MORBIDITY AND MORTALITY IN INTENSIVELY MANAGED POPULATIONS OF BLACK RHINOCEROS (KENYA)

Richard Kock

TAPE 3B 055

R. Kock: I am going to talk about the Kenyan situation. To first introduce you into the subject of Kenya, I am just going to throw out some pertinent information. This gives some idea of the historical situation with rhino. In 1925, you can see then that all the country was good rhino area. They were found all over. It is an excellent country for rhino. As Raoul [du Toit] pointed out, it has a bimodal rainy season, good vegetation in many areas, and large pastoral communities where the rhinos used to live. They were not being poached at that time in large numbers, local poaching probably. And then by 1975, effectively it was getting to the point of extreme restriction. And then now, we are down to a situation which really looks grave *in the 80's* and we have about 400 now. But, they are coming back up.

Then you look at [human] population densities, this is an interesting thing because obviously it has got one of the fastest growth rates in the world, 3 1/2 to 4%. Your main [human] population densities... This is Uganda, that is Kenya and Tanzania. You can see these areas of high [human] population density. In Kenya, these are excellent rhino habitats all around here. But in fact, where the sanctuaries have been set up and where national parks handles it, are not far from heavy [human] population densities. I think the point about that, is that it is in those areas you have resources, it just works out better. Like Nairobi National Park is next to the city and it is a very successful sanctuary. Now whether that is right or wrong, or a concept we should follow, I do not know. But it is just an interesting phenomena.

So, what you have is lots of little populations now all over Kenya. And a few truly free living animals which are still in their original habitat. We are trying to gradually mop them up wherever we find an individual. It is amazing how they pop up. We try and catch it and bring it into a sanctuary. One right up here. It traveled from the time they first spotted it 200 kilometers before they actually got it. That was a good chase. It just kept moving. It had no ears and two bullet holes in its legs. It was a nice male 082. And we found odd ones in Tsavo still. Another one literally three weeks ago popped up. We really thought that they all had been poached out of Tsavo East in the last five years. It is encouraging. So the situation is looking pretty good in many ways. No poaching, no commercial, no old poaching for of course three years, in a sanctuary approach 088 Zimbabwe.

I will give you some individual information about translocation.

Jessup: Can you give a little insight as to why you are not seeing more poaching in Kenya as opposed to the other parts of Africa?

R. Kock: It was basically being orchestrated by the *parks*. So what we called the WCP, the Wildlife Conservation Management Department, was in the 1980's totally corrupt, and with ministers

involved and so on. They lost a lot of money. You see the government stopped paying people. They had no 094 they had no food, no clothes. The whole thing was quite corrupted and it was very efficiently done. In Tsavo East we are talking about 4000 animals systematically removed, using park vehicles.

Jessup: But you do not have a problem of independent entrepreneurs?

R. Kock: It does not appear to be the case. But, there must have been, because your ivory trade was very big there and therefore the samething with the ivory trade as for the horn trade. And so I guess those people are still there. As soon as the Department was changed over, it stopped.

M. Kock: I think that is a very important point, but I do not know how much... *I mean we have* suspicions in Zimbabwe that there is possibly some involvement. We recently had an MD, a member of the ruling party who was convicted of trading rhino horn. He said to the judge 104. Fortunately, he was put away for five years. I do not know how much involvement is there. Our situation is mainly in terms of 106 involvement.

R. Kock: It is a very difficult question. I think it has always potentially been a problem. I think in Kenya it is OK at the moment, but in ten years things can change quite quickly. These people are opportunistic, because they have so little and they are greedy. They want to have a motor car, etc. So it is difficult, their options. It could easily go back again.

Morkel: We have got probably the second largest population of black rhino in 110, but I think we are the next serious poaching area. We are on the edge of an abyss, called 111 abyss. I would be surprised in five years if we have 50% of what we have now. I am convinced 113. We have already seen warning signs of poaching starting to pick up.

R. Kock: I think this is where the private sector is so important. This is the way we saved the rhino in Kenya. People like this guy *Gilman*, who just did their own thing 117*. He just went his own way and was politically astute enough and strong enough to be able to isolate himself 118 security and it worked very well. I think that should be encouraging. Maybe even in places like Namibia. I know this *in situ* population and that sort of thing is very important, but I think what one man can do who is wealthy enough to get on with it, should be our best bet.

