

The 1st South Asian American Health Conference
General Session

Dr. Rohit Arora

Good morning. I am delighted to be here this morning and I also wish to congratulate the sponsors of this outstanding symposium, particularly Celia and Anu and their team and for being so dedicated and tenacious and finally bringing this whole program to fruition. And as I said yesterday, hopefully we need to echo this program or symposium in Chicago later this year. You're invited to that too. My avocation this morning is really to focus on something which is germane to me as a cardiologist and to Dr. Sethi and to all of us who see cardiovascular patients in the United States. And I am trying and attempting to highlight this in the American Heart Association and the American College of Cardiology. For now I'll just show the statistics. If you look back at the registries of the American Heart Association and the American College of Cardiology are two preeminent bodies in cardiology. You have no facts and figures applicable to the Asian Indian. So most of the data that we have we recourse to media search from, not India, but outside of India, but not within the United States. So you can look at the [epidemic] which is what I call this. This is one [one billion] people in the subcontinent. The incidence, as far as the prevalence, which is clearly unknown. The incidence as for mortality is huge. It is projected in the year 2003, that ultimately cardiovascular disease was responsible for death in about 16.7 million people in India. If you look at that's directly related to ischemic heart disease. As we best know, the incidence seems to be the order of magnitude of about 7 million of such patients. And most of these deaths occur in the developing country, rather than the developed country. And one-fourth of these deaths are particularly germane to South Asia.

If you see the evolutionary trends in Western civilization there is similar recapitulation to the South Asian continent. It took us almost 2.5 million years to arrive this phase of metabolic syndrome, which will be well discussed in this lecture and the future lectures this morning and afternoon. And this is the enemy within which is contributing to the pandemic that we know in the United States and in the subcontinent. So what is the data telling us as far as the high prevalence of CAD in South Asia? And

peculiarly, most of the studies, as I alluded to, have been published outside the subcontinent. If you look at the one important study which came out from India, and that was a [] Urban Population Study, is the incidence is really in younger folks about 11%, as you see, and above the age of 40 the incidence goes up by tenfold in these patients. The SHARE study, which was done in Canada from [Macmaster university] group from MacMaster University, they have shown that distribution of CAD in migrant Indians to be the order of magnitude about ten percent, as opposed to similar populations, for example, Europeans in Canada, the incidence is almost half – 4.9 percent and 1.9 percent in Chinese. And if you look at analysis, which Dr. Jay alluded to, in Canada there is, in 1.2 million deaths, most of the deaths, about 40% of those, occur in South Asian Indians. And so clearly there is this call to arms that Dr. Jay mentioned, that we should take seriously as far as cardiovascular disease is concerned.

So not only do we have a higher prevalence of coronary artery disease and cardiovascular disease, we also have a bigger price to pay. So if you look at the distribution in these studies as far as the death rate, not only the incidence, the death rate seems to be higher as opposed to some other populations. The Singapore Collaborative Study, as seen in the middle of this slide, also showed that the risk of death is threefold higher as opposed to patients of Chinese origin in Singapore as far as cardiovascular disease is concerned. And so if we look at the share of global mortality that seems to be inordinately high, in the order magnitude, so we have this dubious distinction of contributing, perhaps, to the highest mortality worldwide. And that too, from cardiovascular disease in the subcontinent. So these are some of the incidences that we know from contemporaneous databases in the published literature. And as you see, the incidence is about 10.7 or so, and if you see, the death rate seems to be inordinately higher, about 10 percent higher, in most of the South Asian populations. And if you compare in the demographics, if you just focus on the white bar and these patients, that you see most of the time in here, we are comparative as far as the death rate and the incidence to the Chinese, where I believe is the next pandemic of cardiovascular disease occurring.

