMA 1998,279;839-846.

ABSTRACT OF LOCAL LITERATURE ECHOCARDIOGRAPHY FINDINGS IN HYPERTENSIVE EGYPTIANS RESULTS FROM THE EGYPTIAN NATIONAL HYPERTENSION PROJECT (NHP)

Helmy SM,Gharib S,Sharaf Y ,Ibrahim MM Department of Cardiology,faculty of Medicine, Cairo University.

Background : inspite of the well recognized role of echocardiography in defining LV changes yet there is lack of survey studies utilizing this technique this resulted in the limited data available about the effect of elevated blood pressure on LV structure and function among large populations specially the untreated and uncomplicated case

Objectives : 1) to study the prevalence of different cardiac diseases as diagnosed by echocardiography in a nation wide survey study including hypertensive and normotensive population , 2) to study changes in LV structure and function in hypertensive Egyptians

Patient population : Among 2313 cases surveyed in phase 11 of the Egyptian NHP, 1559 were hypertensive (BP> 140/90 mmHg or receiving antihypertensive medications) and 754 were gendermatched normotensive Echocardiography was attempted on all individuals those with limited image quality (90%) were excluded , different cardiac disease (5.8%) pericardial disease (1.2%) regional wall motion abnormalities (5.9%) cardiomyopathies (1.2%) ,and other cardiac disease (1.8%). results : the prevalence of different cardiac disease was less in normotensives (N) compared to hypertensives (H) as follows: significant valvular heart Disease in 2.8% of N Vs 7.4% of H;pericardial disease in 0.6% of N Vs 1.6% of H; regional wall motion abnormalities in 3.4% of N Vs 7.2% of H; cardiomyopathy in 0.1% of N Vs 0.8% of H;and others cardiac disease in 0.4% of N Vs 2.5% of h the remaining population (n=1796,77.6%;751 N and 1145 H) was further studied for the prevalence of LV hypertrophy and changes in LV geometry. Of those 4.8% had LV hypertrophy ;1.2% of N and 6.9 of H Prevalence of normal LV (Nr), Concentric remodeling (CR) , concentric hypertrophy (CH) and Eccentric Hypertrophy (EH) was as follows; Total : 1796 [N : 651 & H:1145] -NR : 88.6 % [N:96.6% & H:84.1%]-CR : 6.5%[N : 2.1% & H: 9.0%-CH :3.6% [n : 0.19% & h: 5.1%] -EH :1.3% [N :0.3% & H : 1.8%].

Conclusion : results of echocardiographic survey study of hypertensive Egyptian revealed a higher prevalence of different cardiac involvement of compared to normotensives. the prevalence of cardiac involvement was (15.9) with LV Concentric hypertrophy is the least encountered presented at 3rd scientific meeting of the Egyptian hypertension society,port said ,Egypt ,December 1998.

IMPACT OF HYPERTENSIVE LVH ON QT DISPERSION : A COMPARISON BETWEEN ECHO EVIDENCE AND ELECTROCARDIOGRAPHIC EVIDENCE OF HYPERTROPHY

El Ashry MM, Attia H, Fahmy M.
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Objectives : This study sought to examine whether the QT dispersion (QTD) was affected by left ventricular hypertrophy (LVH) in Systemic Hypertension and if there is any difference in the degree of QT dispersion between patients with (LVH) detected only by the Echocardiogram (Echo) & those with LVH detected by both Electrocardiogram (ECG) and ECHO.

Background : This study sought to examine whether the QT dispersion (QTD) was affected by left ventricular hypertrophy (LVH) in systemic hypertension and if there is any difference in the degree of QT dispersion between patients with (LVH) detected only by the Echocardiogram (Echo) & those with LVH detected by both Electrocardiogram (ECG) and ECHO .

Background QT dispersion is a predictor of myocardial instability hypertensive subject QTD is due to anatomic modification induced by LVH .these anatomic changes have the same effect on arrhythmia substrate

Methods : Thirty consecutive newly diagnosed hypertensive subject with hypertension ,not on treatment were age and gender matched for LVH (n=16) versus no LVH as a control group (n=14). LVH patients were diagnosed by 12 lead surface ECG and /or by 2d Echo, divided into 2 groups : group 1 patients with LVH by Echo only (n=7) ,group 11 .patients with LVH by Echo & ECG (n=9) QTD was manually measured on 12 lead surface ECG .A computerized channel Holter system was used to study the average number of premature ventricular beats/minute

Results : QTD was significantly high in the whole study population (96.33 +21.48 msec.) compared to those of control group with no LVH (57.14 + 15.4 msec.) (p=0.001) . QTD was greater in patients of group 11 than those group 1 (p=0.004) the LVH evidenced by ECHO only septum (13.14 + 1.8) post wall (11.17 + 1.25) QTD (65.71 + 21.4) , while LVH evidenced by ECG & ECHO septum (16.96 + 2.5) post wall (15.14 + 2.06) QTD (91.11 + 11.6) .patients of group II had higher septal and posterior wall thickness than those of 1 .premature ventricular beats were more prevalent in group II with a mean rate of (10.66 +0.7) beat/minute (p=0.0008) VS (5.02 + 10.7) beat minute (p=0.1) in Group I the frequency of ventricular arrhythmia's was greater in patients with QTD (n=17 patient)

Conclusions : QTD is greater in Hypertensive Subjects particularly those with LVH evident by EGG and ECHO (with greater LV .wall thickness) tan that diagnosed by ECHO only significant QTD is associated with higher incidence of ventricular arrhythmia's hypertensive patients.

presented in the 26 Th. Annual Meeting of the Egyptian Society of Cardiology ,Cairo,Egypt, February 1999.

AMBULATORY BLOOD PRESSURE CHANGES DURING EPISODES OF SILENT ISCHAEMIA

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Background : Silence of ischaemia does not speak of mildness and silent ischaemia (SI) is expressed first as regional wall motion abnormalities (detected by ECHO) ,and then as ST segment depression (by ECG) before pain may be evoked . less well

known is the possible occurrence of altered autonomic function which classically accompanies pain episodes, namely, changes in blood pressure.

Methods : to address this issue ,we studied 22 pts with Ischaemic heart disease (8 females and 14 males ,

mean age $45 + 8.4y$) with the diagnosis of unstable angina pectoris in 15 pts and post infarction angina in 7 pts with the purpose of recording BP changes during Ischaemic episodes using 24 hour BP recording . Ambulatory recording was periodically carried out for BP and continuously for ECG ,BP was recorded every 15 minutes and a reading was taken at the time of Ischaemic episodes detected by ST depression .

Results : out of 22 patients studied , 18 (27 %) exhibited episodes of silent ischaemia , 13 (59%) did not show ischaemia at one time or another , while only 4 (18%) did not show ischaemia during recording. Systolic BP average $144 + 20.09$ mmHg , (range 120- , 180) during silent ischaemia. $140 + 16.31$ mmHg (rang 120-176) during manifest Ischaemia versus $125.8+22.1$ (range 120 - 184) with ischaemia free periods. diastolic BP ranged from 66 to 108 (mean $82+10.83$) mmHg during silent Ischaemic episodes, from 47 to 112 ($74.3+14.34$) mmHg during Ischaemia free episodes and from 54 to 107 (mean $81.61 +14.68$) mmHg during manifest Ischaemic .

Conclusion : Despite the limitations inherent to ambulatory BP recording ,our data suggest that myocardial ischaemia ,whether manifest or silent ,is associated with an increase in BP .Cause and effect relation between both are to be elucidated , but the fact remains that BP is increased in Silent ischaemia and adversely affected haemodynamics would aggravate Ischaemic and set a viscous circle.

Presented at the 3rd Scientific meeting of the Egyptian Hypertension Society, Port Said , Egypt, December 1998.

THERAPEUTIC AWARENESS NECESSITATES :

Knowing that ,the recently introduced ,long acting non dihydropyridine Ca entry blocker [CCBs],

Mibefradil : which blocks both the T [transient] & L [long] type Ca channels ,bears life threatening drug-drug interactions. The manufactures issued a warning letter to physicians describing the suppression of SA node activity, occurring special in the elderly whom are on concurrent B-blockers and emphasised that use of the drug with digoxin, verapamil , diltiazem requires great caution , Later on June 8th last year, the drug was temporary voluntary withdrawn by the manufacturers after the postmarketing surveillance had cleared that the drug inhibits cytochrome P-450 enzyme CYP 3A4 and interferes with the metabolism of at least 26 other medicines. The dihydropyridine CCBs were not included , yet since case reports unmasked the hazard of beginning with such group of CCBs in patients who are already on mibefradil an B-blockers ,This has called the issue of another warning supplement by June 12th this advises a delay after discontinuation of mibefradil [half life up to 25 hours] to allow a prolonged washout period of 7 days before considering beginning B-blockers & other CCBs This period is to be increased to 14 days in case of felopidine and timolol and is not required in case of ACE inhibitors ,AT1 antagonists & diuretics. Due to such interactions, if myocardial depression or hypotension issue high dose glucagon [5-10mg I.V. diluted safer with or saline] to increase cAMP ,so as to enhance a positive inotropic , dromotropic and chronotropic effects is recommended .Repeated doses of Ca, vasopressors and temporary pacing are often necessary.

JAMA 1998;280:157-8

DIAGNOSTIC UTILITY IMPLIES :

Considering the pulse pressure a predictor of C.V. morbidity & mortality, This was apparent in a french follow up study conducted on 19,083 men aged 40-69 subjected to routine examination .it was found that men whose pluse pressure were greater than 65 mmHg had a three fold increase in faftal coronary heart disease over than those who had pulse pressure 45 mmHg or less . this sinifites that pulse pressure is a good diagnostic predictor. Thus, it is assumed that wilde pluse pressure indicates indicates increased stiffness of larger arteries and calls for aggressive therapy, it is proposed that drugs that prevent or reverse sclerosis may be needed in addition to the rational antihypertensives used ,

Hypertension 1997 ;3 (12):1410-5

EHS News & Calendar

EHS NEWS :

The annual Ramadan social gathering was held last January at cairo sherton, New ideas to expand the service of the society to the different governorates was raised and will be organized by Prof ,Dr . Fathi Maklady ,Public awareness and education at their social gathering was also suggested.

The editorial committee of the News Letter of Egyptian Society of Hypertension, is due to change in the coming edition Prof. Dr.Mohsen Ibrahim has nominated Prof Dr. Hassan Khaled Prof.of Cardiology Alexandria University editor in chief and is whole heartily thanking Dr.Mohamed Hamed the former editor for his executive elegant and distinguished collaboration in making this News Letter come to reality and progress throughout previous years.

CALENDER:

World Hypertension League 18Th Council Conference and workshop on Hypertension in the Elderly	May 7,1999 Buenos Aires, Argentine.	Dr.Patrick J.Mulrow, Secretary General, World Hypertension League Medical College of Ohio ,Po Box 10008 Toledo,OH 43699-0008,USA e-mail : gmonhollen@mco.edu
Fourteenth Annual Scientific Meeting of the American Society of Hypertension	May 16-19,1999 New York USA	American Society of Hypertension 515 Madison Avenue ,Suite 1212 New York NY 10022,USA.
Symposium on "what is new in hypertension	13th May ,1999 Cairo Sheraton.	Contact : Mrs Amany Kandeel Tel (202)362 4803-Fax (202) 363 9895

A STEP AHEAD WITH PERINDOPRIL

A major challenge to meet when tempting to control a hypertensive patient is the emergence of prehypertrophic or the establishment of overt left ventricular hypertrophy [LVH] as a segregated or consecutive entity to the existing pressure constrain. Once exists, it becomes a notion of warning to the treating physician that the spectrum of hypertensive heart disease [HHD] is now on the roll and that one should be cognizant of its morphological and functional alterations so as to try and halt its progression by every possible mean.

Focusing on how things evolve clears that with pressure constrains the myocardial texture gets disturbed namely; the myocytes, the intramyocardial coronaries and the interstium. Thus myocytes hypertrophy and get encased by endomysial fibrosis, that will enhance their stiffness and increase their O₂ perfusion distance, to induce a state of localized hypoxia. This together with thickening of the intramyocardial coronaries [by medial hypertrophy & adventetial fibrosis] is abet to impair the coronary vasodilator reserve and create a state of microvascular angina. This aside the concomitant progression of perimysial fibrosis and microscars in the interstitium, coupled to the disturbed myocardial relaxation will all contribute to the diastolic dysfunction clinically characteristic to HHD.

