ASSOCIATION FOR ERADICATION OF HEART ATTACK

Transcript of Dr. Eugene Braunwald's lecture at the 2nd Vulnerable Patient Symposium held by AEHA on March 6th in conjunction with the Annual Conference of American College of Cardiology 2004 New Orleans, LA

Dr. Naghavi, Dr. Shah, a pleasure to be here. I want to make a few comments about the way I've been thinking; it's not a unique way. But at least put together the way I've been thinking about identifying and managing coronary risk in 2004. These are basically principals.

So, the 1st is the prevention of acute events has to be the primary goal in cardiology. Treatment which we spend a lot of time on and I've certainly spent many years in treatment, and **treatment is locking the barn door after the horse is stolen**. We have to recognize that so prevention comes first and foremost.

And as you heard from Jim Willerson the magnitude of the problem is really immense, but what Jim didn't say that is a third of cases of sudden death and of acute MI occur in previously a symptomatic persons. And previously, until maybe 15 years ago, these were regarded as acts of God. No one knew where they came from. People were suddenly struck down, often in the prime of life. Now we know, and I think this is one of the great advances in medicine in the last quarter of the 20th century; we now know that these are not acts of God, but these individual usually have pre-clinical disease and or they have classical risk factors those that were identified by Framingham or what we now call novel risk factors. And the imperative is to identify high risk asymptomatic persons prospectively, ahead of time, to provide intensive prevention. All individuals who have clinically apparent atherosclerotic disease require intensive global risk factor reduction, that's a given, but that's secondary prevention. Some patients have unstable plaques, which I would call, accidents about to happen. And of course these have to be

identified. So this is my little pyramid. Everybody has - this is the fashionable thing to have. So, I'll show you my pyramid, if you show me yours! So, this is my little pyramid which takes patients at the base of the pyramid are individual who have no apparent risk factors. And I think people would say that you have less than 1 in 200 chances in having an acute event (adult, this is for adult people) a year that would be defined as low risk. That doesn't mean that that couldn't become intermediate risk somewhere in the future, but we can't concentrate on that group now. Intermediate risk now is 0.5 to 2% a year and I think a lot of the guidelines, the European society and Framingham have focused on the higher risk group the 2 to 15 and I've added a very-high-risk group the more than 15% per year group.

So, how to go about it? Now, these are principals, the details I think we can argue. We can argue about the details. So, the approach to the patient is some kind of clinical evaluation. And I don't want you to argue with me whether the Framingham risk score or whether the Munster risk score and there are a number of risk scores available or how good is the Framingham risk score in Africa or not, or some clinical risk score and for the moment grant me the Framingham. And some marker of inflammation, Professor Masery was the first to identify C reactive protein. There are others, and I think there still need some sorting out but let's begin with CRP. Let's look at cholesterol obviously with fractions as well as glucose and that gives us the opportunity to do a first cut. And with this first cut about 40% of American adults will turn out to be low risk defined as less than half percent a year, less than one event in 200 per year. And these are suggested to have life style recommendations and follow-up and that is the follow-up can depend on the age.

We have the intermediate group. 0.5 to 2% a year and that for American adults unfortunately is about 50%, even larger than the low risk group and these we think, I would suggest require additional testing. I'll give you some ideas. And then there's a high risk group more than 2% a year, that's about 10% of the population that soars with the numbers like Dr. Willerson showed. And for sure these need intensive global risk reduction.

So what is the additional testing? It would be non-invasive imaging in an effort to identify patients who have vulnerable plaques. And here are three methods. We've done the clinical and we have done obvious biochemical such as glucose, cholesterol, hemoglobin A1C, C-reactive protein and now we've identified a group who's at least at intermediate risk and we have a series of non-invasive imaging techniques or other tests. Actually, the ankle brachial index (ABI) is not an imaging technique but it does give us further information about vascular disease. EBCT and intimal medial thickening of the carotids. Those techniques are available right now. And in a patient who was

defined as to being a intermediate risk and if this further non-invasive imaging non invasive testing is negative then they descend back into low risk. But remember they've been identified to have some risk factors, they may have hypertension, isolated hypertension or isolated LDL elevation and they need risk factor reduction of a single risk factor.

If the non-invasive testing in the case they are high risk, then they need intensive global risk factor reduction and some non-invasive detection of unstable plaques. And the non-invasive detection of unstable plaques I think is the subject of this meeting or a major subject for this meeting. I won't preempt what the techniques are, but I think that this is the group in whom such non-invasive detection and if that's positive, then we're now getting into a very-high-risk group. These are the "accidents about to happen" and I think at the present time, we need to think about going after these plaques with some invasive detection device. Again, these will be discussed here so this is how you identify the patients who need the invasive detection and now we're getting close to the top of the pyramid and if we find them, then we are dealing with a population where the risk is in excess of 25% a year.

Now we come right smack up against of what do we do? I think that there is a lot of work going on currently on novel anti-inflammatory compounds because the basis of the unstable plaque the important role of inflammation, antithrombotic therapy at a high level and people are even talking about bypassing unstable plaques that have been identified with surgery or multiple drug-eluting stents. If on the other hand, the invasive detection is negative, then we would recommend continued intensive risk factor reduction. So, I think we're poised at a very important time. This is a very timely conference and I think it would be very interesting to see what comes of it. But this would be the general frame work that I would recommend that you to consider. END