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HEART DISEASE MANIFESTING IN ADULTS STARTING IN CHILDHOOD

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DR. BAKER: Our next speaker, Dr. Gordon Cumming, deserves a long introduction but really needs only a couple of words. All I want to say is that this is his third consecutive appearance at our meeting to talk to us. I talked to him a couple of years ago to present this lecture today. Ross McKenzie came after I did but got him to speak last year. Gordon said to me, "Do you really want me to come back for a third time?" And I said, "Gordon, you are the most popular, highly acclaimed speaker we have each year on our evaluations." So he is coming back. He has one more lecture that he has prepared, one more presentation and we want to welcome him again to this meeting. Gordon Cumming.

(Applause.)

DR. CUMMING: I'm really sorry to impose myself upon you. When I left GFT University position, we only got to travel in my university if we gave a paper and I never really realized up until a few days ago that the company would pay my way to these meetings, if I didn't have to give a paper. Anyway, it's also rather dreadful to try and follow that very dynamic speaker that we had before. I was surprised that Tufts had turned out two such great administrators, with Bill and with this current president of the AMA, but that was a great and dynamic talk.

I want to talk about, perhaps I might retitle by talk in part on how to get along without genetics. Heart disease does start, probably all forms of heart disease, in childhood.

I need a pointer and I need the first slide.

(Slides being shown.)

DR. CUMMING: This is related to coronary artery disease of men born in 1911 and 1930 based on birth weight and on their weight at one year. If you're a fatty to start with, or big, to start with and you stay big, you have about half the incidence of coronary disease as in the population. If you're small, and by small, here is five pounds, and you don't gain much weight and you're still only 18, 19 pounds at one year of age, you have 50 percent more coronary disease than the population.

Well, this doesn't mean we want to underwrite on the basis of birth weight, etcetera. The only point of bringing this up is that there are influences starting very early in life that do influence mortality and there's no real effect here to go on cause. We may have had smoking mothers, we have had low socio-economic class, all sorts of reasons other than health for this.

Let's get down to a few points in congenital lesions that are relatively minor that may cause problems. The first is the simple patent frame, with the clot sitting in the right atrium that's going through the ovale, and is going to cause a paradoxical embolus. It's now been shown that perhaps paradoxical embolus does occur and is responsible for the ordinary or cryptogenic stroke where there is no other reason, such as diabetes, carotid artery disease, hypertension, etcetera.

At autopsy, a PFO is there in 30 percent. By TE echo we can show it 25 percent. By pushing some aerated saline in and sampling in the carotid, we can find it in 25 percent, and in cryptogenic stroke 40 percent. Again, we're not going to underwrite them on the basis of a PFO. They obviously are standard risk. You're going to see the odd person like one recent hockey player with a small CVA who ended up having surgery on his PFO.

If we do a dopler study now and a sample from the pulmonary artery, you can find adductus in about half percent of normal people who can't hear. They can get endocarditis in this but really I don't think this should influence our underwriting in any way. I think these should be fairly forgotten about. Some of these people are getting plugs and things to close them. I don't think this is correct myself. This has been called a technomality.

Prior to antibiotics, however, there was .4, .5 percent per year endocarditis with adductus so these people are all going to be on prophylaxis if they happen to end up getting incidental echo to find this.

The simple VSD, the more we study these people, the more we realize there's rather complexities that are involved. This is a drawing of a 2D echo. In the very small member of the VSD, it's not uncommon to start seeing the tricuspid valve sort of shrouding over the defect. This has been called aneuresymal transformation. It's a mechanism of closure. You're going to see in the reports an aneurysm.

We think of aneurysms as being bad and I get the underwriters coming and saying this looks bad and this is the way a small VSD closes, and it's benign. On the other hand, again when we have ways of analyzing things, we get even more worried. The aneu-

rysmal formation involves both tricuspid valve and septal tissue and in five years the small defects that occurred in 74 percent.

Now in 11 percent of these, they developed what's called a left ventricle to right atrial shunt which has a higher velocity blood flow than a simple VSD and a higher rate of endocarditis. And in addition, when we start looking at detailed ultrasound, we start finding the sub-aorta ridges which may go on to sub-aorta stenosis.

This shows a defect going, here is the aneurysmal transformation and then there's the hole and the shunt goes right through here in the right atrium and it's less likely to get permanent closure after that develops.