If triggers recycling such initially adaptive profile are not curtailed by an appropriate therapeutic utility, a switch to the maladaptive pole of the continuum sets in. Thus, the increase microtubular hyperpolarization within hypertrophied myocytes, will alter their viscoelastic properties and increase the load within them to impede their shortening. Also the microischaemic environment created by hypertrophy and fibrosis, will trigger myocyte apoptosis; a scenario that ends up by overt heart failure with or without ischaemic episodes.

This molecular understanding of HHD is abet to highlight the importance of having an antihypertensive like perindopril, that has much More to offer, than just a 24 hours pressure control, specially when the point of concern is its ability to improve the coronary reserve.

This has been justified in many studies, one of which has assessed the one-year utility of perindopril in hypertensive with microvascular angina pectoris. Results revealed that maximal coronary blood flow was increased by 54% and minimal coronary vascular resistance was significantly decreased [when quantified using Argon method at basal condition and after microvascular vasodilatation with pyridamole]. The calculated coronary reserve increased then by 67% while the left ventricular mass decreased by 11%, signifying that this improvement is more than expected for regression of LVH alone. This preferential improvement in coronary microcirculation validates the utility of perindopril in cutting some of the triggers that switch HHD to the maladaptive end of the continuum.

Moreover, perindopril like other ACE inhibitors was shown to regress interstitial fibrosis in endomyocardial biopsies from hypertensive patients with normal coronary angiogram but with microvascular angina. This seems likely to be linked to the ability of such group to increase bradykinin that will trigger the release of arachidonic acid metabolites; prostacyclin being the one specified in particular to suppress collagen gene expression experimentally, in cardiac fibroblasts.

Thus, a dug with potentialities, that can interplay on collagen metabolism to reverse

fibrosis and that can improve the coronary reserve and the existing diastolic dysfunction, will offer optimum cytoprotection, that enables it to step ahead to encompass HHD before it might deteriorate to its morbid end points.

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4. Hypertension 1998; 32: 84.

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Pharmacologic therapy proved effective in improving morbidity and mortality in hypertensive patients. Since there is no cure from established essential hypertension, it is obvious that once drug therapy is started, it has to be continued for many years and most probably indefinitely. Costs, side effects and accordingly patient's compliance are important limitations when considering life-long drug treatment. For these reasons initiation of drug therapy is not an easy decision, should not be taken lightly and should be based on strong grounds. From the very beginning it is mandatory to establish the presence of hypertension by repeated and accurate blood pressure (BP) measurements over variable intervals depending upon BP level. Non-pharmacologic treatment should be started in all hypertensive patients whether drug therapy is to be given or not. Four factors determine physician's decision to initiate drug therapy Level of BP on initial examination, presence of target organ damage, other cardiovascular risk factors and BP response to non-pharmacologic treatment. Patients with moderate and severe hypertension with persistent elevation of SBP>180 mmHg and/or DBP>110 mmHg need pharmacologic intervention within one week if BP remains high. Close follow-up and repeated BP measurement over days or weeks, depending on its level, are recommended. In the presence of diabetes mellitus or target organ damage, e.g. left ventricular hypertrophy, heart failure, coronary artery disease, retinopathy, renal failure, transient cerebral ischemic attacks, stroke and peripheral arterial disease, drug treatment should also be initiated within days as soon as high BP is confirmed (> 140/90 mm Hg). Patients with other cardiovascular risk factors such as dyslipidemia, cigarette smoking, male gender, age above 65 years, obesity, impaired glucose tolerance, positive family history of cardiovascular death or events at young age and have mild hypertension (SBP 140-180 mm Hg, DBP 90-110 mm Hg) should continue non-pharmacologic treatment for 3 to 6 months and have their BP checked every 1 to 2 months. Drug therapy should be initiated if BP is persistently above 140/90 mm Hg. Non-pharmacologic treatment includes correction of obesity and weight control, limiting salt and alcohol intake, dietary modification, exercise, stress management and addressing other cardiovascular risk factors. Patients with mild hypertension and without diabetes mellitus, target organ damage or cardiovascular risk factors should continue non-pharmacologic treatment for 6 to 12 months before considering drug therapy. If BP remains elevated at the end of this period, antihypertensive therapy should be initiated.

M. Mohse,; Ibrahim, MD

**Prof. & Chairman, Department of Cardiovascular Medicine - Cairo University.
President of The Egyptian Hypertension Society.**

THE PRESIDENT'S MESSAGE HYPERTENSION WHEN TO INITIATE DRUG THERAPY?

Pharmacologic therapy proved effective in improving morbidity and mortality in hypertensive patients. Since there is no cure from established essential hypertension, it is obvious that once drug therapy is started, it has to be continued for many years and most probably indefinitely. Costs, side effects and accordingly patient's compliance are important limitations when considering life-long drug treatment. For these reasons initiation of drug therapy is not an easy decision, should not be taken lightly and should be based on strong grounds. From the very beginning it is mandatory to establish the presence of hypertension by repeated and accurate blood pressure (BP) measurements over variable intervals depending upon BP level. Non-pharmacologic treatment should be started in all hypertensive patients whether drug therapy is to be given or not. Four factors determine physician's decision to initiate drug therapy Level of BP on initial examination, presence of target organ damage, other cardiovascular risk factors and BP response to non-pharmacologic treatment. Patients with moderate and severe hypertension with persistent elevation of SBP>180 mmHg and/or DBP>110 mmHg need pharmacologic intervention within one week if BP remains high. Close follow-up and repeated BP measurement over days or weeks, depending on its level, are recommended. In the presence of diabetes mellitus or target organ damage, e.g. left ventricular hypertrophy, heart failure, coronary artery disease, retinopathy, renal failure, transient cerebral ischemic attacks, stroke and peripheral arterial disease, drug treatment should also be initiated within days as soon as high BP is confirmed (> 140/90 mm Hg). Patients with other cardiovascular risk factors such as dyslipidemia, cigarette smoking, male gender, age above 65 years, obesity, impaired glucose tolerance, positive family history of cardiovascular death or events at young age and have mild hypertension (SBP 140-180 mm Hg, DBP 90-110 mm Hg) should continue non-pharmacologic treatment for 3 to 6 months and have their BP checked every 1 to 2 months. Drug therapy should be initiated if BP is persistently above 140/90 mm Hg. Non-pharmacologic treatment includes correction of obesity and weight control, limiting salt and alcohol intake, dietary modification, exercise, stress management and addressing other cardiovascular risk factors. Patients with mild hypertension and without diabetes mellitus, target organ damage or cardiovascular risk factors should continue non-pharmacologic treatment for 6 to 12 months before considering drug therapy. If BP remains elevated at the end of this period, antihypertensive therapy should be initiated.

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SCIENTIFIC NEWS

- The Kampo medicine Shichimotsukoka-to (SKT) is used to treat hypertension & atherosclerosis in Japan by enhancing serum NO. This suggests that it may become a unique new orally active NO donor.
- An effective choice in patients with hypertension & concomitant dyslipidemia or type 2 diabetes mellitus is the use of Urapidil. It is a newly introduced formulation

with a peripheral postsynaptic α -adrenoceptor antagonistic but with a central agonistic action at serotonin 5-HT_{1A} receptors.

ABPM, beyond being that effective as a diagnostic tool, is advised to be further used as a new determinant of patient's compliance to treatment.

• For essential hypertension, new antihypertensives are now being tested in clinical practice for the new millennium as Selective II imidazoline receptor binding agents, vasopeptidase inhibitors & endothelin antagonists.

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Editorial

LEPTIN: ITS CARDIOVASCULORENAL INTERLINKS AMER NOAM

**Ass.Prof Physiology, Faculty of Medicine, Alexandria
University**

Obesity is rapidly increasing in most industrial societies. In particular upper-body obesity [android or visceral] is considered one of the strongest predictors for the development of hypertension along with other cardiovascular morbidity & mortality. No wonder, body adiposity has deserved much concern and its regulation over the long term has been the focus of intense attention.

In this respect, different strategies are invoked to maintain stable adiposity including alterations in food intake, spontaneous activity, metabolism, metabolic efficiency, and thermogenesis. Although all of these responses, or efferent mechanisms, are coordinated by the central nervous system, yet how this can be achieved was not fully clear. Among what has been advocated to elucidate this is the lipostatic theory for the regulation of food intake. This states that an afferent signal produced by, or indirect relation to body adiposity provides the brain with the necessary information to control body fat stores. At a minimum, such proposed afferent signal must be; proportional to body fat, circulates in the blood, acts in the brain and have predictable effects on behaviour, particularly on food intake and metabolism.

Luckily, after intense molecular research, the promising candidate that satisfied the criteria of a long term regulator of adiposity signal, turned out to be, the ob (obese) gene product leptin (from the Greek leptos meaning "thin". Thus, ever since the initial report of the ob cDNA in December 1994, the literature concerning leptin is endlessly expanding and its attributes to many diseases is the interest of many researchers.

Now the baseline knowledge states that, obese individuals have high, and lean individuals have low leptin circulating levels; meaning that leptin is found to correlate positively with body adiposity. Subsidiary, ob/ob mouse, which do not produce functional leptin, and the db/db mouse and fa/fa rat, which are insensitive to leptin (now known to have a leptin receptor defect) are profoundly obese and may additionally develop type II diabetes. The treatment of the ob/ob mouse with exogenous leptin normalizes considerably its eating behavior, obesity and metabolism, while this effect is much less marked in the db/db mice and fa/fa rats that are hardly responsive to exogenous leptin.

Beyond this, increasing evidence suggests that leptin influences autonomic, cardiovascular and renal regulations. In this domain, the role of leptin in regulating the sympathetic outflow is evolving whereby leptin-treated animals have higher core temperatures, higher metabolic rates and a simultaneous increase in norepinephrine turnover i.e it is involved in the sympathetic modulation of thermogenic organs. Also, a correlation between basal muscle sympathetic nerve activity (MSNA) [a direct measure of sympathetic nervous outflow] and plasma leptin concentration was of a magnitude similar to that between MSNA and body fat. All such reports emphasize that epinephrine may play a role in mediating the effects of leptin to reduce body weight.

Still tackling the cardiovascularrenal links of leptin, its ICV administration to normal rats, increased their arterial blood pressure by decreasing the arterial flow to skeletal muscles and splanchnic vascular bed. Yet, on the other hand, other experimental studies have cleared, that intravenous leptin administration in anaesthetized rats did not concomitantly induce an overt increase in arterial pressure nor heart rate despite increasing the overall sympathetic nerve activity. This denotes that leptin may have acted simultaneously by other mechanisms to offset the appealing vasoconstrictor effects of increased sympathetic outflow. This was perceived, as to an increase in renal tubular sodium and water excretion, by leptin, that could have ameliorated, on short term, its pressor responses. This highlighted the necessity of a longer term exposure to hyperleptinaemia for full expression of its renal sympathoexcitation, a situation comparable to what actually happens in obese rats subjected to chronic administration of leptin, whereby their arterial pressure and heart rate are found increased.

Clinically, a significant positive correlation between mean arterial blood pressure and leptin was demonstrated in patients with essential hypertension. These data suggest that leptin may serve as a link between obesity and hypertension.

The mechanisms through which leptin cause sympathoactivation are not clear. Given that leptin is transported into cerebrospinal fluid by a specific saturable transport process, the CNS thus becomes the plausible site for the actions of leptin on sympathetic nerve traffic. A receptor-mediated effect is supported by substantially

decreased sympathoactivation in Zucker rats, that are known to possess a mutation in the gene for the leptin receptor. Furthermore, specific binding sites for leptin were observed in the adrenal medulla with no specific binding in the adrenal cortex suggesting that leptin may have a direct effect on epinephrine-secreting cells in the adrenal medulla.

In light of the aforementioned and based on the fact that human obesity is associated with circulating hyperleptinaemia, an overview as to the problem of obesity was posed. This conceives obesity in part to be a deficiency in penetration of leptin into the CNS or resistance to its actions. This will be expressed as deficiency in thermogenesis as well as increases in appetite that will recruit obesity. Thus the overall sympathoactivation secondary to resistance to leptin may offer an explanation to the deleterious cardiovascular consequences of obesity and may be clinically relevant to the therapeutic potentialities of leptin administration or its synthetic analogues in preventive cardiology.

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ABSTRACTS OF WORLD LITERATURE

AGE-RACE SUBGROUP COMPARED WITH RENIN PROFILE AS PREDICTORS OF BLOOD PRESSURE RESPONSE TO ANTIHYPERTENSIVE THERAPY

Preston RA, Materson BJ, Reda DJ, Williams DW, Hamburger RJ, Cushman WC, Anderson RJ.