And if you look at the prevalence in the same distribution as far as any type of cardiovascular disease that too seems to be inordinately high. And in this study that you

see the distribution of South Asians seems to be the highest as opposed to any other population and including those patients with a history of MI, PTCA, bypass surgery, angina or a history of stroke in these patients. Not only do we pay a higher price, we get cardiovascular disease much earlier on. And coronary artery disease starts regularly and occurs very early and the price we pay is much earlier in these patients. If you look at some of the data from the WHO as far as the proportion of death occurring below the age of 70 years in the developed countries it was the order of magnitude about 26% and if you see, in India that incidence was in the order 52%. One of the largest studies which has been recently published by [Salem Ysefs] group, the INTER-HEART study which really looked at the correlation of risk factors after having a heart attack. And in the Inter-Heart study it was found that South Asians, at an average, had their heart attack at the age of 52, as compared to the age of 62 in Europeans and 63 for the Chinese. So when our heart attacks do occur they occur very early. So this is the impossible age. So the moral of this story, when you blow that candle at the age of 53 and if you're of Indian origin you should breath a sigh of relief.

Now just to give you some statistics from the American Heart Association, and Dr. Jay talked about lifetime events. And this will get you to the heart. And the highest chance of getting a heart attack in the American population from the stroke and heart attacks in the year 2005, is about 63 in the United States. The highest chance of developing heart failure seems to be about 64. And the highest chance of developing a stroke seems to be about 67. So as an American if you blow the candle or you're lucky to pass to the age of 68 you should again breath a sigh of relief. And in our lifetimes it's an inescapable fact of civilization that 90% of us, if we live in the United States, will eventually develop hypertension. And Dr. Jay mentioned, it's almost a 40-50 chance that we will develop diabetes and if we are of South Asian origin there is more a 50-60 percent chance that we will develop diabetes mellitus in these patients.

Now the Inter-Heart study is a remarkable study, perhaps the largest database of different ethnic groups. And I want to share some data from this magnificent study. As you see, it was the large known studies in cardiovascular disease – a total distribution of about 25,000 patients in 52 countries across the world, including India and China. It was really a cross control study, a longitudinal study. And the association of a first heart

attack, that is these patients who have a heart attack, a documented heart attack, they were looked at as far as the relationship to other risk factors and there were nine risk factors looked at. And this was Citation which published in the Lancet 2004 in the November issue. The risk factors that they looked at were smoking, diabetes, physical activity, the lipid profile, obesity, alcohol consumption, hypertension, diet and psychosocial factors. The follow up was over four years of these patients from 1999 to the year 2003 in these patients.

And you can look at the first risk factor, the most important risk factor in this study, that is those patients who suffered a heart attack, the highest association was reapply in the APO-3 and APO-1 ratio, which is really a surrogate for your good cholesterol and bad cholesterol versus good cholesterol, LDL versus HDL ratio. And if you see the increase, there's an exponential increase to that in as far as the odds ratio for the first MI, versus those patients who had a high LDL/HDL ratio. And if you look at other risk factors, and what stands out in the Inter-Heart study in the South Asians, as you see here, to the right, the line of neutrality, that there is almost a five to six times higher chance that you will develop a heart attack if you have hypertension and if you're of South Asian origin. The risk factor is not known to us. We really do not know the incidence of hypertension. We don't even know the prevalence of hypertensions in the 1 billion such patients that we have in the South Asian subcontinent.

If you look at multiple risk factors, and this is applicable to South Asians in the [] study, whereas after you go beyond two risk factors you really are in more trouble. The most important risk factors which have been identified besides the APO3/APO1 ratio, next to that is smoking in these patients. And if you have these two your risk goes up four times higher and if you have more of these risk factors then your risk goes up five times to almost 10-12 times in these patients.

So the clinical implication of this large study, what does this tell us? That there were these nine simple modifiable factors which are applicable for cardiovascular disease and that account for most of the population attributable risk in 90% of the patients who suffer a heart attack and smoking and APOB1 ratio seems to be most prevalent as far as endangering patients to get a heart attack. And the distribution in the Inter-Heart study as far as the age of the first MI, as I was alluding to earlier, in proportionate terms, is very

apparent to us and it seems to be really [early] as opposed to anybody else as far as suffering of first heart attack.