Again I think we should look overall at the mortality in the small VSD, in people who have been followed 25 to 40 years, is normal. We should stop and not really worry too much about these nitty-gritty details perhaps as looking at the global picture. But the more you look at the fine reports, you're going to see things that perhaps frighten you a little bit.

The bicuspid aortic valve occurs in two percent of the population. It always has one cusp larger than the other and this large cusp usually has a median raphe in it. This has led to a change. This is not thought now to be congenital right at the formation of the aortic valve, but probably there were three leaflets and then there's been fusion of two of them.

Thirty percent of valves removed are bicuspid and it's probably not abnormal embryogenesis but it's an in-utero of separate cusps. You can have either anterior or posterior right and left. There's a difference between valves that get stenosis and valves that get regurgitation. The valve that gets stenosis has a lot of calcification. The valve that regurgitates starts prolapsing and has very little calcification.

We tend to want to, if we see a diagnosis of bicuspid aortic valve, to automatically go standard in our insurance underwriting. I think one of the problems is that some of my colleagues will label something a bicuspid valve and they forget to tell you that it's got a little stenosis and a little regurgitation or perhaps a lot of regurgitation.

You have to be sure you have a hemodynamically fairly normal valve if you're going to go standard mortality. This means no greater than ten, that you have very trivial regurgitation, you have no associated heart disease. If you haven't got a recent echo or any echo at all, the intensity of the murmur in a bicuspid valve is just grade one or two and a lot of the patients just have a click and no murmur at all.

So if they've got a grade three murmur, you're dealing with someone with some stenosis and that is not standard mortality. If you have more than a little bit a diastolic and if you've got a pulse pressure of 50 or 60 you don't have a trivial amount of regurgitation.

Mitral valve prolapse, of course, occurs in children. It starts in pediatrics. It's not there in the first six, seven years of life, although I've seen a few real dandy prolapses in babies and children one to two years of age, it's unusual.

We need theories of children with discovered mitral valve prolapse that are followed for 60 years, not seven years. But in this one pediatric study reported there was no progression, no death, no restriction of activity and only two cases out the theory ended up getting arrhythmia.

This isn't the entire picture. I've had several patients needing valve surgery for severe mitral regurgitation. They respond very well to a carpinchy ring. I've had one death of a patient with ventricular tac that unfortunately ended up getting her propanol stopped by a local physician and she was a sudden death.

Let's go on then to a subject we usually either a straight define or what's surprising is some of the manuals are now underwriting these as plus 100, 200 and this is hypertrophic cardiomyopathy which is the asymmetric hyperpathia in the thicken septum. We don't usually consider this very much in pediatrics because for underwriting the mortality rate is one that has discovered hypertrophic cardiomyopathy in childhood is six percent a year which is sort of like 10,000 mortality ratio.

But some of the things that you do get, we had one of these a few years ago. The client was 12, the father had died at 36 of hypertrophic and the child was to come into a little bit of an inheritance and the mother wanted some insurance.

Of course, genetic studies not available and will not be ordered, and where do you stand? Well, you can't order just a chest X-ray and cardiogram so you ask for an echo and it's normal. I wanted to point out that it doesn't help. At 11 this is an echo and a very borderline septum for a child, but it's about eight millimeters and then here, at 15, it's 28 millimeters.

The hypertrophy in children with hypertrophic cardiomyopathy often doesn't appear until 16 to 20 years of age. I've had a few families where I've reassured the parents of their 12 and 13-year-old child was normal and then unfortunately by 18 or 20 it became obvious. The kids weren't very appreciative because it took them out of hockey and one of the kids became a philosopher and he said, "The worst thing you can do is to screen anybody."

This is a paper from Maron that he had 16 patients at 11 with a ten millimeter wall and there was no change at 16 but in five of them, they had increased considerably and in three of them they became 18 to 28 millimeters, and two of them 15 millimeters. So a childhood echo does not rule out hypertrophic cardiomyopathy, very parallel to say, polycystic disease.

This is how thick a septum can get: a seven-year-old patient of mine with this huge 30 millimeter septum darn near filling the heart. This is the outflow tract to the left ventricle. This is a long

axis to the echo, the small left ventricle. This guy is now 27 years of age and still thriving. The surprising thing about hypertrophic cardiomyopathy is the severity does not predict mortality.