Department of Veterans Affairs Cooperative Study Group on Antihypertensive Agents, Miami, USA.

Objective: To compare the plasma renin profiling and age-race subgroup methods as predictors of response to single-drug therapy in men with stage 1 and 2 hypertension as defined by the Joint National Committee on Prevention, Detection, Evaluation and Treatment of High Blood Pressure. **Design:** The Veterans Affairs Cooperative Study on Single-Drug Therapy of Hypertension, a randomized controlled trial. **Setting:** Fifteen Veterans Affairs hypertension centers. **Patients:** A total of 1105 ambulatory men with entry diastolic blood pressure (DBP) of 95 to 109 mm Hg, of whom 1031 had valid plasma and urine samples for renin profiling. **Interventions.** Randomization to 1 of 6 antihypertensive drugs: hydrochlorothiazide, atenolol, Captopril, clonidine, diltiazem (sustained release), or prazosin. **Main Outcome Measure-Treatment response** as assessed by percentage achieving goal DBP (<90 mm Hg) in response to a single drug that corresponded to patients' renin profile vs a single drug that corresponded to patients' age-race subgroup. **Results.** Clonidine and diltiazem had consistent response rates regardless of renin profile (76%, 67%, and 80% for low, medium, and high renin, respectively, for clonidine and 83%, 82%, and 83%, respectively, for diltiazem for patients with baseline DBP of 95-99 mm Hg). Hydrochlorothiazide and prazosin were best in low and medium-renin profiles; Captopril was best in medium- and high-renin profiles (low-, medium-, and high-renin response rates were 82%, 78%, and 14%, respectively, for hydrochlorothiazide; 88%, 67%, and 40%, respectively, for prazosin; and 51%, 83%, and 100%, respectively, for Captopril for patients with baseline DBP of 95-99 mm Hg). Response rates for patients with baseline DBP of 95 to 99 mm Hg by age-race subgroup ranged from 70% for clonidine to 90% for prazosin for younger black men, from 50% for Captopril to 97% for diltiazem for older black men, from 70% for hydrochlorothiazide to 92% for atenolol for younger white men, and from 84% for

hydrochlorothiazide to 95% for diltiazem for older white men. Patients with a correct treatment for their renin profile but incorrect for age-race subgroup had a response rate of 58.7%; patients with an incorrect treatment for their renin profile but correct for age-race subgroup had a response rate of 63.1 % (P= .30). After controlling for DBP and interactions with treatment group, age-race subgroup (P<.001) significantly predicted response to single-drug therapy, whereas renin profile was of borderline significance (P= .05). Conclusions: In these men with stage I and stage 2 hypertension, therapeutic responses were consistent with baseline renin profile, but age-race subgroup was a better predictor of response.

JAMA 1998; 280: 1168-1172.

EFFECTS OF A TRADITIONAL LIFESTYLE ON THE CARDIOVASCULAR RISK PROFILE: THE AMONDAVA POPULATION OF THE BRAZILIAN AMAZON. COMPARISON WITH MATCHED AFRICAN, ITALIAN AND POLISH POPULATIONS

Pavan L, Casiglia E, Carvalho Braga LM, Winnicki M, Puato M, Pauletto P, Pessina AC.

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Lucas de Ouro Preto do Oeste, Rondonia, Brazil & Department of Hypertension & Diabetology, University of Gdansk, Poland.

OBJECTIVE: To determine the relationships between lifestyle and cardiovascular risk factors among the Brazilian Amondava, one of the world's most isolated populations. **DESIGN:** Cross-sectional, population-based study. Four age- and sex-matched samples from Brazil Africa, Italy and Poland, representing different levels of modernization, were compared. Body weight, height, blood pressure, serum cholesterol and glycaemia were measured, and a standard questionnaire administered. Data concerning dietary habits and physical activity were collected. A personal socio-economic score was calculated, on the basis of type of economy, level of formal education, type of occupation, type of habitat, availability of piped water and electricity, main source of income, housing conditions, availability of radio, television or personal computer, knowledge of a second language, and organized health facilities. **SETTING:** Primary epidemiological screening, at an institution. **RESULTS:** Among the Amondava blood pressure was always < 140/90mmHg, it did not increase with age and was not correlated with any other variable; 46.6% of subjects had systolic blood pressure < 100mmHg. Blood pressure among the Amondava ($109.6 \pm 11.1/69.5 \pm 6.4$ mmHg) was on average lower (P < 0.0001) than in all other samples. Among the Amondava, the concentration of total cholesterol was always < 200mg/dl, i.e. similar to that of Africans whose diet included large amounts of vegetable foodstuffs; 90% had glycaemia (< 80mg/dl), and their mean value was the lowest (55.1 ± 14.9 mg/dl) of all the groups. **CONCLUSIONS:** In addition to a possible genetic predisposition not analyzed in this study, a traditional lifestyle (no contact with civilization, diet based on complex carbohydrates and vegetables, high energy expenditure) may protect against the development of hypertension, hypercholesterolaemia, and diabetes.

Hypertension 1999; 17(6): 749-756

**ABSTRACTS OF LOCAL LITERATURE
CAROTID ARTERY WALL THICKNESS AND ENDOTHELIAL
DYSFUNCTION IN PATIENTS WITH ESSENTIAL HYPERTENSION**

M.El-Masry, M. Seteha, M. Salama

Department of Cardiology, Faculty of Medicine, Tanta University. Egypt

The purpose of this study was to evaluate the relationship between the vascular reactivity and carotid intima-medial thickening (IMT) in essential hypertensive patients. The study population included 20 normotensive subjects and 45 patients with essential hypertension. None were smokers, or had other vascular risk factors. Each subject underwent the following examinations: B-mode ultrasound imaging of the carotid artery, echocardiographic examination, and arterial physiologic testing: the right brachial artery diameter was measured on B-mode ultrasound images with the use of a 7.0 MHz linear array transducer (Acuson128xp) Scans were recorded at rest, during reactive hyperemia (flow mediated dilatation "FMD" = endothelium-dependent dilatation and after sublingual nitroglycerine "NTG" = endothelium independent dilatation. Carotid wall IMT showed a significant inverse correlation with FMD and age, whereas no correlation was observed with the response to NTG, LV mass index, systolic and diastolic blood pressures, and plasma cholesterol and glucose levels. Moreover, FMD showed no correlation with LV mass index The present data indicate that in patients with essential hypertension, carotid wall thickening is associated with reduced endothelium-dependent in the systemic arteries and suggest that endothelial dysfunction might be involved in the preclinical phase of arterial disease in essential hypertensive patients who are at risk for atherosclerosis and its complications in later life.

Presented in the 26th Annual Meeting of the Egyptian Society of Cardiology, Cairo, Egypt. February 1999.

**STUDY OF HEART RATE VARIABILITY IN HYPERTENSIVE PATIENTS
BY USING 24-HOUR AMBULATORY ECG RECORDING**

Hanaa M.Fereig, Watia B.Eteiba, Mervat A. Nabih, Osaila H.El Khatib.

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Faculty of Medicine, Am Shams University.**

Background It is well known that blood pressure is under the influence of the autonomic nervous system (ANS) . Heart rate variability (HRV) represents a promising non invasive marker of autonomic activity The aim of our study is to evaluate ANS response in hypertensive patients . Methods : The study included 40 subjects, 30 hypertensive patients and 10 normotensive control subjects. We excluded patients with ISCHEMIC heart disease, atrial fibrillation, congestive heart failure, diabetes mellitus, and digitalis medication. All of the 40 subjects were subjected to medical history, cardiac examination, ECG, transthoracic Echocardiography and 24-hr. Holter monitoring. Both time and frequency domain measures of HRV were calculated over 24-hr period. Results: Our results revealed the following: In control

versus patients; SDNNi (58.8±12.9) vs (47.1±15.2) rMSSD (46.9±15.3) vs (36.1±22.7) pNN50 (10.4±7.2) vs (6.2±7.2) LF power (890.3±620.6) vs (541.2±493.4) HF power (417.9 ± 251) vs (358.9±501) LF/HF (2.2±0.98) vs (2.9±0.75). Conclusions: SDNNi, RMSSD, pNN50 and LF power were significantly decreased in hypertensive subjects. The decrease in HF power and the increase in LF/HF ratio in hypertensive subjects were insignificant.

Presented in the 26th Annual Meeting of the Egyptian Society of Cardiology, Cairo, Egypt. February 1999.

CHALLENGE YOUR SELF !!!

A 51-year old man had been in his usual state until the day of admission when he complained of acute onset of dyspnea. There had been no previous orthopnea, and he denied angina, palpitation or syncope. He had a 3-year history of hypertension that has been poorly controlled by hydrochlorothiazide. The patient reported episodes of diaphoresis and flushing. Physical examination: Vital signs: pulse 110 and regular, Blood pressure: 240/120, General: moderate respiratory distress. Fundi: arterial narrowing, arteriovenous nicking but no papilloedema Chest: diffuse bilateral rales. Cardiac: Summation gallop, no murmurs. Laboratory findings: CBC: normal. Na: 140mEq/L; K: 4.2m eq/L; Cl: 102meq/L; HCO₃:26mEq/L; BUN 30mg/dl; Cr: 1.2 mg/dl. EKG: normal sinus with LVH. Hospital course; After administration of oxygen, nitroglycerine furosemide 40 mg intravenously and sublingually, the patient improved rapidly.

Question,' What should the evaluation of a patient with severe hypertension include?

Pick up the solution at CARDIOLOGY PEARLS on P. [7] of this issue.

PATIENTS' ADVISORY CORNER DIET IN HYPERTENSION

- For years, doctors have advised reducing sodium to help lower blood pressure[BP], s'o one should check the levels of sodium listed on food labels. One should avoid canned or prepared foods unless their label clears that no salt is added. Effervescent medication should not be forgotten as a source of high Na intake.
- The quality OF food in the diet also matters. Now, they've discovered that a diet rich in fruits, vegetables and low-fat dairy products can lower blood pressure. That is why a new eating guide called the DASH diet, published by Mayo Clinic in April 1998 to help one prevent or guide patients to lower their high BP. The name evolved from the study" Dietary Approaches to Stop Hypertension." where for 8 weeks, participants followed one of three diets - a diet that matched the average American diet, a diet rich in fruits and vegetables, and a "combination" diet that was reduced in saturated fat and emphasized fruits, vegetables and low-fat dairy products. Sodium consumption in all three was about 3,000 milligrams (mg) a day.

Results revealed that fruits and vegetables and combination diets both lowered BP, but the combination diet was most effective. In that group, the decrease was the

greatest for those with high BP(above 140/90) with an average drop of 11.4 points in systolic pressure and 5.5 points in diastolic pressure. That's about the same effect as some medications. Researchers are n't sure why the combination diet fared better. However, they believe it's due to the mixture of nutrients provided, rather than any single ingredient.

The original publication has submitted the following table where the number of servings one should consume daily from each food group is cleared. Serving amounts are based on a diet of 2,000 calories per day.

Food/servings	1 serving equals	Food examples
Grains & grain products to 8 daily	1 slice bread 1/2 cup dry cereal	Whole wheat breads, English muffins, pita bread, bagels, cereals, oatmeal grits
Fruits & vegetables to 5 fruit servings daily to 5 vegetable servings daily	6 oz. fruit or vegetable juice 1 medium fruit 1/2 cup frozen or canned fruit 1 cup raw, leafy or 1/2 cup cooked vegetables	Apricots, bananas, grapes, oranges, grapefruit, melons, strawberries, tomatoes, peas, carrots, potatoes, broccoli, squash, leafy greens
Dairy foods (low-fat or non-fat) 2 to 3 daily	8 oz. Milk 1 cup yogurt 1 1/2 oz. cheese	Skim or 1% milk, nonfat or low fat yogurt, nonfat or part-skim cheese
Meats, poultry & fish or fewer daily	3 oz. cooked meat, poultry or fish	Lean meats only; trim visible fat, remove skin from poultry; broil, roast or boil
Nuts, seeds & legumes to 4 a week	1/3 Cup nuts 2 tablespoons seeds 1/2 Cup cooked legumes	Almonds, peanuts, mixed nuts, sunflower seeds, kidney beans, lentils

The merits of such a diet are many;

If your BP is normal, the DASH diet may help you avoid BP problems. While if it is only slightly elevated, the diet may actually eliminate the need for medication and if severe, it may allow you to reduce your medication. However, don't stop or alter your medication without first consulting your doctor.