So what about the risk factors? There are these conventional risk factors that we are well aware of. There are these unique risk factors and perhaps hyperinsulinemia and metabolic syndrome, which I'm sure Dr. Rghuwanshi will discuss more at length, but there are other factors which we don't know of. The change in dietary patterns and exercise patterns, the incidence of obesity and association of elevated CRP. And then the hypothesis of low birth weight and the association of this unique lipoprotein, the lipoprotein A levels. And it may require most of us to be on [a statin] which is the only drug which can alter this factor and then there are certain novel risk factors which have been identified. So looking at the conventional risk factors, in this SURE study South Asian the highest rates of diabetes, the highest total cholesterol and highest LDL and the lowest HDL. In the profile of patients coming from South Asia even without the presence of diabetes, the lipid profile is similar to the profile of a diabetic and that is associated with low normal LDL and their cholesterol is low normal but the HDL is remarkably low and they have a high incidence of the poisonous lipoprotein, lipoprotein A which leads to athero and is identified and invoked as the enemy within, as far as causing directly heart attack and stroke and causing plaque instability in these patients. Rist and colleagues also showed that the prevalence of diabetes not only in Indians but in Pakistanis seem to be much higher. And in talking about South Asians, colleagues in Pakistan and Sri Lanka, also seem to be having a preponderance of diabetes mellitus and other risk factors that we know of. And in England, Dr. Jay pointed out that the study by Whincup and colleagues, a remarkable study. And it's telling us that this is not environmental problem. And what Whincup showed essentially was that in South Asian kids compared to European kids, or British kids, if you look at their levels of glucose or fasting glucose levels they seem to be equal and normal. Not statistically different from the British kids. However, when you look at their insulin levels they seem to be inordinately higher, very reflective of a very early onset of insulin resistance syndrome. And we equate the insulin resistance syndrome and the clinical format of that, the metabolic syndrome, to cardiovascular disease. And if you have metabolic syndrome, even without diabetes mellitus your risk of

getting a heart attack is three times higher and your risk of getting a stroke is almost five times higher as seen in some of the data from San Antonio.

Now what is hyperinsulinemia resistance? Essentially the proclivity to produce amounts of insulin and the amounts of insulin are normal but you have a subnormal biological response in these patients. So as I was talking to you earlier about Lincock and colleagues, there are other studies which have also substantiated that indeed in South Asians there is a very early onset of the insulin resistant syndrome. And when you talk about the prevalence of diabetes, and this is from the ABA, there is no distribution or incidence that we know of. And if we want to project the analysis for somewhere here, as far as the incidence in South Asian, if you were to compute our data as far as the ethnic distribution of diabetes mellitus in the United States of South Asian Indians.

So the insulin resistance syndrome is a difficult physiological situation to have in the battle, because it leads to multifactorial, pleiotropic dysfunctional effects to the endothelial level, which translate into essentially what we call the atherosclerosis that is endothelial dysfunction. It leads to more clotting manifestations so therefore more chances of a thrombotic event, and if you look at the vascular biology of patients with insulin resistance you have more of proliferation of muscle, more lipid synthesis and obviously therefore more increased atherosclerosis in these patients. And the metabolic syndrome, and the problem with the traditional definitions of metabolic syndrome, the National Institute of Health Blood Institute has this definition for metabolic syndrome, applicable to the United States and Western Civilization. You have to be obese with a waist circumference of 102 centimeter in men and 88 centimeters in women. Your triglycerides should be more than 150 and your good cholesterol should be less than 40 in men and 50 in women. Blood pressure more than 135/85 and the plasma glucose more than 110 in these patients. So the constellation of the syndrome requires if you have at least three of these five risk factors to really define yourself as somebody with metabolic syndrome.