We have the genetics now of picking up of the mutation and this was an interesting echo study of ten families that's been published in the *Journal of the American College of Radiology* in the last year. We won't go into the genetics because we have Sandy here to explain the things that we don't understand. I don't want to really necessarily dwell on this but it does show some of the limitations of echo.

These numbers sort of obscure the fact that if we kind in a little more detail, here is the genetically affected and they had mean LV walls, 11 to 40, whereas it was seven to 16 in the unaffected. Well, you usually consider anything over ten or 11 as being abnormal. The other was the ratio of septum to free wall, was greater than 1.3 and only 70 percent of the genetically affected but still present in six percent of the unaffected. The three of the 39 genetically affected have normal echoes.

What happens to these people that have the disease and they haven't developed the echo change yet? Are they going to have a normal mortality? Nobody knows. If you have to go by echo and you don't have genetic studies, if you have a wall thickness of 16 which we consider very abnormal, only 75 percent were genetically affected. If you had a septal posterior wall ratio of 1.5, where you think everybody has hypertrophic, then only 75 percent had an abnormal gene.

What's going on? It's just that perhaps neither test is perfect. I don't necessarily think we have to accept the genetic test as perfect.

I don't want to spend much time on hypertrophic cardiomyopathy. You'll see in the manuals and various statements, here's a great thing in the *American Journal of Cardiology,* hypertrophic cardiomyopathy has a relatively benign prognosis. This is because instead they were emphasizing that in a clinic population they do better than ones referred to NIH.

Instead of having a two percent annual mortality, it was .3. Actually in the study the average mortality was one percent. Well, if you calculate mortality ratios you're left with those kind of numbers in a clinic population. So obviously we shouldn't start looking at them to underwrite them until they're 50 or 60 years old.

If you look in the asymptomatic patients that were followed at NIH, at 33 to 55 years of age their mortality was eight and a half percent. These are an asymptomatic group and again mortality ratios, they're out of sight. If you look at the patients at NIH who were over 65, you can see that under 40, only two percent had symptoms and it was only after they exceeded 55 that the severity of the disease became obvious.

If we look at the kind that they're trying to tell us has normal mortality, there was the so called Japanese form or the apical, there's one fairly reasonable study with one out of 36 deaths followed for four years, but that's still three percent mortality out of 100. And then Dr. Weigel, one of the leading experts in hypertrophic cardiomyopathy in Toronto, ended up with eight pure cases and no deaths. But I'd feel a lot better if they had 200 cases that they had followed 30 years before they told us that.

If we look at the Japanese, they're expecting an annual mortality of about .3 percent in the apical form and this still has mortality ratios of 700 and 450 percent and only after age 50 does that level of mortality get to be an insurable. So the manuals that tell you plus 100, plus 200, well, that's fine, but I'm a bit of a disbeliever.

I want to go now to the Marfan syndrome which is an automatic decline for most of us, so you wonder why are we talking about it. This is the pioneering study of Mecusek in Baltimore showing the mortality in the Marfan which is rather horrendous. The average age of some of the males in the Marfan series is around 35 to 40 years without surgery on their aorta. You can see a little leveling off in the females after about age 55.

Does that mean they have a less severe disease or it may even mean that they didn't have Marfan syndrome because the diagnosis is not always clear cut.

Two situations can arise in underwriting and it has to do not with the proven case. It has to do with the one where the odd history you have, which really helps you when you're doing underwriting, you'll see a question mark, Marfan and then no details. You say, oh, great.

Or you get a child born to a family with Marfan. I've had four children born to mothers that I follow with Marfan in the last three or four years and three have ended up with Marfan and one not. But of course, the first thing they do is want to insure the one-year-old and do they have Marfan or not.

You can tell right at birth whether they've got Marfan or not. Their fingers are long and their aortas are big and surprisingly the aorta starts stretching in utero. You can get the first case, where you sort of question Marfan, you want to look for family history and 30 percent don't have it. And you want to look for the ectopic lens which you need an ophthalmology report and then the echo of the aorta. With those you can be fairly certain, if those are all negative, that probably this is just a tall skinny person.

Of course, we're not there to do physical exams, but the arm span is often at least three inches greater than the height. We divide people into upper and lower halves from the pubis down and in the Marfan, the segment is always larger than the upper and ratio is less than .9. If these things don't fit, it's likely you're not dealing with a Marfan.