The DASH diet can't do it alone, but it is important that one takes other steps to control or prevent hypertension, including exercising, losing excess weight, not smoking and limiting alcohol.

Beyond this the DASH diet may improve health in other ways, as fruits and vegetables may reduce the risk for some cancers, the calcium in dairy products can lower risk for osteoporosis and a diet low in saturated fats and cholesterol can reduce cardiovascular disease risk.

Mayo Clinic Health Letter, April 1998.

- Soft drinks containing caffeine should be limited, refraining from alcohol is advisable & smoking should be prohibited.

Focusing on tea in particular, the caffeine within tends to raise the blood pressure acutely temporarily more than expected specially in black tea, while the flavonoids

are thought to protect against heart disease and other disorders, specially when present in appropriate concentrations in green tea. However, over long terms neither type of tea were thought to have a significant effect on blood pressure.

In an Australian study, conducted to illustrate the acute effects of administration of regular black tea, Japanese green tea, in comparison to hot water with the same amount of caffeine; the blood pressure after one hour was increased about 6 mm Hg systolic and 3 mm Hg diastolic, by caffeine. However black tea produced a bigger rise [16/8 mm Hg] while green tea produced an intermediate rise [12/6 mm Hg]. When this was repeated in borderline hypertensives that drank 5 cups of tea a day for three weeks, and their 24 hours blood pressure was measured by ambulatory monitoring at the end of each week , then no effect of either tea or caffeine on blood pressure was recorded.

The acute and long term effects of tea deserve to be well delineated in wide scale control trials.

J Hyperten 1999; 17457.

CARDIOLOGY PEARLS

1. Although many patients with pheochromocytoma have episodic elevations in blood pressure thought typical of the disease, in 50% of cases the blood pressure is consistently elevated without distinct variations.
2. Other symptoms, including palpitations, headache, and diaphoreses, are common in phaeochromacytoma but are usually absent in other forms of hypertension.
3. CT scanning is an excellent diagnostic tool for localizing the tumor once biochemical evidence has established tumors existence.

• The editorial committee of the News Letter of Egyptian Society of Hypertension, is now changing Prof Dr. Mohsen Ibrahim on behalf of the editing board is whole heartily thanking Dr. Mohamed Hamed the former editor for his executive elegant and distinguished collaboration in making this News Letter come to reality and progress throughout the previous years. His words of gratitude implies:

The Egyptian Society of Hypertension members wish to express their thanks to professor Mohamed Hamed M.D, editor of the EHS Newsletter since it was started in 1995. His dedication and interest were manifest in every issue of the paper and helped to make the work and aims of the EHS known to doctors in all health care centers in Egypt and Hypertension Societies abroad. We wish Professor M. Hamed every success in his present endeavor and look forward to his contribution to the newsletter.

M. Mohsen Ibrahim

Now, Prof. Dr. Hassan Khalil, Prof of Cardiology, Alexandria University has been nominated, editor in chief from this June issue and onwards.

- The EHS has held a symposium on the 11th of March in El-Menia at Upper Egypt to enlighten physicians there about hypertension, its prevalence, its diagnosis and its lines of treatment. Another symposium was held in Cairo on the 13th of May entitled "What's new in hypertension?" during which the general assembly of the Society was held and the annual report on scientific activities, publications, research, finances etc. The assembly settled on dividing active participants into several working committees namely for ; continuous medical education & symposia, publicity & public education, finances, publication, Internet advertisement & newsletter, scientific research & drug assessment, communication with international & Pan-Arab Societies, ... etc. These committees will run cyclic meetings to encompass all such expandable activities of the society.

- A book on "Ischaemic Heart Disease." is due to be completed and issued by the society. Its manuscript has been already finalized in El Gona on January 1999.
- The society has issued a 2nd edition of ' Short review on hypertension- Egyptian Hypertension Society Guidelines.' Also three guideline booklets were issued on ; How to measure blood pressure - Hypertension in pregnancy-Diagnosis of chest pain.
- The Internet web site of the society has been logged in.; <http://www.ehs-egypt.net>.
- Our E-mail address is ehs@link.com.eg

LOCAL MEETINGS

The Summer Meeting of the Society	Helnan Palestine Hotel June 24-25 th ,1999	Prof. Dr. Mohamed Sobhy Tel (203)4203288 Fax (203) 420 32 88
1st Annual Meeting of Working Group of Heart Failure	Helnan Palestine Hotel September 9-10, 1999.	Prof Dr. Wagdy Ayad Fax (203) 425 11 57
4th Annual International & Pan-Arab Meeting of Interventional Cardiology	Main Conference Hall Alexandria	Prof Dr. Mohamed Sobhy Tel (203) 420 32 88 Fax (203)

INTERNATIONAL MEETINGS

9th European Meeting on Hypertension	Milan Italy. June 11-15 1999	Contact: AISC via A. Rom,Italy Fax 39-6-8088491
XVII Interamerican Congress Of Cardiology	Buenos Aires ,Argentina Aug 22-25,199911-15 1999	Contact: Argentine Society of Cardiology ,Buenos Aires Fax : [54-1]9616020
XI Congress of European Society of Cardiology [ESC] 1 st Meeting,Asian-Pacific Society of Hypertension	Barcelona,Spain.Sep 4-8, 1999 Ball,Indonesia,September 16-18,1999	Contact : ECOR,Sophia Antipolis Cedex, France.Fax :+33-492947601 National Cardio Center,Jakarta, Indonesia

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Salwa Morkos, MD

Volume 5 Issue 3

Obesity is a common health problem in many Western Industrial countries. It is an important risk factor for hypertension, coronary and other cardiovascular disease. Prevalence of obesity and its correlation with other cardiovascular risk factors was not known in Egypt and many developing countries. During a cross sectional survey - the Egyptian National Hypertension Project - body weight, body mass index (BMI) and waist/hip (W/H) measurements were made on a random sample of 2292 individuals, 751 normotensives and 1541 hypertensives, age ranged 25-90 years. The survey was conducted in 21 sampling locations representing all Egyptian geographic regions and socioeconomic groups. Blood pressure was measured using a standardized protocol and blood samples were taken while fasting and 2 hours after 75 gm oral glucose samples were analyzed for sugar, cholesterol and triglycerides. Individuals were evaluated clinically for signs of heart failure, the presence of two or more of the following was consistent with clinical heart failure; cardiac dyspnea, ankle oedema, pulmonary congestion, raised jugular venous pressure and abnormal third heart sound. Obesity defined as BMI greater than 30 kg/m² was present in 26.6% of NT (13.9 in M, 35.6 % in F) and in 46.3% of hypertensives (26.4 in M 50.7% in F), W/H was 0.87 in NT (0.9 in M, 0.85 in F) and 0.90 in HT (0.93 in M, 0.88 in F). Systolic and diastolic BP correlated best and significantly (p0.000 in all). Triglycerides correlated best with W/H (r0230, p25 kg/m²) showed that obese individuals whether NT or 1-IT have faster heart rate, more urban distribution, higher levels of F, PPBS, triglycerides and cholesterol levels. All levels were higher in HT than in NT. Diabetes mellitus, hypertriglyceridemia, hypercholesterolemia and clinical heart failure were more prevalent in the obese group, while cigarette smoking was more common in the non-obese individuals.

Conclusion:

Obesity is very prevalent in Egyptians, especially hypertensive women and more common in urban than rural areas.

DM, HTG, H Cholest.. and HF are more common in obese than non-obese individuals. BP correlates more with body fat distribution (W/H) than with BMI or body weight.

M Mohsen Ibrahim M.D.

**Prof & Chairman, Department of Cardiovascular Medicine -
Cairo University President of The Egyptian Hypertension Society**

THE PRESIDENTS MESSAGE

Obesity is a common health problem in many Western Industrial countries. It is an

important risk factor for hypertension, coronary and other cardiovascular disease. Prevalence of obesity and its correlation with other cardiovascular risk factors was not known in Egypt and many developing countries. During a cross sectional survey - the Egyptian National Hypertension Project - body weight, body mass index (BMI) and waist/hip (W/H) measurements were made on a random sample of 2292 individuals, 751 normotensives and 1541 hypertensives, age ranged 25-90 years. The survey was conducted in 21 sampling locations representing all Egyptian geographic regions and socioeconomic groups. Blood pressure was measured using a standardized protocol and blood samples were taken while fasting and 2 hours after 75 gm oral glucose samples were analyzed for sugar, cholesterol and triglycerides. Individuals were evaluated clinically for signs of heart failure, the presence of two or more of the following was consistent with clinical heart failure; cardiac dyspnea, ankle oedema, pulmonary congestion, raised jugular venous pressure and abnormal third heart sound. Obesity defined as BMI greater than 30 kg/m² was present in 26.6% of NT (13.9 in M, 35.6 % in F) and in 46.3% of hypertensives (26.4 in M 50.7% in F), W/H was 0.87 in NT (0.9 in M, 0.85 in F) and 0.90 in HT (0.93 in M, 0.88 in F). Systolic and diastolic BP correlated best and significantly (p<0.000 in all). Triglycerides correlated best with W/H (r=0.230, p<0.000). A study (p25 kg/m²) showed that obese individuals whether NT or HT have faster heart rate, more urban distribution, higher levels of F, PPBS, triglycerides and cholesterol levels. All levels were higher in HT than in NT. Diabetes mellitus, hypertriglyceridemia, hypercholesterolemia and clinical heart failure were more prevalent in the obese group, while cigarette smoking was more common in the non-obese individuals.

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SCIENTIFIC NEWS

- A Three-dimensional helical CT angiography of renal transplant recipients presenting with hypertension, graft dysfunction or both, is now available and can provide valuable information that might be used to guide their further therapy.
- For perioperative hypertension & in hypertensive emergencies & crises a new parenteral antihypertensive; Fenoldopam: [a dopamine receptor (DA I selective) agonist] has recently been approved by the (FDA).

Pharmacoeconomics of therapy, is a hot issue in all cardiology meetings in trial to solve the challenge with balancing healthcare costs & quality of life.

As &1—adrenergic hypothesis is now linked to the pathophysiology of pulmonary

hypertension, so the use of α_1 agonists for appetite suppression and other disease should be avoided.

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MOLECULES IN FOCUS

“AN INNOVATIVE ANTIHYPERTENSIVE CONCEPT FOR THE NEW MILLENNIUM”

Omnia Nayel

**Prof. of Pharmacology, Faculty of Medicine,
University of Alexandria.**

Despite five decades have lapsed in the progress of management of hypertension; the disease still commonly worldwide prevails. This necessitates an endless need for emergence of new therapeutic trends that could be preferentially tailored reasonably well to patients needs; whether to dosage requirement, metabolic profile neutrality, safety to concomitantly associated diseases or other cardiovascular risks etc. Furthermore, the patient's compliance to a seemingly symptomless yet progressive disease adds a third dimensional depth that weighs to blood pressure control; whereby tolerance will mean compliance and compliance will mean efficacy of the antihypertensive that is meant to be addressed in this new millennium.

In such a domain the concept of striking with a drug on higher central controls initiating and maintaining high blood pressure is now being revived but with a newer insight. This renewed interest resides on a global trend to perceive the increase in pressure as a malfunction [loss of receptor sensitivity] of the adaptive emergency response that pertains cerebral and coronary supply sufficient enough to cope with body demands. The logic then would be to intervene with an antihypertensive that deals with such complex regulation in a rational, physiological mode rather than to fight against it.

The concept set to achieve this was through the use of a substance with an imidazoline-like structure as rilmenidine, that can bind selectively to I1 [imidazoline] receptors, located in the rostro-ventrolateral medulla of the brain stem, resulting in a decrease in the sympathetic outflow. However, it still remains debatable how

rilmenidine provokes this genuine agonistic action but it has been hypothesized that it acts there as an inverse agonist.

Through this, rilmenidine will reinitialize such maladaptive responses, resetting the set point of baroreflex back, so as to normalize blood pressure. The consequence of such sympathetic inhibition will be, a reduction in peripheral vascular resistance, yet fortunately without interfering with adaptation to standing or sitting upright [particularly in elderly] nor to exercise ... etc.

The drug thus controls the sympathetic overdrive to the heart and reduces the left ventricular end-diastolic and end-systolic volumes, whereas the stroke volume, cardiac output, and pulmonary artery pressures remain largely unchanged. Moreover such decrease in sympathetic outflow, is apt to reduce the renin, secretion

Beyond this, by selective binding of rilmenidine to b receptors in the kidney, it can simultaneously inhibit H⁺/ Na⁺ exchanger situated at the basolateral membrane of the proximal convoluted tubules which is responsible for Na⁺ absorption. Through this the drug can excrete natiuresis directly by decreasing water and sodium overload and indirectly by changing neural and hormonal influences on the kidney. In humans the drug was also reported to decrease glomerular filtration rate and infiltration fraction.