M Kock: I think Rick, I would just emphasize from that 121 conversation is that this sort of workshop, this liaison and assistance from the National Rhino Foundation and Zimbabwe and 122 in Zimbabwe. This work is so vital, because make no doubt, in the wild our backs are against the wall. We are trying desperately. Our white rhino population in Hwange National Park has been decimated in less than 6 months. We are talking about 87 rhino taken out in 6 months, without us even knowing about it. We do not have the resources. I think if we can sort out these problems with rhino with the *ex situ*, with the captive breeding in places like Australia and America, it is absolutely vital, absolutely vital if you are going to see this animal around in 20 or 30 years.

Tolt: I can not agree more. I think we must also put the Institute of Conservation 129. While we have got our own *critique capacity whatsoever*. And while there are obviously ever longing forces of poaching operating in many areas. Where things *have got a problem, I think it is* 131 one can maintain effective field techniques for free-ranging black rhinos in Africa. And I think we must be very careful, that while we outline these concerns about the prospect for rhino conservation in Africa, that we do not paint too gloomy a picture, and therefore lead to distortion in the way in which we develop our strategies for rhino conservation. We must maintain a balance between *ex situ* and *in situ*.

R. Kock: Absolutely, and I think that is the point, not to be too pessimistic, and to expect again to make some 138, and to come to terms with the fact that maybe black and white rhino will always be around for the next thousand years. If we can accept that and come to terms with it, and just manage them, I am sure they will be around.

Anyway, if you look at mortality data and unfortunately again, all the stuff is so poorly documented. You know, it might be documented on bits of pieces of paper lying around in old... The problem is accessing some of these things. I am trying to now go through some old Tsavo stuff. If you look over a long period, especially 20 to 30 years, it is not an enormous amount of translocation that has been done. There were 97 translocations between 1961 to 1984. Mortality again is difficult to tie down. There is something *around 35%* mortality. They were quite high at that time. In 1984 to 1993, 96 were translocated, and then seven capture related mortalities; and there were *two drownings*, these were due to long induction period; two stress anesthetic effects, again long induction periods; and three traumatic, the animals sprained their necks 151 one individual. But that was that level. And the release was a higher problem. Nine animals were fighting, mainly because of the bull, *and maybe this age* 152. So a total now of 16%, that is obviously a bit better. I think a bit can be gained from what has been done. 155* Things are better all the time. The key is to get this figure down below this increment that is important for increasing population. Whatever your management is for the population , you have got to have a mortality lower than the recruitment. Otherwise, you are not going to go anywhere. And because of the dynamics of this, you know one place *closes down, you have got to move them out*, you know, et cetera, et cetera, genetics. If you can not get this management level well below the recruitment, we are really wasting our time.

Just to briefly go through the techniques that we are using... We try to use the helicopter whenever possible, using Pete [Morkel] and Mike's [Kock] techniques, and four to five minutes of flying. We try and load in 60 minutes. And we use a nalorphine partial reversal for loading and 164. Perhaps I could just run you through the translocation technique.

I think it is very important to not get too tied down with one species, because your mind gets out of *sync*--no reference to anybody. Anyway, you have to make sure the family is happy and well protected. And again, I think in whatever we are doing in terms of technology and all this veterinary stuff, is that protection of the animals is absolutely vital. It is resources and people which are ultimately keeping these

animals from being poached. This is actually a poacher in Tsavo East going after one of the last remaining free-living. This are local poachers, they were meat guys and going for ivory. But they were in this area called 183 Valley 184*.

Helicopter, we have been using this *Hughes* 500, it is very nice. Always identify your animal--make sure you have got the right species! After immobilization, we bring the crate down, put a cotton rope around the head and right back under the chin, if you do not do that it slips off. Obviously have a *mask*. We run the rope to the back of the crate and have at least six people and then pull it in after giving the nalorphine. We titrated it. Give them a bit as Pete [Morkel] said in the beginning, then give another "dollop," sometimes we do, sometimes we do not give another "dollop" and then they go in.