One almost always has mitral prolapse in the Marfan and a dilated aortic root. The biggest problem with the aortic root is that

there is a large standard deviation in the normal population and I don't feel they really sample enough subjects to give you normals for a tall person. While the average is 30 millimeters in the tall person, it can be up to 45, and I don't think any aorta normally hits 45. That's using standard deviations derived on a smaller population. The chest X-ray is of no value.

This is an 11-year-old with an aortic aneurysm. You can see how big his aorta is here and it's hidden right within the heart shadow. Is there any hope for us underwriting Marfan? Here's the Cleveland Clinic series and if they didn't have an aortic diastolic murmur at entry into the study, you can see that mortality was pretty leveled off and that's in follow up, going out as far as 30 years. So that some Marfans can have a good prognosis.

The other thing that's encouraging is that propanol will retard the rate of progression of dilatation of the aorta and this is a study just published this summer. We've been using propanol in these patients for the last 25 years without really knowing whether anything was being accomplished, but this is the controlled series with a ratio which is the normal aorta size, went up .084 percent per year whereas in propanol it was one fourth of that.

So quite encouraging that perhaps that Marfan may be a manageable disease, even to the point of being able to underwrite it if one has an older patient with aortic dilatation or progression. In control series on the propanol there were six out of 38 deaths or dissection or only two of those on propanol.

I don't want to get into the very touchy issue of cholesterol in children, but it's of obvious concern with long term development of problems. The National Cholesterol Education program does recommend screening children and there's considerable objection and debate on the problem.

They recommended if a parent has any cardiovascular disease, if the parent's cholesterol is over 240, which would be half of our insurance applicants, and if there's a family history, if the child is adopted or one of the parents is missing and there's no family history which in today's society is a large number of children, and they recommend drug therapy if the child has an LDL greater than 160 despite diet.

I thought there was an excellent title from Dr. Goldstein who had a one page editorial in the *American Journal of Medicine* and his conclusion was that there are a lot more important things for children to do than watch their cholesterol, like growing and surviving and eating ice cream.

But here again one's faced with, this is not an atypical family history from Quebec in our experience that the father had a bypass at 43 and that the father's brothers had coronary disease under 55 and then there's a sibling with an infarct two months ago and then they want to insure this 18-year-old.

Obviously there's some problem in the father's history and the 18-year-old has a cholesterol of 260 and a HDL of 34 and has he inherited the gene, and we don't have genetic studies and you know, someone will come up with a mystical plus 100 out of the air and sort of gamble and hope that they're dead before the child is dead.

(Laughter.)

DR. CUMMING: Here's a study and looking at, this is the original person that had coronary artery disease under age 60, proven by angiography and then they looked at the family enough to sort of rule out inherent lipid disorders and they found a probably inherited dislipidemia in 57 percent of the patients with premature coronary artery disease and no abnormality in 43 percent.

What was the frequency of these abnormalities? Very interesting. Lipo-protein A, excess 19 percent, a thing we do not usually investigate, familial dislipidemia, familial combined hyperlipidemia which starts usually more after 35, 40 years of age and then the one we would think might be there, familial hypercholesterol, we see only in three percent.

So ordering a cholesterol is not the greatest starting point for these patients unless at least one throws in something where the low HDL will also be picked up.

There's an interesting study available on, again, I think what's going to happen if we can't do genetic testing, we have these genetic markers and cholesterol is one. We're using it and we'll continue to use it.

And the genetic studies will teach us what we should know about the markers we're using, that if you have a general population and have 98 percent specificity in just a total cholesterol, if you've got a 360 cholesterol in a 40-year-old, 98 percent of those have heterozygous familial hypercholesterolemia, if the person is under 18 to 70. So that our 18-year-old we saw with 260 cholesterol probably has an 85 percent chance of having FH.

If we look at where there's relatives that have FH, if you have a child and the cholesterol is only 220, there's a 98 percent chance they have FH. If they've got a cousin, it needs to be up to 230. These are studies that will be becoming more available as we get the combination of genetics and standard testing.

You can look at it the other way. If a person has a cholesterol of 300 and in the general population they have 100 percent chance of having FH, this is an 18-year-old, but if you have them in the general population, they're not, and it's 220, it's not as likely because of the infrequency in the general population.