From aforementioned, it is clear that rilmenidine encompasses two key organs involved in pressure regulation whereby it controls the immediate [nervous] and delayed [renal] setup in a comprehensive physiological way without neglecting the body's abilities to adapt.

When this was translated to long term antihypertensive monotherapeutic efficacy assessment, over a year, in an open study conducted on mild to moderate hypertensives or in a multicenter pharmacoepidemiological open trial involving 2072 general physicians and 18,257 hypertensive patients many of which were suffering from concomitant conditions [diabetes, dyslipidemia, heart failure, renal failure, dysrhythmias, ... etc] all results came up with a solid statement” rilmenidine remains effective in long —term without any fading of effect.”

And since selective b receptor antihypertensives do not bind to any other receptors in the therapeutic range specified, it is not surprising that their adverse effects on other bodily functions is down to minimal. This adds to their safety utility to be used in patients whatever their comorbid disease and/or age is. Also a rebound (withdrawal) phenomenon has not been reported for such I~ receptor stimulants.

Thus striking on imidazoline receptors proved to be an innovative acceptable modality of an effective antihypertensive, that has come in to focus in this new millennium.

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ABSTRACTS OF WORLD LITERATURE
CLINICAL VALUE OF AMBULATORY BLOOD PRESSURE MONITORING
Mallion J7M, Baguet JP, Jean-Philippe Tremel SF, De Gaudemaris R
Medecine Interne et Cardiologie, CHU de Grenoble, France.

Ambulatory blood pressure monitoring (ABPM) has now become an established clinical tool. It is appropriate to take stock and assess the situation of this technique. Update on equipment: Important improvements in equipment have occurred, with reductions in weight, in awkwardness and in noisiness of the machines, better acceptability and tolerance by the patients, and better reliability. Validation programs have been proposed and should be referred to. Limitations of the technique persist with intermittent recording in current practice. The reproducibility is limited in the short-term while recording over 24 h is acceptable. Diagnosis and prognosis: White-coat effect (WCE) is manifested as a transient elevation in blood pressure during the medical visit. The frequency of this phenomenon, the size of the effect, age, sex and level of blood pressure (BP) or the situation of occurrence (general practitioner, specialist or nurse) have been interpreted differently. It does not seem that WCE predicts cardiovascular morbidity or mortality. White-coat hypertension (WCH) is diagnosed on the evidence of abnormal clinical measures of BP and normal ABPM. The latest upper limits of normality by ABPM recommended by the JNCVI are 135/85 mmHg

while patients are awake 120/75 mmHg while patients are asleep. If we accept these upper limits of normality in ABPM, WCH does not appear to be a real problem as regards risk factors or end-organ effects. In terms of prognosis, data are limited. Cardiovascular morbidity seems low in WCH but identical to that of hypertensive subjects in these studies. However, further studies are needed to confirm these results. WCH does not appear to benefit from anti-hypertensive treatment. It is obvious that the lower the BP regarded as the limit of normality, the less likely the occurrence of secondary effects of metabolism, or end-organ effects or complications in those classified as hypertensive.

24 hour cycle: One of the most specific characteristics of ABPM is the possibility of being able to discover modification or alteration of the 24 h cycle of BP. Non-dippers are classically defined as those who show a reduction in BP of less than 10/5 mmHg or 1000 between the day (06.00--22.00 h) and the night, or an elevation in BP. In contrast, extreme dippers are those in whom the BP reduction is greater than 20%.

Cardiovascular system: The data remain inconclusive with regard to the existence of a consistent relationship between the lack of a nocturnal dip in blood pressure and target organ damage. As regards prognosis, it seems that an inversion of the day--night cycle is of pejorative significance. Cerebrovascular system: Almost all studies have shown that non-dippers had a significantly higher frequency of stroke than dippers. In contrast, too great a fall in nocturnal BP may be responsible for more marked cerebral ischaemia.

Renal system: Non-dippers have a significantly elevated median urinary excretion of albumin. There is a significant correlation between the systolic BP and nocturnal diastolic BP, and urinary excretion of albumin. Various studies have confirmed the increased frequency of change in the 24 h cycle in hypertensive subjects at the stage of renal failure.

Diabetes: BP abnormalities should be considered as markers of an elevated risk in diabetic subjects but cannot be considered at present as predictive of the appearance

of micro-albuminuria or other abnormalities. ABPM is thus of interest in type I or type II diabetes both in the initial assessment and in the follow-up and adaptation of treatment. Pharmacotherapeutic uses: The introduction of ABPM has truly changed the means and possibilities of approach to the study of the effects of anti-hypertensive medications, with new possibilities of analysis such as trough--peak ratio smoothness index, etc.

J Hypertension 1999; 17(5): 585-595.

INSULIN RESISTANCE, EXERCISE CAPACITY AND BODY COMPOSITION IN SUBJECTS WITH TWO HYPERTENSIVE PARENTS

Andersen UB, Dige-Petersen H, Ibsen H, Skø P, Bruun NE, Vestergaard H, Cliristiansen C.

Department of Clinical Physiology and Nuclear Medicine, Hvidovre Hospital - Department of Clinical Physiology and Nuclear Medicine & Internal Medicine, Glostrup Hospital - Medical Department, Rigshospitalet & Steno Diabetes Centre, Gentofte Centre for Clinical and Basic Research, Ballerup, Denmark.

OBJECTIVE: To study insulin resistance in subjects with strong genetic predisposition to essential hypertension compared with non-disposed subjects.

SUBJECT: Thirty normotensive subjects aged 18--35 years whose parents both had essential hypertension, and 30 age- and sex matched subjects whose parents were both normotensive, were studied. Subjects or parents with diabetes and morbid obesity were excluded.

METHODS: The study comprised (1) a frequent sampling oral glucose tolerance test; (2) an isoglycemic hyperinsulinemic clamp study; (3) an analysis of body composition by dual-energy X-ray absorptiometry; (4) an exercise test with gas exchange analysis; and (5) investigation of composition of usual diet by diet registration for 5 days.

RESULTS: The 24-h diastolic blood pressure was higher in subjects predisposed to hypertension compared with the controls: 78.1 versus 74.0 mm Hg, but the insulin sensitivity index was similar: 312 versus 362. The two groups were similar in terms of body composition, exercise capacity and composition of usual diet. Resting and 24-h diastolic blood pressures were correlated to abdominal fat mass but not to insulin sensitivity.

CONCLUSION: Subjects with a strong genetic predisposition to essential hypertension had increased diastolic blood pressure compared with subjects with normotensive parents, but they were not insulin resistant. This may be due to the subjects being highly selected as to confounding factors. The increased blood pressure in the hypertension prone subjects could not be attributed to differences in body composition, exercise capacity or dietary habits.

I Hypertension 1999; 17(9); 1273-1280.

**ABSTRACTS OF LOCAL LITERATURE
APOPTOSIS MEDIATOR SOLUBLE FORMS IN PATIENTS WITH
ESSENTIAL
HYPERTENSION; COMPARATIVE EFFECTS OF CAPTOPRIL AND
FOSINOPRIL**

Enas Frafa, Morad Ahmed and Magdy El-Masry
Dept. of Cardiology and Clinical Path. Faculty of Medicine, Tanta University,
Egypt.

Fas receptors are cell-surface proteins and apoptosis signaling molecules. Apoptosis may have an important role in the pathogenesis and progression of cardiovascular disease. Angiotensin-II stimulates apoptosis in cultured cardiomyocytes. ACE inhibitors are used for treatment of hypertension. The present study was designed to determine the relationship among the soluble Fas, hypertension and ACE inhibitors. We determined the serum sFas in 40 patients with essential hypertension before and after 6 weeks of randomly allocated treatment with Captopril (n=20) and Fosinopril (n=20). The control group consisted of 15 age- and sex-matched normotensive subjects. Baseline sFas levels were higher in hypertensive patients than in normotensive controls (6.72 ± 0.32 versus 5.61 ± 0.21 units/mL, $p.05$). There was a direct correlation between baseline sFas and systolic Bp in hypertensive patients ($r= 0.365$, $P.05$). The 2 ACE inhibitors caused similar reductions in sFas levels and BP after the treatment period. Conclusion: These results indicated that circulating sFas levels are increased in essential hypertension and suggest the ability of ACE inhibitors to normalize sFas.

Presented in the 26th Annual Meeting of the Egyptian Society of Cardiology,
Cairo, Egypt. February 1999.

RENOPROTECTIVE EFFECT OF CALCIUM CHANNEL BLOCKERS
(AMLODIPINE AND
NIFEDIPINE) COMPARISON WITH ACE INHIBITOR (CAPTOPRIL) AND
ANGOTENSIN II
RECEPTOR ANTAGONIST (VALSARTAN)
Salah Naga, Salah Marzouk, Eman El-Eetr
Nephrology, Clinical Pathology & Physiology Departments, Faculty of Medicine,
Alexandria University

The renoprotective effect of CCBs : Amlodipine and Nifedipine were compared with the ACE inhibitor (Captopril) and AT1 antagonist (Valsartan). Eighty patients with mild to moderate hypertension secondary to mild to moderate renal insufficiency were divided into four groups (20 for each). Each group received one drug to control blood pressure for ten weeks. Blood urea, serum creatinine, GFR, and total urine protein and α_2 microglobulin were measured before and at the end of the study. Urea and creatinine decreased by Amlodipine ($t=3.95$ and 4.87), Captopril ($t=0.87$ and 2.95) and Valsartan ($t=5.75$ and 6.20), and increased by Nifedipine ($t=3.6$ and 3.15). GFR increased by Amlodipine ($t=2.83$) and Valsartan ($t=3.7$). Amlodipine, Captopril and Valsartan were equally effective in reducing urea ($F=15.53$), creatinine ($F=9.86$), α_2 microglobulin ($F= 8.54$) and proteinuria. In conclusion, Amlodipine, a calcium channel blocker was found to have a renoprotective effect similar to Captopril and Valsartan.

Accepted, in 3rd Conference of the Pan—Arab Hypertension Society, Abu
Dhabi, due to be held on Feb 2000.

CHALLENGE YOUR SELF !!!

A 60-year old man was admitted to the hospital 3 hours after the onset of sudden, severe interscapular pain. His pain reached full intensity immediately. A few hours later he noted that his right hand was cold . He had hypertension for many years and was a heavy smoker.

Physical Examination: Vital signs; pulse; 80 bpm & regular, blood pressure on the left arm: 220/140, after receiving nitropruside IV 220/110, on the right arm: 90/80. General: diaphoretic. Cardiac: faint diastolic murmur over the left sternal border. Extremities: right hand cold, right radial pulse absent; pulses present in the lower extremities; systolic bruit heard over the femoral arteries.

Laboratory investigation; Hct: 40% . EGG: LVH Chest X-ray: widening of the Aorta with intimal calcification.

Question: What is the most likely diagnosis?

Pick up the solution at **CARDIOLOGY PEARLS** on p. 171 of this issue.

THERAPEUTIC CONSIDERATIONS; DIABETIC HYPERTENSIVES

in such a category of patients wisdom implies knowing that;

Thiazide Diuretics: are cheap but can induce metabolic upset, especially at high doses (eg hyperglycaemia, hypokalaemia, hyper-uricemia, hypercholesterolaemia) & may cause impotence.

B-Blockers: are particularly useful in presence of Ischaemic heart disease but may be poorly tolerated (eg lethargy, impotence) & may mask symptoms of hypoglycaemia & delay recovery from it.

ACE inhibitors: are specific renoprotectives in IDDM with microalbuminuria or overt proteinuria & can reduce C.V. morbidity & mortality in patients with heart failure. However they may precipitate renal failure in those with renovascular disease & can induce troublesome dry cough.

Calcium Channel Blockers: are particularly effective in treatment of blacks & non-dihydro-pyridine prototypes may be useful if patients develop tachyarrhythmias but in those with renal disease dihydropyridines may occasionally worsen urinary albumin excretion despite improvements in BP.

&-Blockers: may improve lipid profile .& insulin sensitivity but can induce postural hypotension especially in patients with autonomic neuropathy.

Postgraduate Doctor ME 1999; Diabetic Supp: 10-13.

ENVIRONMENTAL HAZARDOUS: NOISE POLLUTION

A broad array of demographic, ergonomic, nutritional, and environmental factors are critical determinants of blood pressure.