Blyde: How much does it take for them to get up?

R. Kock: Sort of between 50 and 75 mg, you should be...

M. Kock: The way we do, is we have *the right* increment. But firstly, we give at least 40 mg rather quick. And then either use the *other part* of it; or in fact, what is much better is to pour water in their ears.

R. Kock: Yes, we do that--pour water in their ears. All these things can help. The reason we do this, it is an expensive drug, so if one can get away with less, great.

M. Kock: Actually I think Pete Morkel is the one that told us about water in the ear.

Bolin: How did you figure out how to do that? Was it an accident?

Jessup: They do it to horses all the time.

R. Kock: I think the other thing is fatigue. These are 211*. I think the point is some of these guys 212 poaching. But it is motivation at the end of the day. It is amazing, these locals actually... This is a free-ranging animal, and we had to track it the whole week to find the thing. And then one day 217 are you looking for rhino? We had a lovely discussion with them, the helicopter crew. They kept looking for the exhaust of the helicopter, they thought that is where the feces came out. The guy could not understand how it could cross rivers and somehow get across the idea 223.

Heat, I am not going to sort of mention heat too much, I know Pete [Morkel] is particularly concerned about heat. And obviously in Namibia it is critical, and in certain parts of Kenya too it can get really hot. Hyperthermia, I think the damage that can come from a combination of hypoxia and hyperthermia, is actually important. I hope to speak to the pathologists about that. You know I wonder about the liver and so on. 229 what happens when you give that combination.

Morkel: 229 I think what is important is that prolonged induction and excessive exertion obviously are the most important ways of making an animal hot. In fact, environmental conditions are usually not a problem, it is prolonged induction. We have often put animals in extremely hot conditions, but if you have got a quick induction it is usually not a problem.

M. Kock: Rick, I think also for those individuals who have ever immobilized a calf... Even on a cool day those animals temperatures continue to climb, and they will go up to 236 centigrade. They are a real problem, the young calves, because in spite of what you do, it continues to climb. You have to watch young calves very closely.

Jessup: But you know with a lot of animals with small body size, even if they get hot faster, you can cool them off faster. With the big body size, once they get hot, you can not get rid of the heat.

R. Kock: OK, running into bomas now. The bomas that Mike [Kock] in Zimbabwe has are so much more advanced than us. They do a lot more translocations as well. We are a bit sort of "Georgia" in our approach to building bomas. We sort of get away with things out here. You can see a little bit of blood on the door, and obviously this is one of the problem areas where they crash their faces. We have a 248.

I think with release, quietness is the important thing. Often people get excited about these animals--*I want to see, it won't take very long*. One animal that went into a boma at 251 place 251* after we had gone, he had been mostly quiet until then.

One of the things I have noticed with more stressed animals is serous nasal discharge. Have you guys seen it?

Morkel: At what stage is that?

R. Kock: Within seven to eight hours and the next day you will see it, and then it begins to dry up slowly. This animal had been given long-acting tranquilizers, then just went bananas, like 261 the minute it was put in the pen. You should have seen the dust, it was sort of a cloud which formed above the whole complex. It was going around in circles, it actually broke its way out of the crate. So I am interested by this. I think there must be some lung related phenomenon. I do not know, is it coming up from the lung? It seems to take a bit of time.

Morkel: I think the amount of dust in the boma, etc. is also quite important.

R. Kock: But the other one is fine.

Morkel: And you said this specific one was making the dust?

R. Kock: Yes, but it was right in with four other rhinos, the dust was everywhere.

Toit: When they are snorting a lot, they are ingesting the dust and then bringing it up.

R. Kock: Maybe yes, that is a possibility.

M. Kock: I think actually we have seen it in a lot of rhino we have been working with.

R. Kock: It is only the ones who appear to be a bit more stressed *during the hot season*.

Morkel: We sometimes see it the whole duration while they are in the boma. 271 discuss it and tell people, they are not terribly worried about it, they see it an awful lot.

M. Kock: It seems to be heat related. If it is a very hot day they will do it.

Munson: It never foams?

R. Kock: No.

Munson: So it is not apt to be from the lungs if it is not foam.