Now the WHO, which more of a non-American distribution, defines metabolic syndrome differently and their definition is a definition of hyperinsulinemia where fasting glucose is more than 110 and your glucose two hours after glucose load is more than 200, plus two of the following – abdominal obesity, dyslipidemia or blood pressure in the syndrome. And why this is important, is because again, where do South Asians fit

in as far as these two traditional definitions are concerned? The definition is different. The prevalence is different and the definition you should apply to South Asians, in my opinion, should be different. Because we have some components of the metabolic syndrome which are at a higher incidence than the general incidence in patients with metabolic syndrome. And if you look at the prevalence, the projected prevalence of metabolic syndrome, which is really cardiovascular disease in clinical terms, in South Asians it is the order of magnitude about 21 to 28 percent. So what is the trouble as far as this is concerned? The trouble is, obesity in South Asians is kind of different. We tend to have, as opposed to more Europeans, more accumulation of abdominal fat. And as shall be discussed later in risks, abdominal fat, the [visceral] in the abdominal fat seem to be more harmful to the vascular system because we now know that they release more cytokines and inflammatory agents which damage the endothelium than opposed to the abdominal obesity is concerned.

So there was just a recent, I would say about three years ago, a Western Pacific Region Conference by the WHO. They have redefined obesity for Asians, particularly South Asian. And they are advocating that the BMI values, because we are a little smaller, our values should be the order of magnitude of 22-25. So when we apply this, the incidence of cardiovascular disease, metabolic syndrome, is going to be about 50-60 percent in the South Asian patient population. And the changes in dietary and exercise habits. We also love [to eat] and Dr. Jay discussed this environment of plenty, the sedentary lifestyle which we all somehow come to when we come with the migratory stress that occurs in South Asians immigration. So as I was talking about the obesity phenomenon, this is a Texan phenomenon way down in the South, but more applicable to us is a different kind of obesity, and it is the abdominal obesity which I want to discuss with you. Because associated with central obesity and associated with that is very clearly shown one of the gold standards used for cytokines or inflammation, which is what vascular disease is all about, that is the highly sensitive CRP. CRP levels seem to be much higher in patients who have abdominal obesity. If you look at the literature there is no real sort of demographic available to see the general incidence of this inflammatory marker, HS-CRP, in South Asians that we know of. CRP is dangerous because it is and behaves like a cytokines and it causes more atherosclerosis and leads to instability of the

atherosclerosis and leads to a pro-inflammatory state and leads to complement activation. And this is the real McCoy, because the definitions are important for us to be included in the category of diabetes and metabolic syndrome, and we talk about abdominal obesity, the tailors are usually the ones to first identify and intra-abdominal fat is the one. And most of this fat is really visceral fat. The ideal way to measure this is a CT scan of the abdomen, which has become, now, the gold standard for measuring abdominal obesity. And that begets elevated levels of CRP, which leads to more activation of LDL and leads to more atherosclerosis.

And then the phenomenon that Dr. Jay alluded to, this fetal origin hypothesis. And this hypothesis suggests that if you have fetal under nutrition in the middle and late gestational period, that is associated with a higher incidence of CAD later in life. And it's kind of a paradoxical phenomenon and it seems to be because of an adaptive response of the fetus to impaired nutrition. And these adaptive responses continue later in life and they can possibly contribute to the higher incidence of CAD in South Asian patients.

And then this lab reporting which identify – this is another enemy within, and that is that South Asians seem to have a higher level of genetically driven lipoprotein A and apolipoprotein, this is really a [problem]. It appears in the clotting phenomenon. And we have proclivity in our endothelial system to break down a clot by the fibrinolytic system, the endogenous phagolytic system, and that seems to be impaired by this lipoprotein, a fatty substance called LpA. And Enas, and Enas actually from Chicago, and Dr. Jay mentioned, also have a body of data to suggest, and it's not randomized data, but South Asians seem to have compared to a control population in the United States, a higher incidence of LpA. And the only therapy – statins may not do a good job as far as lowering this, that Lifespan may be the only thing which [works] as far as reduce both the primary risk and the secondary risk of cardiovascular disease.

So when the [hdl/ldl] study looked at the [cholesterol] ratio, which really is incorporating [total cholesterol], the ratio seems to also affect the same thing. This is data of [Interheart study], also the increasing incidence of association with [cholesterol]. The same thing that you have in the ratio of apolipoprotein B with [apolipoprotein].