Due to the current existing noise pollution that modernization has posed on us, the overall information processing of humanity has changed and the rate, quality and behavior at work has deteriorated. In blood pressure domains, the current hypothesis that exposure to noise is positively associated with hypertension is now being raised.

Occupational noise in particular was specified, where studies revealed that workers subjected to such stress constrain may recover a significant impairment in audiogram, a concomitant increase in HR, SBP, DBP and a decrease in galvanic skin stimulation, auditory & visual reaction times!!!

In such studies, initial pressure levels and duration of employment had an overall impact as evidenced by being more affecting borderline hypertensives. The age, body mass index, cumulative noise exposure, current use of blood pressure medications, and alcohol intake were significant predictors for systolic blood pressure while a threshold effect of cumulative noise exposure was a significant predictor of diastolic blood pressure.

On the contrary to occupational exposures, there is no support to the suggestion that there exists a simultaneous correlation between impulse noise exposure[as gun shots], noise-induced hearing loss, and self-reported elevated blood pressure.

In mothers subjected to repeated noise exposure and their preterm newborns were already suffering from chronic intrauterine stress, such as maternal smoking, maternal hypertension, and intrauterine growth retardation, the liability to a change in the maturation of the autonomic nervous system was raised and was thought to reflect a possibility of developing essential hypertension as they grow.

It was explained that extra-auditory effects of noise exposure is mediated by activation of the autonomic nervous system & hypothalamic-hypophyseal adrenal axis with a resultant increase in catecholamines from the adrenal medulla secondary to noise stress.

In this respect a study has elucidated that with buzzing noise; a parallel increase in plasma catecholamine, corticosterone, Angiotensin II, glucose and lipids were found. While electrophysiologically, the acetylcholine and choline acetyltransferase in rostral ventrolateral medulla were increased markedly raising the assumption that stress-induced hypertension was closely related to the activation of a Colinergeric system in rVLM.

Realizing the world wide prevalence of such a problem, the hazardous impacts of noise pollution, on cardiovascular morbidity and mortality deserves further attention in wide scale community controlled studies.

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4. Arch Environ Health 1999 Mar-Apr 54(2) : 7 1-8.

BED-SIDE TIPS:

ASSIST IN PERTAINING YOUR PATIENT'S COMPLIANCE.

- You should advise patients to keep on taking the pills and never to cut it down by just feeling well. "A patient has to know that there is no way that he can tell~ just by the way he feels, whether pressure is high or low.
- Even if your patient has his own blood pressure measuring device, and his blood pressure seems controlled, he has to know that nobody can ever tell what would happen if medications are stopped. So clear to him that; "Hypertension is a l~e long, initially symptomless but progressive disease with many morbidity outcomes, that one is fighting to halt. Insure that this is deeply posed in his mind.
- Also stress that; "Pills are not magic, and that they need his help c/they are to work as efficiently as they can to reduce pressure to normal." Emphasis that his contribution is worth a lot and could be achieved in part by his ability to change his lifestyle.
- Help your patient to bypass the major obstacle hindering his compliance; that is forgetting to take medication. So; "Raise to him the tools to combat forgetting by submitting such PILL — TAKING TIPS "
- For remembering taking the pills;
 - suggest: -Put the pills where they are linked to a part of your routine daily scenarios; as keeping them by the toothbrush, razor, where one eats or watches TV.
 - Write a changeable weekly reminder aside them otherwise one may stop noticing them. This can be stuck on the refrigerator, kitchen .. doors - or on the bedroom, bathroom mirrors
 - Set a watch to keep at pill-taking time.
 - Ask the help of relatives or friends to remind taking the medications.
 - Sort the drugs in a " medication organizer" that can be bought or constructed, if more than one type of medication are to be taken.
- For not running out of pills;
 - suggest
 - Set up a system for renewal of the prescription by marking the date on the calendar or ask for the pharmacist's cooperation by sending reminder cards or phone rings if his system implies...
- For remembering taking pills while traveling;
 - suggest
 - Put reminders with the baggage; fasten it on the suitcase or travel kit.
 - Travel with a spare prescription, in case pills are forgotten.

**The Blood Pressure Book,
Bull Publishing Company, 1996**

CARDIOLOGY PEARLS

Diagnosis: Type I aortic dissection producing aortic regurgitation.

1. Aortic dissection should be suspected if the patient has chest, back, or abdominal pain that reaches maximum intensity immediately. Hypertension despite a shocky appearance, coupled with a normal ECG should further increase suspicion.
2. Aortic dissection should be suspected in any patient with chest pain who develops aortic insufficiency.
3. Marfan syndrome is likely if the dissection occurs under the age 40.
4. Drug therapy with nitroprusside and a beta blocker is the initial treatment in almost all cases of dissection; surgery should be performed if the ascending aorta is involved

EHS NEWS:

- The EHS has held its Summer Meeting of the Society in Helnan Palestine Hotel on June 24-25th, 1999 in Alexandria. The program covered many hot issues evolving these days within the etiopathogenic arena and has also witnessed scientific debates on updated guidelines.
- The EHS is organizing its fourth scientific meeting of the society on the 26th of January 2000 at Marriot Hotel, Cairo. The Chairman; Prof Dr. Mokhtar Gomaa has issued the 1st announcement. He has focused on how pathophysiology and management of hypertension is escalating in the direction of molecular biology. He wholeheartedly advises the necessity of focusing on such new concepts as we step in this new millennium. This will be aside the regular topics that basically should be discussed in such an important event. Awards will be presented by the society for young investigators.
- Members of the EHS are collaborating with Dr Abdulrahim Jaffer the Undersecretary — Ministry of Health of The U.A.E and the president of the 3rd Conference of the Pan — Arab Hypertension Society in finalizing the arrangements of such important event, due to be held in Abu Dhabi, 5-9 February, 2000.

EHS News & Calendar

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CALENDAR

LOCAL MEETINGS

4 Annual International & Pan	Main Conference Hall Alexandria	Prof Dr. Mohamed Sobhy Tel / Fax (203) 4203288
The fourth Scientific Meeting of the EHS	Marriot Hotel, Cairo Egypt January 28-29th, 2000	Prof Dr. Mokhtar Gomaa Tel (202) 3026871 -Fax (202) 3026871 E-mail : mogomaa@idscl.gov.eg

INTERNATIONAL MEETINGS

4th European Meeting Calcium Antagonists	Amsterdam, Netherlands. October 27-29, 1999.	Prof P.A. van Zwieten. Fax + 31206968704.
5th International Symposium Multiple Risk Factors in C.V.Diseases	Venice, Italy. October 28-31, 1999.	1st. M. Gotto, Jr? Fondazione Giovanni, Via Appiani, 720121Milan ? Italy.
4th World Congress of Echo- cardiography & vascular Ultrasound	Mena-House Oberoi,Cairo Egypt January 19-21, 2000.	Prof Dr. Osama Abdel Aziz Tel: (202) 3926650, Telefax: (202) 3602800 / 3958000

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Volume 5 Issue 4

The Egyptian Hypertension Society is planning to prepare a new book containing its recommendations and guidelines for management of heart failure. The success of our

two previous guideline books titled "Management of Hypertension" and "Management of Coronary Artery Disease" has stimulated the board of directors of the Egyptian Hypertension Society to approve, in its meeting on March 24th 1999, the establishment of a heart failure guidelines working group to prepare a new book titled "Heart Failure". The book will be addressing the practitioner, the internist and the cardiologist. It will contain the essential and modern information that will guide our Egyptian physicians in their everyday practice. Heart failure is possibly the most important cause of hospital admission to cardiology and general medical departments. Furthermore, developments in drug therapy in the past decade have changed favorably the course and outcome of this serious disorder in many patients. For a number of reasons heart failure has become an increasingly important problem in our clinical practice. First, there is a rising incidence of heart failure in the population, and it is expected that this trend will continue to increase in the coming years. The incidence of heart failure rises with aging. There is a worldwide increase in average life expectancy with an increase in the number of the elderly in many countries. Furthermore, the introduction in the recent years of effective therapies for hypertension, coronary artery disease and valvular heart disease has prolonged survival in many cardiac patients and delayed the development of heart failure.

Second, in the last two decades, a number of new therapeutic interventions were introduced for the management of heart failure. Many of these interventions not only improved patients' symptoms and quality of life but also prolonged survival of patients with heart failure and decreased rates of hospitalization and cardiovascular events. Angiotensin converting enzyme inhibitors (ACEI), beta adrenergic blockers, Angiotensin receptor blockers and spironolactone have now an established role in the routine management of heart failure patients. In addition, to these established drugs, new pharmacologic agents are under extensive investigation, which include neutral endopeptidase inhibitors (NEPI) (Candoxatrilat), combined NEPI and ACEI (Omipatrilat), endothelin receptors antagonists (Bosentan), natriuretic peptide (Nesiritide), tumor necrosis factor receptor antagonists (Etanercept), new classes of inotropic agents with a novel mode of action independent of the cyclic adenosine monophosphate (cAMP) pathway such as calcium sensitizers. These agents proved effective in improving the hemodynamics and effort tolerance in short term studies. The plethora of pharmacological agents combined with non-pharmacological interventions such as cardiac pacing, LV assist devices, surgery, gene and immunotherapy make the management of heart failure patients something more than simple digitalis, diuretics and bed rest.

Finally, the recognition that asymptomatic left ventricular dysfunction is a common condition, being the precursor of clinical heart failure in about one third of patients and the observation that ACEI therapy can delay the progression into overt heart failure, have two implications. First, that echocardiography should assume a central role as a screening tool for asymptomatic impaired LV function. Second, there are new insights into the possibility of prevention of heart failure through early intervention, particularly with the use of drugs (ACEI and beta blockers) in asymptomatic patients.

M. Mohsen Ibrahim M.D.

**Prof & Chairman, Department of Cardiovascular Medicine -Cairo University
President of The Egyptian Hypertension Society**

THE PRESIDENT'S MESSAGE INCREASING IMPORTANCE OF HEART FAILURE

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Second, in the last two decades, a number of new therapeutic interventions were introduced for the management of heart failure. Many of these interventions not only improved patients' symptoms and quality of life but also prolonged survival of patients with heart failure and decreased rates of hospitalization and cardiovascular events. Angiotensin converting enzyme inhibitors (ACEI), beta adrenergic blockers, Angiotensin receptor blockers and spironolactone have now an established role in the routine management of heart failure patients. In addition, to these established drugs, new pharmacologic agents are under extensive investigation, which include neutral endopeptidase inhibitors (NEPI) (Candoxatrilat), combined NEPI and ACEI (Omipatrilat), endothelin receptors antagonists (Bosentan), natriuretic peptide (Nesiritide), tumor necrosis factor receptor antagonists (Etanercept), new classes of inotropic agents with a novel mode of action independent of the cyclic adenosine monophosphate (cAMP) pathway such as calcium sensitizers. These agents proved effective in improving the hemodynamics and effort tolerance in short term studies. The plethora of pharmacological agents combined with non-pharmacological interventions such as cardiac pacing, LV assist devices, surgery, gene and immunotherapy make the management of heart failure patients something more than simple digitalis, diuretics and bed rest.

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M. Mohsen Ibrahim M.D.
Prof & Chairman, Department of Cardiovascular Medicine-Cairo University
President of The Egyptian Hypertension Society

SCIENTIFIC NEWS

Unraveling some of the etiopathogenic aspects in hypertension cleared that a difference in endothelium activity was detected between black and white population and was suggested to play a major role in the clinical differences observed among them and the therapeutic outcome specially in relevance to the different antihypertensives available..

Novel therapies to treat cardiovascular diseases as hypertension are aimed at striking the critical steps in vascular disease progression. This includes reversing endothelial cell dysfunction, correcting dysregulated cell growth and Apoptosis ,modulating vascular phenotype, modifying mechano-transduction and reversing vascular remodeling.

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EDITORIAL

**INSULIN RESISTANCE AND
ESSENTIAL HYPERTENSION**
Mona Aboul-Seoud, MD
Endocrine Unit, Internal Medicine, Department,
Faculty of Medicine, University of Alexandria.

The past decades have witnessed a major surge of interest in the cardiovascular actions of insulin. On one hand, this interest has stemmed from epidemiological studies demonstrating an association between obesity, insulin resistance, and hypertension, which has been named the "insulin hypothesis of hypertension." On the other hand, this interest has been stimulated by experimental evidence suggesting that the vascular actions of insulin contribute to its essential role in promoting glucose uptake in skeletal muscles.