R. Kock: No, it just looks like a very serous...

Jessup: Is it likely to be lacrimation, from the nasal lacrimal duct? I have seen animals highly stressed out off their range, just pour tears from their eyes, and you can see this discharge from their nose. But, once they calm down... I just wonder if that is sometimes just associated with the stress response.

M. Kock: We can just call it "runny boma nose syndrome!"

R. Kock: It needs to be investigated.

Cttino: You see that a lot in captive animals. I have never really been that concerned about it.

R. Kock: Well, it is an observation. Ticks, you know we have had quite a high level of ticks in buffalo. But, sometimes we get quite high levels of ticks, particularly in Nairobi Park and quite a variety. My hairdresser in Nairobi keeps finding them in my hair!

This again is not fancy system you understand, pretty simple. This metal... The great advantage of this, you can push it and put it up and take it down very very quickly. You can move a boma, it is a very flexible system. And actually it seems to work extremely well. We always find a tree to put it around to give it a little shelter. This is in Tsavo. We put fly cups out for tetse. And that is part of 292 mention a bit later in more detail. We make sure there is plenty of browse around.

Now, one point I wanted to bring up was on hematology. Obviously, we are trying to do as much as we can. We are not getting *as much data as Mike [Kock]*. Our real problem is Nairobi about getting stuff processed. We have stored a lot of things we could do some retrospective, like *chemistry and that*. But, we do PCV's quickly in the field immediately after capture, and then our *path* is different from Zimbabwe's, as much in that we transport, then boma; we do not boma, then transport. It is a different approach. Probably the reason obviously is different country and time and so on. Is that the main...?

M. Kock: Yes, we have been talking about getting some of these animals out of where we capture them to... Well, we are talking about some animals that are kept up for 12-13 hours in a crate in a truck, six hours can end up killing them. If an animal has been down for 12 hours, you do not need to put it in a crate and transport it right then. We get it straight to the boma, wake it up and evaluate it, and see how it is doing. *We can guarantee that putting them in a crate...* And we have done it with the conservancies now, we are not keeping them in bomas. We are catching them, and they go straight to the truck, and they get to the conservancy bomas within 12 or 6 hours, and that is quite acceptable. The only reason... Some of the areas are extremely remote, we just can not get them out. I am not sure evaluating them for a two week period is acceptable.

Morkel: There is no doubt the longest translocation we have done was a northern animal shipped from 314* about 12 hours. Good capture 316.

M. Kock: To give you an example of our day to day. One animal due to logistics was darted at 11:00 in the morning and the trackers reached the rhino at 2 AM in the morning. That is how difficult the capture was.

R. Kock: OK, going on. So PCV. I have noticed in the animals we have done, and I guess 320 now, all these individual's PCV's dropping over three weeks from between 50 and 45 to 30 and 35. So prior to release there is definitely a change in PCV. But the serum is not colored.

Ryan?: Where did you take 325?

325-328 MIXED INAUDIBLE COMMENTS

R. Kock: *Maybe* it is physiological rather than just...

Harvey: How big are the spleens in these? Because most of that changes in the horse is because of a big spleen. A third of the blood volume is the spleen. Are these muscular spleens, contractile spleens?

Morkel: I have got an idea, in fact. If it is definitely a loss of red blood cells, I have seen it in other species as well. You see it at capture and about a week later, inheritably they start a drop.

Jessup: Are they losing weight and getting hypoproteinemic at the same time? Are they anabolic at that time?

Morkel: Sometimes, most certainly. But, often in fact the animal looks to the eye just fine. Yes, the protein picture is not that different, but it is a drop in hematocrit.

R. Kock: The reason I show you a picture of this animal is he has got *sand*. He was rescued from the *Mora* and went to this lady up in 341 for handrearing. He is the tamest animal I have ever gotten, an incredible animal. We moved it to Tsavo East, because it had become urban and a lot of Nairobi animals are now urbanized. They wander into the town and at night they go past the bars and the prostitutes; of course it was *consternation*, our track was also beginning to go into these bars! So, we decided we had to save these animals from moral degeneration. The poor animals have now been dumped in this historically unsafe area of Tsavo East. "Sam" was translocated and he showed absolutely no stress. It was a matter of giving him an intravenous induction and he went into the crate. And he thought this was just terrific, he thought it was a wonderful thing and during the journey *he was chatting with all of the guys*. He came out of the crate, thought it was great with all this grub around--totally relaxed. You could walk with him, sit on him. And his PCV showed absolutely zero change from the first immobilization to the last. This was the only animal to actually do that. So that was one animal.