And then we talk about other risk factors. We have a higher incidence, perhaps of novel risk factors, more than the traditional risk factors, which are the proton guided

markers, fibrinogen, Pyl, homosistine concentrations, as opposed to some other ethnic populations, including the Chinese and East Asian. So when you look at levels of TPU we seem to be higher as far as the TPU antigen is concerned in South Asians as compared to Europeans and since it's produced by the endothelium it may be [prothrombotic] to the endothelial function, an attribute to endothelial dysfunction in these patients.

And then preventive measures, which will be discussed more at length this afternoon, particularly dietary modification by friend and colleague, Wahida who is here from Columbia, And the other pharmacologic interventions beyond that, how we can proceed with modification of the risks for preventive measures in these patients who are at a higher risk. Obviously it's a population [base] system that has to be added – as far as educational components of reducing this risk. Obviously this needs to be a Federal and governmental instrument more than an individual instrument, as for advocacy and education of higher risk of cardiovascular disease. And mainstream media and the vernacular press also should have an important role in the educational process. I think the educational process needs to go down to the level of health care professions, which is, if you talk to several colleagues in the subcontinent in the health care professions, they also seem to have a poorer understanding of the risk. Not poor understanding of cardiovascular disease in South Asians, as we know of.

So the question is really whether it's gluttony or sloth or is it both that applies to us. And it is now coming down to both. And there's a lack of modification, the dietary modification, which Dr. Jay alluded to, because of misplaced notions what is good for us, and nontraditional alternatives are less available, like tofu, which is available here to really substitute for meals that we have in our diet in South Asia and that we should be talking about. And I think that the new American Heart Association pyramid, as far as the food pyramid is concerned, is well applicable to South Asians too. And we should include at the base complex carbohydrates, with fruit and vegetables in the middle five – five servings per day, and protein intake including fish as two to three servings per week. And then, exercise should not be what I understood as exercise, walking the dog. It should be more physical lifestyle modification. Dr. Jay showed a slide from the DPCT study. They showed that exercise was the most effective way of lowering the incidence of diabetes mellitus and there is, I'm sure that everyone should have it in the package, the

[] study. It's a remarkable study done in Amsterdam in a small country, of 200 patients. And they too showed that not only the incidence of diabetes, but in diabetics when they're give lifestyle modification, medical therapy, lifestyle modification actually increased survival by the order of magnitude of about 52% in these patients.

So there was a new paper last week which was also – the cheapest way of lowering risk, cardiovascular disease risk, seems to be exercise and we should advocate that as a global issue, but more so in South Asians and in the subcontinent, as we talked about. [earlie] stress control, meditation, yoga – as we migrate here we lose track of these agents available to us to reduce the risk. [the inte] Heart study, avoiding smoking, regularly exercising, eating fruit and vegetables, has been shown to reduce the relative risk of a heart attack by 80%. So if you have a heart attack to reduce your risk, we can reduce this with lifestyle modification by 80%. And this is data based on a study from [_____]. And when you look at the risk of the MI, with avoidance of all these factors, it is very intricately linked to each of these factors. As you abandon smoking, as you increase your exercise and [cad risk goes down]. As you see here on this curve, it seems that the incidence seems to become less and less. [END-TAPE 1]

I am now talking really of primary prevention more than secondary prevention, which should also be a major and aggressive goal. And Dr. Raghuwanshi will be talking more about this, that the TZDs are remarkable drugs which can be used and there's more emerging data, as you will see later, that it does restore the insulin sensitivity that we talked about and we have insulin resistance that has beneficial effects and acts on the LDL. In fact, converts the atherogenic LDL ratio to a not atherogenic ratio and by preventing oxidation will oxidize LDL which is atherogenic, and has been shown to improve endothelia dysfunction which is found in South Asians. The statins are remarkable drugs, beyond lipid control. They have pleotrophic effect. I call them magical drugs. And there is highly rich prevention data in the United States, both from the Air Force Texas study and on the Vasscoff study using respectively Lovostatin and provocore, that statins not only decrease levels of lipids but they improve endothelial function, they reduce inflammation and cause stabilization of plaque in [the artery]. So they should be part of our armamentarium. And this is a passion that I have, and this is one slide, if you slept through the whole lecture I would like you to wake up. And I

believe that it would tantamount to a violation of the Geneva Conventions, you see a lot of that going on in the press these days, but in the cardiovascular domain if any of our patients walks out of the office, clinic or hospital and is not on these therapies then that would be a violation of the Geneva Convention.