In such a domain, two tenets have emerged about how insulin may exert its cardiovascular actions. Thus, it is firmly established that acute insulin administration stimulates sympathetic nerve activity in both animals and humans. Also, increasing evidence clearly shows that insulin stimulates muscle blood flow, an effect that appears to be mediated, at least in part, by an endothelium-dependent mechanism.

In this respect, insulin is thought to stimulate endothelial NO production or may act directly to enhance hyperpolarization of vascular smooth muscle cell membranes via stimulation of their Na⁺-H⁺ exchanger and Na⁺-K⁺ ATPase, leading to the consequent closure of their voltage-gated Ca²⁺ channels. While glucose uptake may determine peripheral blood flow via stimulation of ATP-dependent ion pumps with consequent vasorelaxation.

Beyond this, a "third factor" may collectively contribute to both insulin resistance and endothelial dysfunction in cardiovascular disease. Candidates to this include; skeletal muscle fibre type and capillary density, distribution of adiposity and endogenous corticosteroid production.

The aforementioned can highlight how hyperinsulinemia that commonly associates hypertension in the metabolic syndrome-X, can perpetuate the functional and structural alteration in vessels characteristic to essential hypertension. This hyperinsulinemia is attributed to the presence of decreased insulin sensitivity, or insulin resistance, with consequent compensatory insulin secretion.

When the hypothesis of decreased insulin clearance present in hypertensive subjects and its contribution to hyperinsulinemia [independent of the degree of insulin resistance] was tested; it was found that essential hypertension is independently associated with decreased insulin metabolic clearance rate in addition to insulin resistance. Thus, a low insulin metabolic clearance rate may be a contributory factor to the hyperinsulinemia observed in essential hypertension.

The pathophysiological mechanisms linking hyperinsulinemia to hypertension are varied. Insulin might increase blood pressure through sympathetic nervous system stimulation and enhancement of renal sodium absorption. Evidence exists linking both of these mechanisms to hypertension. Also, insulin is independently associated with myocardial infarction and microalbuminuria, two long-term complications of high blood pressure. While experimentally induced decreases in insulin resistance and hyperinsulinemia have been associated with decreased blood pressure. Moreover, normotensive offspring of hypertensive parents are also, as a group, insulin resistant and hyperinsulinemic.

The relationship between hyperinsulinemia, insulin resistance and hypertension is more marked in the obese, yet is present in lean hypertensive as well. However this relationship is not present in secondary forms of hypertension and may persist despite adequate antihypertensive therapy.

When the impact of therapeutic agents on insulin resistance were raised, their appeared that treatment of essential hypertension with beta-blockers and diuretics has been associated with increased risk of developing diabetes mellitus in three prospective cohort studies. While prospective, randomized studies with antihypertensive drugs have demonstrated differences between different classes of drugs regarding effects on insulin sensitivity. Accordingly, treatment with beta-blockers or diuretics is associated with impairment in insulin sensitivity, whereas most modern calcium channel blockers and Angiotensin converting enzyme (ACE) inhibitors are neutral. However, still to hold, there are exceptions within the different classes. For instance, Captopril seems to differ from other ACE inhibitors and results in improvement of insulin sensitivity yet the most pronounced improvements have been obtained with alpha1 blockers.

Despite of the raised therapeutic control, new concepts in therapy of insulin resistance are endlessly added, realizing how much this syndrome is rapidly expanding in industrialized countries, with its dramatic consequences on public health.

For instance, insulin sensitivity can be improved by non-pharmacological means as, the essential reduction of excessive body weight, the promotion of regular physical activity and the modification of dietary habits, as well as, the possibility of cessation of smoking and correction of subclinical magnesium deficiency. While the currently available pharmacological means should mainly include the biguanide compound metformin and possibly anti-obesity agents, such as dexfenfluramine, fluoxetine and benfluorex. New compounds aiming at improving the action of insulin, that are called 'insulin sensitizers' as the thiazolidinedione derivatives like troglitazone and pioglitazone are better added. By this therapeutic modality, hopefully the cardiovascular prognosis of numerous individuals having some or all components of the insulin resistance syndrome is improved.

In conclusion, evidence suggests that hyperinsulinemia and insulin resistance exert a pro-hypertensive effect that contributes to the pathogenesis of hypertension and hypertensive complications in some patients with essential hypertension. The associated complex interaction between endothelial dysfunction, abnormal skeletal muscle blood flow and reduced insulin-mediated glucose uptake observed, links between insulin resistance, blood pressure, impaired glucose tolerance and the risk of cardiovascular disease. An understanding of the primary mechanisms resulting in these phenotypes may reveal new targets to therapeutically strike when tempting to achieve appropriate control. While in those at high risk for diabetes mellitus, it may be justified to select drugs that improve insulin sensitivity when treating hypertension in insulin resistant individuals

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**ABSTRACTS OF WORLD LITERATURE
ECHOCARDIOGRAPHIC DEFINITION OF LEFT VENTRICULAR
HYPERTROPHY IN THE HYPERTENSIVE: WHICH METHOD OF
INDEXATION OF LEFT VENTRICULAR MASS?**

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Objectives: It has been suggested that hypertensives at high risk of cardiovascular complications can be identified on the basis of their left ventricular mass as determined echographically. However, there is as yet a lack of consensus on the mode of indexation (body surface area, height, height^{2.7}) of left ventricular mass (LVM), and on the cut-off values for definition of left ventricular hypertrophy (LVH). The main objective of this study is to test the influence of the different modes of indexation for LVM on the prevalence of LVH in a population of never treated hypertensive patients on the basis of cut-off for LVM based upon its relationship with ambulatory blood pressure (BP) measurement.

Methods: A population of 363 untreated hypertensives was investigated using a standardised procedure. The men and women were analyzed separately. We studied the relationship between mean daytime ambulatory systolic BP and LVM and calculated the LVM cut-off for a BP of 135 mm Hg using three different methods of indexation. On the basis of these criteria, the population was divided into those with and those without LVH.

Results: The prevalence of LVH was found to be higher when LVM was indexed to height^{2.7} (50.4%) or height (50.1%). Prevalence was lowest when LVM was indexed to body surface area (48.2%), which tended to minimize the hypertrophy in obese individuals. Only indexation by height^{2.7} fully compensates for relationships between height and ventricular mass in this population.

Conclusions: Indexing LVM to height^{2.7} thus appeared to give a more sensitive estimate of LVH by eliminating the influence of growth. Cut-off of 47 g/m^{2.7} in

women and 5 g/m².7 in men corresponded to a cardiovascular risk indicated by a daytime systolic BP 135 mm Hg.

J Hypertension 1999;13 (8): 559- 563.

TRIALS OF ANTIHYPERTENSIVE THERAPIES IN CHILDREN.

Wells TG

Divisions of Pediatric, Nephrology and Pediatric Clinical Pharmacology and Toxicology, University of Arkansas for Medical Sciences and the Arkansas Children's Hospital, Little Rock, Arkansas, USA.

Clinical trials assessing the safety, effectiveness and pharmacokinetics of new antihypertensive medications have been numerous as new classes of medications have been developed and brought to market over the past two decades. However, very few clinical trials have been initiated and completed in children with hypertension. Excluding diuretics, only one antihypertensive medication marketed within the past 20 years has any pediatric pharmacokinetic or dosing information published in the drug label and none have a pediatric indication. There are many reasons that these studies have not been done. Summation of the data collected in large epidemiologic studies that establish normal blood pressure and define hypertension using casual measurements have been a relatively recent event in pediatrics. Although ambulatory blood pressure measurement has been studied for the past decade there is still uncertainty with respect to the standardization of devices, measurement technique and normal values in a multi-racial pediatric population. As a result, no large scale, industry-sponsored clinical trials involving antihypertensive therapy have employed this measurement technique in children. In recognition of this problem, US Congress passed the Food and Drug Administration Modernization Act in 1997. Among the many provisions of this law, the US Food and Drug Administration (FDA) is required to publish a list of approved drugs for which additional information may prove beneficial for children. This law and subsequent action by the FDA also provides a mechanism by which manufacturers may gain six months of additional market exclusivity if adequate and well-controlled pediatric trials are completed and submitted to the FDA in response to a formal written request for these studies. Because such studies have not been previously undertaken and the new rules provide a significant financial incentive, written requests have been issued for pediatric studies involving more than a dozen antihypertensive agents. The FDA published a sample written request for oral antihypertensives in children and several potential study designs were presented.

Blood Press Monit 1999;4(3):189-192

ABSTRACTS OF LOCAL LITERATURE PRO INFLAMMATORY CYTOKINES IN ISCHEMIC HEART DISEASE: EFFECT OF CONVERTING ENZYME INHIBITORS.

Saraa Abd EI-Sha.fee, Ahmed Abd El-Aziz,, Abd EI-Hameed Ibrahim, Abeer Mohamed, Sobhy Said, Sherif Mokhtar.

Critical Care Medicine, Cairo University & Clinical Pharmacology, Helwan University.

Pro-inflammatory CYTOKINES are mediators released from leucocytes and vascular endothelium in response to various inflammatory and noxious stimuli, but have recently been reported also in myocardial ischemia and different grades of heart failure (HF). The exact pathogenic role and mechanism of production in HF and Ischaemic heart disease have not been clearly explained. Moreover the role of converting enzyme inhibition has not been elucidated in this work, we assessed the level of Cytokines and studied the effect of Angiotensin converting enzyme (ACE) inhibition on such mediators using Captopril (75 mg/day) and Benazepril 5-10 mg /day in 2 groups of pts with ischaemic heart disease (IHD). Group 1: comprised 16 pts with chronic myocardial ischemia (12 males, mean age 49 ± 15) all complicated by heart failure (class II NYHA). Group II: comprised 20 pts (17 males, mean age 52 ± 10) with acute myocardial infarction complicated by left ventricular dysfunction (Killip's classification II). Following admission all patients had clinical and laboratory evaluation including serum estimation of Cytokines IL6 and IL8 measured by chemiluminescence immunometric assay technique. Converting enzyme inhibition was started in the absence of contraindication using Captopril and Benazepril for one week with repeated measurement of serum Cytokines. Compared to the group of pts with chronic IHD, those with acute myocardial infarction had significantly higher levels of interleukin 6 (180 vs 10 pg/ml) and interleukin 8 (400 vs 20 pg/ml), respectively prior to treatment with ACE inhibitors. Following ACE therapy there was significant reduction in interleukin IL6 (from 180 to 20 pg/ml) and IL8 (from 400 to 90 pg/ml) in group II. We concluded that pro-inflammatory Cytokines are elevated in acute MI apparently as a response to the acute Ischaemic insult rather than being related to heart failure. More important, converting enzyme inhibitors were shown for the first time to significantly reduce the pro-inflammatory Cytokines in acute myocardial infarction a further addition to their cardiac protective action. Presented at the 3rd Scientific meeting of the Egyptian Hypertension Society, Port Said, Egypt. December 1998.

BETA ADRENOCEPTOR MEDIATED VASORELAXATION OF RABBIT AORTA IS ENDOTHELIUM-DEPENDENT: ROLE OF PROSTAGLANDINS AND SODIUM PUMP

Hassan Heialy Abo Rahma

Department of Pharmacology, Assiut Faculty of Medicine, Assiut, Egypt

B-adrenoceptor mediated vasorelaxation is thought to be through activation of adenylate cyclase within the smooth muscles. Such a mechanism is not dependent on the presence of the intact endothelium. There are now several findings suggesting that the B-adrenoceptor induced vasodilatation is endothelium dependent. However, others have shown that isoprenaline-induced relaxations in rat aorta are not endothelium dependent. Moreover, there is accumulated evidence showing that hyperpolarization through opening of K-ATP channels partly mediates vasodilatation induced by B-adrenoceptors. Recently, it was reported also that nitric oxide mediated Na⁺ pump as well as cyclooxygenase activation are also involved. The present study was designed to evaluate the role of endothelium in B-adrenoceptor mediated vasorelaxation in isolated rabbit aortic rings by using the selective B₂ agonist, terbutaline and to investigate the possible involvement of other mediators e.g. prostaglandins and other cellular components e.g. Na⁺-K⁺ ATPase (sodium pump) in the

B-adrenoceptor mediated vasorelaxation. The results of the present work indicate that the removal of the endothelium reduces significantly the relaxant effect of terbutalin. Propranolol treatment reduced significantly ($p < 0.05$) the terbutaline induced vasorelaxation & shifted its cumulative dose-response curve to the right. Terbutaline concentration response curves were obtained after treatment of the aortic rings with the prostaglandin synthesis inhibitors, dexamethasone and aspirin. The results showed that the terbutaline induced vasorelaxation was significantly augmented ($p < 0.01$) in presence of dexamethasone. This means that not only the prostaglandins are involved but also other substances e.g. adenosine, leukotrienes, etc. may be involved and the net result depends on the proportion of the vasodilator and the vasoconstrictor agents that may be affected by the cyclooxygenase and the phospholipase A2 inhibitors. Pretreatment of the aortic rings with the sodium pump inhibitor, digoxin, produced a significant reduction ($p < 0.01$) of the terbutaline induced vasorelaxation: pretreatment with dexamethasone, aspirin and digoxin didn't affect significantly the endothelium independent sodium nitroprusside induced vasodilatation. In conclusion, B-adrenoceptor vasorelaxation is mediated partly by NO release from the endothelium. The results of the present work suggests also the involvement of other mediators e.g. prostaglandins and the sarcolemmal sodium pump in the B-adrenoceptor mediated vasorelaxation of the rabbit aorta

Presented at the Joint International Conference of Egyptian Society of Pharmacology & Experimental Therapeutics, the Union of African Societies of Pharmacology & the Arab Union of Pharmacology. Cairo, Egypt, December 1999

CHALLENGE YOUR SELF !!!