Harvey: And what was his PCV?

R. Kock: Nothing, no change at all.

Harvey: No, I mean...

R. Kock: 50.

Q? 363: How often do you see *hepa-grade* urine?

R. Kock: Well, only on this one individual have I. And another animal was as I said, quickly... It was quite stressful, the journey and everything, very very rough roads. The roads going up to 366 are horrendous. There was a lot of bumping and it was pretty difficult. Obviously very very *remote*, very stressed animal.

Harvey?: The red cell life span is so long, that the hematocrit is not going to drop due to nutrition *in three or four...*

R. Kock: No. of course not. I have got a picture of urine *sediment*. That is urine in an animal I took that about three days after translocation.

Harvey: Does the urine have a lot of 376 in it?

R. Kock: Try and think about translocation, you have got 378. Our policy is prerelease, we do prerelease as well. If it is short translocation just to similar habitat, I think we are sort of keen just to release it straight away. And then obviously otherwise, boma for 30 days, and *we use sugarcane* a bit. But, mainly on browse 386 pretty grim there, so our main emphasis is just on browse. And I do not see a very significant 388. They feed very quickly back. I have had some animals within 24 hours munching, but certainly within two to three days.

Morkel: We have had them actually walk out of the truck and start eating.

R. Kock: The other sort of policy thing is like tetse. We have this highland situation, where you take animals from 6,000 feet or higher down to 1,000 feet, and going from non-tetse to tetse areas. There has been a lot of talk about this. It makes the tryps people in Nairobi vary nervous. Steve *Mayhak* has been helping us, he is a very excellent *trypanizone* scientist. But, they get sort of carried away with it. We do have a policy of... There have been some problems with rhinos in the past with tryps. I think they were stressed animals who went down with something, perhaps a *fever*, and were ill. Historically, there appears to be some deaths associated with this in a very concentrated group, very related. But, we have a policy of putting the bomas now away from the hills, from the main vegetation areas where the tetse are and into other areas when we can. We try and time things. We are not too strict about it, but we do try and get to a situation where at least the vegetation we go to will be good, but before the flies hatch. It takes about three weeks for the pupae and so on to come through. So they get their challenge, but it is a growing challenge, rather than in the middle of the rainy season, or the end when there is very high level of flies. At those *troughs* we use an indicator, sort of *count*. This guy, Steve *Mayhak* comes down and he has these feeder *flies*, and this wonderful little apparatus which the fly can put its proboscis through. They are wonderful, because when they feed and fill their stomachs, they sink. This guy is always wearing a white coat. He is slightly crazy, and suddenly he goes, "They are sinking, they are sinking..!." The flies are fat.

M. Kock: Just one comment about your prerelease thing. I think if there is anyway you can justify prerelease, is your monitoring closely these things. You are releasing animals into the bush there and you are not monitoring them. And I think you are doing...

R. Kock: Yes, I agree. And you have to remember most of these are sanctuaries, and therefore I do 422. And therefore, prerelease is the case. In Tsavo East, where now we are prereleasing in, we are putting radio transmitters into *them*. We always make sure we have a health check before release. So we do *not have the same problems as you*...

M. Kock: And, I have looked at some of the national parks historical information. You read in there that the animals were prereleased and sort of did not know where they were and three months later 427. You can not do that these days.

Morkel: On the prerelease, it certainly has become policy now in South Africa. Almost all white rhino which are caught and translocated are prereleased, and a very small percentage are boma transfers, just because the problems involved with boma and the white rhino.

R. Kock: Just going through the main points I guess.that we are concerned about... I think trauma... No mortality or social disease in the past ten years or so, there have been a *couple metabolic probems and* stress, in boma in a complicated animal. So it is not the disease that is the issue, trauma definitely, obviously good induction we have had, damage to the crate, procedures, aggressive behavior, horn plate damage and fighting. These are areas of concern. Again, the stress 439. And obviously hypoxy damage is interesting.