[Dr Salim Ysef] who is one of the masters of meta-analysis and he's based in McMaster University and has taught us most things that we know of from the ARC meta-analysis and the incidence, has done this outstanding meta-analysis of secondary prevention. I was alluding earlier to secondary/primary prevention and I want to go over this slide with you because suppose we have a patient who has known cardiovascular disease. My grandmother was [had] a heart attack and we follow her, for example. And if you don't give any treatment there's an 8% chance that she will have a heart attack, stroke, or die at two years. If you give an anti-platelet, and I call this the ABCD. Jerry Cohen, who wrote the paper in *Lancet* says ABC and modified it to the Ds, so I really have to give homage to Jerry about this. But if you look at use of anti-platelet, aspirin will reduce relative risk by 25%. This is secondary prevention. Aspirin is not good in all patients for primary prevention. Actually, the FDA this year did not approve that medication for that. But for primary prevention in anybody with known cardiovascular, previous cardiovascular disease, you cut down your risk of an event by relative risk of 25% and absolute risk of 2%. When you use a beta blocker in patients who have CAD, ischemia, hypertension, CHF, you have another 25% reduction of risk in these patients. When you add to that C – that was B, and C, that is cholesterol lowering therapy, you have another 30% or more, robust reduction in risk in these patients. So the absolute risk is driven back from 8% to 3%. And then on top, the other A, is the emerging data from the HOPE, PEACE and the ORACLE study has shown us that there is an ACE inhibitor, with tissue ACE inhibitors, you can get another 25% reduction in risk, so we're almost to one percent. And on top, if you combine this, you have an ABC of 75% reduction risk. This is a lot of good that you can do for pennies on your patients when they go out, more than aggressive therapy, for example, like intervention and bypass surgery. And if you then add preventive therapy of don't smoke, and I say don't die, then you have an 80% reduction of risk, if all therapy was done, if you add on smoking cessation. So this should be the mantra that should be talked about in our own cardiovascular – this the least that

we can do in patients who already have cardiovascular disease, as far as advocacy to reduce cardiovascular risk in patients.

And then, India and China are going hand in hand in sort of the Hippocratic oath I took, not the Hippocratic oath, but from Hypocrites – I believe one of the oldest known physicians, [Hun Dee] from China. And he said long time ago, “Superior doctors prevent the disease, mediocre doctors treat the disease before it is evident, and inferior doctors like myself treat the full blown disease.” And this is what we are doing here. But it is, I believe, the ingenuous doctors who should treat the disease before it is evident, so we should take all efforts for primary prevention in the high risk group of patients that we talked about this morning, and the emerging risk in South Asian area. So with this I’d like to end my talk, and I thank you very much for your time.

Dr. Sethi: I think we certainly have time, Dr. Arora has finished well within the time, and I think we’ll invite some questions, and there are two microphones on the two aisles. You’re welcome to walk to those microphones if you please. If somehow you don’t want to walk there, you can put it on the index card and I’ll read the question. While we are waiting for questions let me ask you a question. It is very apparent from this morning’s presentation as well as for the past presentations that being a South Asian Indian or being a South Asian in itself is a risk factor. So if that is a risk factor and if you now move South Asian as a risk factor into the ATP3 guidelines, would you be considering a much earlier treatment if you have one risk factor as a South Asian Indian or if you have two risk factors, South Asian and one of the other risk factors that you listed? And be more aggressive?