I'll go onto another coronary problem that does interest us. Kawasaki disease is more common than rheumatic fever in the United States and Canada, even with the little recent blip on rheumatic fever. These are the figures in Japan where the numbers are enormous. I visited three different universities in Japan several years ago and it was not uncommon for a single hospital

to see 40 cases of Kawasaki disease on the ward in the acute phase of the illness. It was mind boggling.

Coronary artery disease in Japan is present in 50 percent of them. Probably in this continent it's 25 percent. In 30 of these it's a transient dilatation and this always regresses. The question is then, is anything going to happen to these people, and the theory is perhaps they're going to get arthrosclerosis. But with 20 years of follow up now available this doesn't seem to be coming true.

In those that have coronary aneurysms, a certain number go to large aneurysms which will get thrombosis and ischemic heart disease, but a certain number can get infarction and a certain number will die suddenly. We saw an 11-year-old present with acute myocardial infarction in our emergency and we ended up two days later doing emergency by-pass and there was no definite history of Kawasaki disease but going back in this pediatric illness, it was obvious he did have Kawasaki when he was one year of age.

So it is a potential problem. Can we pick out those that are going to have trouble? In the first place if they have an aneurysm eight millimeters in diameter, they don't get better and they're going to have trouble. These aneurysms can get thrombi in them and fortunately most of the deaths occur during the first few months and the infarctions during the first year, but still some of them are late, like the boy that I told you about. They often have only single vessel disease.

So the real punch in doing any assessment in these patients for insurance is did they have an aneurysm in their artery. And it was pretty easy when they used to do angiograms, injecting into the aortic root, but we rely entirely on echo. But you really don't need your echo being done in a small community hospital by a technician and a radiologist.

You need, unfortunately for the 95 percent accuracy that has been touted and that is generally in a teaching hospital that has had a lot of experience looking at proximal coronary arteries. There's 95 percent sensitivity for the echo because most of the aneurysms are in the proximal septum, at least proximal coronary artery.

Echo will not pick up stenosis, echo will pick up a aneurysm, it will not pick up an stenotic artery.

This is about the only good news in Kawasaki disease, is that once they've healed there's no current evidence, with a fairly good follow up, that they develop coronary artery disease. It's still a theory. There can be positive exercise tests.

A few other things that happen to coronary arteries is fistulas. You'll see a report of a coronary artery fistula, not uncommon in an ordinary coronary angiogram done on an adult for coronary artery disease. You may get a little concerned but most of these are small little fistulas that can be left alone and not that important.

They may have a fairly large shunt and can have a continuous murmur and usually those you end up getting tied off with a surgical cure. The large fistula can have symptoms and the surgery cannot, sometimes they're sticking wire coils in these instead of sending the surgeon in.

So a fistula looks this way. You have a normal right and a normal left, and here's the left connected to the pulmonary artery and if this is tied off, or it's trivial and it doesn't need tying off, then these really don't influence mortality. If they've got underlying coronary disease, of course, that's the issue.

Very different is a thing called the anomalus origin of the left coronary from the pulmonary artery. Here we don't have the coronary coming from here at all, it comes from here. These patients get a variable degree of collateral circulation across and they look like an AB fistula. Some of them will die suddenly in early infancy, some of them present as having angina in the first months of life. This is a very different underwriting situation. These people are probably never normal.

Here's an angiogram on one such person and here is the right coronary coming from the aorta and there's the tremendous collateral and then there's the left and it's coming from the pulmonary artery.

I don't need to go into the treatment but their problems are related to damage of the left ventricle, we have a thin walled coronary, we can get rhythm disturbances and sudden death. They can get mitral regurgitation from papillary muscles and even though they've been surgically corrected, their long term prognosis is uncertain. I've had one 24-year-old die suddenly.

Another coronary problem is the aberrant right coronary that comes from the left coronary sinus and it may course in between the aorta and pulmonary artery. I found it very difficult to identify these at an angiogram. You end up standing on your head trying to look and see between those two things and you can't. The echo is better. These patients can die suddenly during exercise in adult years. Enough for coronary disease.

The one congenital heart disease presenting in infancy that is fatal that should have normal mortality if corrected is the total anomalus pulmonary venous return. These babies are sick, they're in heart failure, they're blue and yet the surgery nowadays, when I started in cardiology the mortality was 100 percent and then we got it down to 50 in the '60s and today it's down around ten or 15 percent, repairing these sometimes in the first week of life.