Parasites, 442 very *heavy stuff that varies* in Nairobi animals. But, as soon as you move to dry conditions, within three weeks they decline. What is interesting in the rhino is that the *muscat* fly is one of the main ones in Nairobi. If you look under the microscope you can see actually the filarial thing round the *proboscis*. It actually wraps itself around the *proboscis*. I think it requires constant biting from these flies to maintain 447.

Bacterial, I think *Raoul [du Toit]* never mentioned that Mike, about treatment for 449. But occasionally we have seen...

M. Kock: Standard.

R. Kock: Standard. And obviously the skin necrosis associated with trauma. I think it is something we need to talk about. And then obviously conditions in the boma: the lack of shade, the wet-cold, 453 in whites, but pneumonia is another thing in the season in the cold. In Kenya it can get very very cold.

To go on to mortality. Just generally,...

Morkel: On the stress, the one that I think is very important is the stress on a lactating female with the whole capture process. If your female does stop lactating you might have a clear chance of losing that calf, or certainly ending up with an orphan situation which often means that the animals are not going to go back to your breeding populations. So it is a breeding problem essentially.

R. Kock: We are not translocating animals with calves, is what we have done. Except, now we just tried one with a three year old, put the two together, and see how that would go. Because of the intraspecific aggression problems at release sites essentially.

Morkel: I think actually even more important than that... We had a lot of success moving cows with very young calves. To actually catch, put them in the boma, and have them settle down is generally not a problem. The problem is at the release. The cow is in a strange area, she goes of like a bomb, and the little fellow falls behind. I think before we can catch and move them 477, they do not do well personally. So 478. So, it is really a problem 479.

Montali: Richard, in the ones you noted stress in, physiologic stress, did you see any of the cutaneous ulcers, or anything related to it? You noted PCV drop, was that hemolysis?

R. Kock: It does not look like hemolysis looking at the serum. Within three to four weeks, I am not sure, the hematologist can tell us whether that could happen without a serum change.

Montali: No evidence of liver disease?

R. Kock: We have material from some of these animals, for *biochemistry.* Simply, *have not got it* done. *We need* to do that.

I think that one of the problems... You know once the animal is released, if you can get close enough to detect some of these. But, I have seen these in skin ulcers. In other words, there is punctate all over the body. I think I have picked that up.

Munson: In these animals?

R. Kock: No, I have not seen anything like what I have seen in captive animals, in any of the wild.

Munson: In translocated animals?

R. Kock: I have seen skin problems with trauma. You get an animal that appears... and you caught it and looked at it, and it is in bad shape. It has got focal areas and necrosis with skin loss, sometimes epidermal, sometimes the whole skin layer, and this rotten skin goes black. I wonder whether these historical *trauma* were in fact skin 499 epidermal, that is why you have got a change in color, but related to some *vasicular* problem. But, it looks horrific and 503 allow to go through that. But, we have found penetrating wounds going into the chest sometimes, fractures, obviously trauma related. We observe these individuals at a water hole which have been hammered. One of the young groups sort of fight with a resident male, and then subsequently develop these skin things. So they get hammered and *I guess damp gets a hold* of that skin, then gradually goes to a 509. But, how much of it relates to some of these zoo cases, I do not know. But, I remember in captivity with whites, once or twice seeing these very disseminated skin lesions after 511. I think these are punctate lesions, focal lesions, as mentioned 514 information. You see dermis sloughing off. Never seen any of these typical punctate wounds 516. Actually, *Linda [Munson] or Richard [Montali*], you have probably got a lot of pictures you can show of that . Do you have any punctate lesions?

Munson: The vesicles or little...

R. Kock: The little sort of focal...

Munson: I do not have one.

R. Kock: You do not, well...

Munson: I was minimizing my slides so Eric [Miller] would not yell at me!

Montali: The reason why I asked, is because I think we are trying to focus in on some of these things that have been putatively related to stress. Now, here is a good example of physiologic stress, and you are not seeing some of these things. So, it is hard to sort out.