Dr. Arora: That’s a great question. I did want to take this opportunity to thank Virender Sethi for being here. As some of you know and some of you don’t, Dr. Sethi has been an institution in the state of New Jersey and one of the most outstanding [cardiologist]. When I need my stent I shall go to him from Chicago. And we are pleased to have him as a moderator and peripherally a friend I’m very proud of him, but he has singularly, over 25 or 30 years, busted more lesions in South Asians than anybody else

that I know of in the United States. So he has that distinction. So I'd like a clap of hands for him.

Dr. Sethi: Thank you for those kind words.

Dr. Arora: And in addition to that I also was fortunate to teach, to be the Director of Cardiology for his daughter, who is an outstanding cardiologist too. She's not here today. But in any case, the answer was how do you apply the South Asian information database and then patient management to the ATP3 revised guideline, as you know, which came out in June? If you are interested it's published in June's simultaneous issues of *Circulation* and the *JACC*. And these guidelines have become very stringent, as have the JAC7 guidelines. And to simply put, they have suggested that in the high risk patient population, which is anybody who has ever had heart disease or cardiovascular disease, either stroke, heart attack, peripheral vascular disease, abdominal aneurism, for example, in those patients, from the Framingham classification, that group falls in the highest risk of getting an event, which is the order of magnitude about 20% at ten years of follow up. Then there was an intermediate group which has a risk of 10% at ten years of follow up. And then there is this low risk group which has less than 10%, that is less than 1% per year risk. And where do South Asians fit in as far as the ATP3 NCPD guidelines is the question Dr. Sethi had. And believe it or not, and I agree with you, and the suggestion is common, that we will fall in the high risk group. We fall in the high group even if we do not have cardiovascular disease. So my advocacy in our patients who have no cardiovascular disease, who are of South Asian origin, would be to meet both of the goals of the high risk group, which is really getting LDL down to 70. And the five large scale randomized control studies, including the most latest, which was published in March, the TNT, have suggested to us that we now know what is the ceiling as far as the cut off, upper levels. The upper levels should be 70 in the high risk, intermediate risk about 100 or less and the low risk, under 130. This is not totally cholesterol, LDL, which is a primary goal of the ATP3 revised guidelines. So I think we should fall into that goal, which a high risk for ATP3 guidelines, and also, in congress, for management of hypertension we should be in the group which is high risk as far as renal failure and

congestive heart failure, where our blood pressure there should be 130 and 80 or less. Not 130 or less than 85 in patients who are of South Asian origin. And this is a speculative belief based on strong beliefs and data.

Q: Hi. Wahida Kamally from Columbia University. That was a wonderful talk. In continuation of what you just mentioned, you know, South Asians are known to have low small dense LDL particles.. So I think it becomes more relevant in treating elevation of triglycerides and low HDLs, because then you can make them into larger particles I mean it's correlated, the high triglycerides and low HDL are correlated with small dense LDL particles. The second part is, has there been any attempt to relegate the Framingham equation algorithm in South Asians?

Dr. Arora: Yes. It is good to see you this time in person. I usually see her on CNN, if you haven't seen her before. She's often on CNN, a contributor for nutritional aspects. And she had two questions, the second one is the Framingham, is the Framingham applying to South Asian populations. No they are not, in fact, Framingham is not – is only white Americans, predominantly males, and does not even have a coterie of African Americans or Hispanics, leave aside South Asians. The incidence of South Asians in the Framingham population is 0.3%, for your knowledge, as far as the total demographics. So we don't have the answer to that.

Then your first question was should we look more at lipoprotein A, the atherogenic particle, in our lipoprotein profile? Yes we should. This is based on data from the Quebec Heart Study, where they have shown that it's not the total amount of LDL or HDL, which is what the APT3 revised guidelines are advocating. It's really your atherogenic particle and we can measure that, as you all know, from various tests, as far as the profile of the lipids. There's dense particle versus the buoyant or fluffy particles. The high dense particles seem to be more atherogenic based on the Quebec Heart Study. The buoyant particles seem to have less atherogenic particles. So statins, magically, also can lower the atherogenic particles to the nonatherogenic particles. So in advocacy as far as primary prevention and secondary prevention, statins seem to do that very eminently and so I don't think we need to recourse to anything additively in this patient population.