In this situation, this is one of the more common kinds. The pulmonary veins come into a kind of a common chamber in behind the heart and then this goes up what's called a left vertical vein to enter the anomonate system and then eventually the blood comes down, mixes totally in right atrium, some crosses of ovale to the left side, most of it goes through the lungs. When this is repaired, a window is created between this common vein

and the left atrium so all pulmonary vein venous return now comes into the common vein and then into this side of the heart.

In underwriting these, one has to be sure there's no residual stenosis here. I've had a few patients die in later years of pulmonary hypertension and the equivalent of mitrostenosis. The more easy kind of repair is where all the veins come in and empty into the right atrium and all one does is take out the atrium septum and put in a patched atrial septum that comes in on this side of the veins and they all automatically join in the left side.

You won't get the stenosis that can occur in the other kind. But if these patients are underwritten around age ten or 11 years of age and there's no obstruction at the start and none now, if they don't have a rhythm disturbance, if they have no residual pulmonary hypertension, they don't have any shunt, then I think one can underwrite these.

Congenital corrected transposition of the great arteries, this is a little thing you can throw at the med students and sometimes these may be present if they're septal defects, they don't do as well as standard septal defects.

They're a caution for any underwriting process. Even the ones with normal hearts run into trouble. You're not going to see the main arteries being wrong from a standard X-ray. When you do a heart cath you immediately find out that you've got something a little unusual because instead of going with your catheter into the right ventricle and go up the usual course out here, it goes up medially.

Here's a drawing and it shows what the problem is in corrected transposition. One has the pulmonary artery going up this side of the heart and then the aorta up here and the reason they're not blue is inversion took place in the ventricles and I don't really want to get into that. The main thing to realize is that, this is the angiogram of the same, the aorta going up here. Patients get AV block in later years and mitral regurgitation and can run into serious problems.

I think we'll end that right now. This was a hodge-podge of different things that can happen to the heart. We have to think that in childhood there are heart problems that can carry on into adult years and yet in previous talks we've emphasized that 90 percent of congenital heart disease is repairable. It may not be curable but it's repairable.

And a lot of them will have, we expect, fairly normal mortalities. We've presented some here that have really a difficult time to look forward to, some that we haven't a clue to what's going to happen to them.

(Applause.)

DR. BAKER: We can take a question or two. If anybody has one, please come to the middle microphone.

AUDIENCE MEMBER: Good job, as usual. Dr. Cumming I've always enjoyed your talks.

Your slide regarding the bicuspid valve, could you make some comment on the age at which a non-stenotic problem is discovered. Does it make a difference, say, at age 30 there is no evidence of stenosis as opposed to no evidence of obstruction, a phenomena say, at age 60?

DR. CUMMING: My experience following a fair number of people every five or ten years that have a click only, is that they never, I haven't lived long enough to find one that's gone on to become stenotic. I feel the ones at 30 that have a little bit of stenosis, they started out with a little bit of stenosis. Or if they've got a moderate stenosis at 30, they had a little bit of stenosis when they were five and they've now got calcification fibrosis.

I've seen a fair bit of valve calcification and mild stenosis at fluoroscopy in 16 year olds with a bicuspid aortic valve and just a gradient of 20. So it's that little bit of stenosis that I think makes the potential for a problem at age 30, that stenosis would be evident then right from childhood.

Don't know whether that answers the question. I think the one that's a click only is obviously, to me, they're going to at least hit age 65 or so before they have significant hemodynamically significantly valve problem. Their main risk to health is endocarditis.

DR. BAKER: Thank you very much. When we were putting this meeting together, Gordon wanted to be one of the first speakers because in other years when he speaks on the third day, you have to keep your suit pressed, you have to shave every day, you can't get your tie dirty. So Gordon, we're grateful you could come this year and that you're on the first day. Gordon has always gotten top grades and if we had a one to ten judge here, he'd hold up the ten, I'm sure.

Which leads me to remind you that you do have evaluation sheets in your kits and Jay Smith asks that you make out these evaluation sheets, hand them in at the registration desk at the end of the meeting or send them to me, the address is on the sheets. Also if you want CME credits for this meeting, you have to sign in at the morning session and the afternoon session. They'll be on the back table as you go out this auditorium.