R. Kock: Well you see, maybe later on they may develop something.

Munson: Yes, but what is the time frame? That is really an important thing, is how long were they stressed for and how long do you follow them for?

R. Kock: They are followed continuously.

Munson: But, the stress is...

R. Kock: Four weeks.

Munson: Four weeks, that is fairly a long time.

R. Kock: But, then afterwards, nobody has called us back saying I have found these skin lesions.

Montali: And, they go on to, I mean they usually recover and they get better?

R. Kock: Yes, I mean basically the skin 535.

Montali: Yes, I understand that.

R. Kock: But, the animals that have been stressed I think generally speaking have not have many problems. I mean observing stress related changes: depression and weakness from the boma after the short boma period. But generally with good nutrition and so on, they recover pretty well and settle down. So it may be that the procedures are good and therefore 541 translocation 542.

Morkel: I think the stress period for you, you know four weeks, it is usually one week at that, 543, it is obviously pretty minimal stress.

R. Kock: *Yes, I think that is very true.*

Munson: What is the distribution of these--all over the whole body?

R. Kock: All over the whole body. You can see these little circular things all over the whole body. And you will see some epidermis still left on them, but actually they develop... But, you know, all over the whole body.

Munson: Is this a captive one?

R. Kock: Yes, it is a captive animal.

Jessup: But, you've never seen this in free-ranging?

R. Kock: No.

Jessup: Has anybody seen it in free-ranging animals?

M. Kock: No, we have had a wild caught animal that had been in the boma for a period develop quite deep ulcers or skin lesions.

Jessup: Like this?

M. Kock: No, big large lesions like that that were raised, with a *hypotic* center and on the sides of the chest. We just treated it symptomatically and then it eventually disappeared.

Munson: And how long had it been in the boma when this happened?

M. Kock: Two months. It was not particularly stressed, but none of the other animals had it. We have not seen any other, I am not sure absolutely though.

R. Kock: OK, just coming back to this. Certainly in recent years disease related problems do not appear to be fairly significant. But, the fact is what *probable* mortality there is, people do not just find a carcass until quite late; and therefore who knows, there may be something going on. Kenya is very bad for disease--everything. You know the epidemic diseases there are terrible. So I often wonder. Fortunately it is a perissodactyl and one guesses less likely to many ruminant diseases. Things like rabies, I am waiting for my first rhino rabies case! Because it is a really bad 578. So I think we must be prepared for something at some point, one of these *long-term things*. It is surprising nothing has happened 580.

Snaring, I think this is our biggest threat. This is just meat poaching snares, buffalo maybe. I have had to take off some. One case in the last year around the head, was actually affecting 584 of its mouth. The best part, it took a long time to get it, nearly half the night. That is quite a fun story, but I will leave that for the bar. I think that is going to become more and more important, because of all of the [human] population growth, and all of these sanctuaries have a lot of people around the edge, et cetera, et cetera.

Interspecies aggression, this is definitely our most concern with movement of animals around these smaller sanctuaries. Carrying capacity problems where limited resources are, like a salt lake at the *arc* and *tree tops and* 594. The number of rhino coming, you get nine or ten rhino coming at one lick at one time, plus 30 elephant, plus lion, plus all sorts. These animals have a terrific amount of aggressive behavior. And we kept getting these young animals with occurring skin problems and 600.

Tolt: Rick, that is why I do not understand why you prerelease these animals. The one reason for keeping them in the bomas obviously is monitoring 603. The other reason and there is no doubt it works, is to set them in the immediate areas, so when they come out, they are familiar with that area and they are used to the browse, the smells and everything like that. And, they do not want to part. 607 animals translocated to *Chewore* and it shows *absolutely convincing* the value of sticking them into a boma in terms of the distance that have moved for release. I will report in a sanctuary type situation, the animals are unsettled at prerelease and wandering about bumping into other animals. Your risk of interspecific aggression in this situation is a considerable risk.