A 73-years old man with known hypertension for many years was admitted for evaluation of progressive exertional dyspnea that developed over several days and for the sudden onset of severe dyspnea. He denied recent chest pain but did note that 1 year earlier he had experienced several episodes of nonspecific chest pain. He underwent coronary arteriography at that time, which demonstrated normal coronary arteries. He had no history of diabetes and did not smoke.

Physical Examination: Vital signs; pulse; 100; BP 220/120. General: severe dyspnea. Neck: no venous distension. Chest: crackles throughout all lung fields. Cardiac: no S3 or murmur. Laboratory investigation; Cardiac enzymes: normal. Chest radiography: pulmonary edema with normal heart size. EKG: normal. Echocardiogram and Doppler study: normal chamber sizes with some left ventricular hypertrophy and an ejection fraction of 60%; mitral flow revealed a reduction of the F to A ratio.

Question: What is the cause of this patient's acute pulmonary oedema?

Pick up the solution at CARDIOLOGY PEARLS

on p. [7] of this issue. PRACTICAL CONSIDERATIONS:

HYPERTENSIVE WOMEN

When your patient happens to be a woman please;

→ Concentrate on the ways to make this patient day by day live healthier and feel better and emphasize that it's a task every woman can do.

à Explain the dimension of the problem first and clear that three of every four women with high blood pressure know they have it, yet fewer than one in three are controlling it.

à Encourage overweight patients to a gradual loss of weight either by a lower calorie intake or increased physical activity and preferably both... Advice from a registered dietitian, or a qualified nutritionist to plan for a sensible, balanced eating pattern to lose weight slowly should be encouraged.

à Explain the importance of physical activity for the heart and blood vessels and for maintaining optimal weight to look and feel better. Luckily, this can be achieved without having to run marathons. Just a 30 minutes of moderate activity would be a brisk walking, gardening, cycling, swimming... .Breaking this into periods of at least 10 minutes, helps one to get started. If the issue that your patient is pressed for time, just make it clear that physical activity doesn't need that much time for a reward and a sense of accomplishment.

à Explain that women, should have no more than 2,400 milligrams of sodium a day (about 1 teaspoon) and clear, that this amount includes all of the salt and sodium consumed in processed foods, added during cooking, and used at the table. The importance of this hold of salt, is equal, whether there is high blood pressure, high-normal blood pressure, or to prevent high blood pressure.

à When you prescribe an antihypertensive, be sure that your patient understands the instructions and secure that if something is not clearly understood, she should call back and ask. Explain that, as with all drugs, side effects like; sleepiness, being tired, rash or cough etc, could happen. So it is important that she should pay attention to how she feels and notify any change that issues.

--□ Guidelines to which antihypertensives allowed during pregnancy, lactation, with contraception or better avoided in special health problems will be highlighted in the forthcoming editions.

High Blood Pressure Information for the General Public
: NHLBJ Home Page A collaboration of the Alliance for Aging Research,
National Heart, Lung, and Blood Institute, National
Institutes of Health & Sponsored by Hoechst Marion
Roussel, Inc.

ENVIRONMENTAL HAZARDS: CIGARETTE SMOKING: A GLOBAL LOOK

Many epidemiological and experimental studies has recognized and established the links between cigarette smoking and its cardiovascular hazardous impacts on morbidity & mortality.

Speaking of its relevance to hypertension per se, it was reported that smoking a cigarette raises the blood pressure by 5-10 mm Hg for about 30 minutes and lowers coronary reserve for 20 minutes. This aggravates and lasts longer if this is combined with drinking a cup of coffee.

Despite of this, numerous epidemiological studies have found that people with hypertension are not more likely to be smokers than those with normal blood pressure, and conversely that smokers are not more likely to be hypertensive than non-smokers. One possible explanation for this, might be, that smokers tend to weigh less than non-smokers, and that the effects of obesity and smoking on blood pressure cancel each other out. But even when smokers and nonsmokers of the same body weight are compared, their blood pressures are the same. This is probably because the rise of daytime pressure, after one smokes a pack a day, will raise your average pressure by about 5 mm Hg, a value that might not be detected during an office visit to the physician.

However, the important thing about smoking, is not what it does solely to your blood pressure, [the mechanisms through which remains to be clarified] but is centered more to how it greatly increases the vascular and cardiac risks, the extent of end organ damage and the likelihood of co-morbid conditions. These issues then, will even weigh more.

Moreover, breathing sidestream smoke [passive smoking] is also partially associated with the same detrimental effects as active smoking. To imagine how figures could be striking, the U.S. Environmental Protection Agency estimated that in USA 30,000 to 60,000 cardiac deaths are associated with passive smoke exposure. The question that will pose itself at this juncture is: Will these risks go down, if one quit smoking? The answer will be a big ' YES". This statement is based on the findings of several studies clearing that if you smoke, your risk of heart disease is about three times higher than if you don't. If you quit, it will go down to twice the normal risk in two years, and after that, it will be the same as if you had never smoked.

Realizing all this, calls on the need to really under stand why and how cigarette smoking does all this.

So let us know first, what are some of the noxious chemicals in tobacco smoke???

It was reported that tar, phenol, benzopyrene, nirtosamine and polycyclic aromatic hydrocarbons are all carcinogenic and some are pro-oxidants thus perpetuating lipid peroxidation, endothelial disruption and impairing NO vasodilatory potentials, which are all relevant to hypertension and comorbid atherosclerotic changes. While formaldehyde and oxides of nitrogen are severe bronchial irritants and toxic to epithelial, endothelial,.. etc structures. In addition, carbon monoxide impairs oxygen transport and utilization and shares in a pattern of Ischaemic myocardial insults that may also be linked to microvascular ischaemias in hypertensive heart disease. Lastly nicotine the known ganglion stimulant and depressant, beyond being carcinogenic, it sets the sympathetic drive of blood pressure regulation, at a higher regulatory setpoint.

The impact of some of such hazardous products and there relevance to vascular and cardiac functional and structural integrity will be more detailed in the forthcoming editions.

Br Med J 1994; 309: 901

JAMA 1995; 273: 1047.

N Engl J Med 1996; 334: 1189.

J Hum Hypertens 1996 Feb;10 Suppl 2:S13-6

**Basic Pathology. W.B . Saunders Company
Publications1997; 8: 221.**

PATIENT'S TIPS:

Help your patients stop smoking by initiating therapy with the antihypertensive mecamylamine HCl [Inversine] 10 days before their quit day starting by 2.5 mg twice daily for five days & going up to 5 mg twice a day. Mecamylamine reduces nicotine craving because it blocks ganglionic receptors. So tell the patient to stop smoking completely & start wearing the nicotine patch. By this you can strike a double benefit ; cessation of smoking & improving the therapeutic outcome of any antihypertensive intended to be used.

Modern Medicine 1998; 15 : 32.

CARDIOLOGY PEARLS

1. Diagnosis: Pulmonary oedema secondary to hypertension with diastolic dysfunction.
2. One-third of patients with signs of congestive heart failure have diastolic dysfunction and normal systolic function.
3. Diastolic dysfunction should be suspected in a patient with signs and symptoms of congestive heart failure who has normal-sized heart and normal ejection fraction.
4. The differentiation of systolic versus diastolic dysfunction is important, as therapy is different for these two causes of heart failure. Inotropic agents and arterial vasodilators used for unloading therapy may be harmful in diastolic dysfunction

NATIONAL & INTERNATIONAL RECOGNITION:

- Prof Dr. Mohsen Ibrahim, Head of Dept. Cardiology, Faculty of Medicine, Cairo University and the President of the Society, has been chosen a member of the Editorial Board for the Journal of Hypertension, for a term of three years. This journal happens to be the official Journal of the International Society of Hypertension (ISH) and the European Society of Hypertension (ESH).
- Prof. Dr. Ebtihag A Hamdi, Prof in Cardiology Unit, Faculty of Medicine, Alexandria University and a senior member of the editing board of this News Letter has been elected as a member of the High Blood Pressure Council, the Scientific Council of the American Heart Association, starting from 1st June 1999 with a membership ID 000106559901

EHS News & Calendar

EHS NEWS:

- The EHS has held its annual social event of getting together in Ramadan at Cairo Sheraton Hotel on Friday the 24th of this month. The members enjoyed the splendid iftar and the whole heartily gathering in this holy month . Prof Dr.

Mokhtar Gomma clarified the final arrangements and theme of organization he has adopted in the forthcoming annual congress of the society next January. Also, Prof Dr. Soliman Gareeb highlighted the preliminary data pooled from the scientific committee of the Egyptian Hypertension Project that is conducted to test the efficacy and tolerability of anti-hypertensives among Egyptian hypertensive population. A short statement was addressed by Prof Dr. Omnia Nayel on the current status of the news letter of the society and the topics that has been added to the issues. Lastly Prof Dr. Mohsen Ibrahim delivered a small lecture to delineate the criteria that should be fulfilled when probing the utility of an antihypertensive agent He also handed out a questionnaire form termed " Physician Hypertension Survey" to investigate Egyptian physician's knowledge and attitudes regarding hypertension.

CALENDAR:

LOCAL MEETINGS

\$th Meeting of the Egyptian Society of Atherosclerosis	Mena-House Oberoi,Cairo Egypt. January 21-23, 2000.	Prof Dr. Osama Abdel Aziz Tel: (202) 3926650, Telefax: (202) 3602800 / 3958000
The fourth Scientific Meeting of the EHS	Marriot Hotel, Cairo Egypt January 28-29th 2000	Prof Dr. Mokhtar Gomma Tel: (202) 3026871 -Fax (202) 3026871 E- mail : mogomaa@idscl.gov.eg
7th Annual Meeting of Egyptian Society of Cardiothoracic Surgery	Sheraton El Gizzera Hotel Cairo,Egypt, February,9-11,2000	Prof Dr.Shabaan Abul-Ela Tel : (2050) 374111.Fax (2050) 360138 E- mail : SHAABANABUELA@netscape.net
The 27th Annual Congress of the Egyptian Society of Cardiology	Marrior Hotel ,Cairo Egypt February 21st - 25th ,2000.	Prof Dr.Adel Imam Tel (202) 3489383-Fax (202) 3489383 E- mail : emam@internetegypt.com

INTERNATIONAL MEETINGS

4th World Congress of Echo-cardiography & vascular Ultrasound.	Mena-House Oberoi,Cairo. Egypt. January 19-2 1, 2000.	Prof Dr. Osama Abdel Aziz Tel : (202) 3926650, Telefax: (202) 3602800 / 3958000
3rd Conference of the Pan -Arab Hypertension Society	Abu Dhabi, 5-9 February, 2000 Intercontinental Hotel	Conference Secretarial Office: Tel:+ 97 1(2) 347478-Fax 349225 E-Mail: yassinl@emirates.net.ae
9th International	Salvador, Bahia,	Congress Secretariat; JZ

Congress on
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Adherence to evidence-based interventions for hospitalized cardiovascular disease patients is not optimal. This study assesses the impact of a national quality improvement program on adherence to guidelines in these patients. Data from 92 hospitals from across the United States participating in the Get With The Guidelines program for at least 1 year for 11 acute and secondary prevention measures from a preintervention baseline period and the subsequent 4 quarters of a quality improvement intervention were analyzed. A patient group of 45,988 patients with acute myocardial infarction, unstable a