R. Kock: I mean, I am not the decision maker on that. I agree with you. I think obviously at Tsavo we could obviously put them into bomas before release. There are no rhino there we could worry

about. But, that is the *reason for getting them used to the browse*. Where in sanctuaries, we just tried a release, we prereleased six at a location where we lost two from intraspecies aggression. I thought well, lets do this whole group together including some big bulls, a couple of really big bulls. A couple of things could happen. The aggressive bull might end up fighting and you could lose one. A chance that you could lose the green bull on that place, but the genetics will be improved. Swap over 629 bulls. That was one thought, sort of cruel, but we will see what happens. It is still a matter of I guess the more it is done, the more information. Maybe we will go your way and we will start putting in bomas for all the sanctuaries.

Morkel: I think with that bull and in that initial sorting out, or that sorting out, it is not just a question of the first two weeks. I mean that sorting out you have got for six months to a year, that you sort these animals. And I think that one must consider those animals which die a few months afterwards are still a part of that whole...

R. Kock: That is why we talk about six months translocation length. The other thing we are trying to do is 641 *to mix* young age groups. We are now trying to go toward mothers with three year olds, and try that, obviously. A nice strong aggressive mom with a three year old. Because that little group might survive better, than just dumping...

Jessup: It is kind of obvious, would not something as simple as putting out some blocks of salt, in some separated areas reduce this concentration more?

R. Kock: Yes, it is being done now, using salt licks.

Stover: Do you provide salt licks in the boma?

R. Kock: Yes, we put blocks in.

Stover: Do they lick them?

R. Kock: But, it is not a major concern in the boma. They like salt.

Morkel: In 657 we are at a stage now where when we introduced some new animals into the sanctuaries we identify in that sanctuary where there are gaps, where there is not too much pressure, where you can bring in a new bull without too much problem. And you do place your boma there, let your animal settle down there, release there, and hoping it stays there, at best that is where it will stay. You move your boma to where you want it to go.

R. Kock: I think it is going to become increasing 665 for intensive management in the sanctuary. I think it is getting more and more sophisticated 668. All the males, you know you have got a situation where you either have to move all the males out and put one new male in. Because your chance of a male you put in there surviving is very remote. And they are all related, the whole group is related, very close--the mother, father, daughter and so on. And you suddenly have this real conflict of interest. You know, you think what can you do? Where do you put all of those males? And maybe we will end up with bachelor herds here, actually in sanctuaries.

Morkel: *Who had some*, Richard?

R. Kock: Yes, there you are. I just thought I would throw this up, because I had this review... Again, just to show the difference in whites. This is sort of lumped in sort of general physiological areas, rather than 687. But, accidents in captivity reported. As it is in wild situations, in game management, there are no excuses for poor management. And then in whites the same as blacks. But, you do not get this sort of blood disorders and so on in the whites. But, you do get gastrointestinal disturbances, and that may be related. It *can be too, perhaps concentrates in the feed* or something. But, I threw this into show it is a very dynamic situation. This is the one that was done through San Diego. This reminds people that Zimbabwe had half the population in 1991 and we are now talking about something like this. This is very very dynamic and we must keep that in mind, and certainly 704.

END

PATHOLOGICAL CONDITIONS IN FREE-RANGING BLACK RHINOCEROS

Nancy Kock

BEGIN TAPE 4A

N. Kock: ...If you have got an open wound, that flies are attracted to, that is 001.

R. Kock: Why?

N. Kock: Well, I do not know if that they do clear up 002. In your experience...

R. Kock: All up.

Munson: Are the flies gone, are you saying the secondary flies?

R. Kock: Yeah, the flies are not there.

N. Kock: Do you have nematodes in the lesions as well?

R. Kock: You got the tissue, what did you find in the tissue?

N. Kock: You sent them to me?

R. Kock: No, I have not sent them yet.

M. Kock: Well, I think Nancy, there is no doubt that in the Zambezi Valley, that those rhinos that were further towards Mosambique where there was more water and plenty around *and indeed moisture* there, [the lesions were] more aggressive compared to the animals near the escarpment. It is definitely related to fly density.

N. Kock: Yeah.

M. Kock: I do not think it is 011. I mean, if a rhino wants to rub something, he will rub it. These lesions... I think it is just a localized reaction and does not seem to bother them at all. And they vary, some that are next to a river always have water on them, they are very extensive lesions. And when we translocate them to the valley and the high veldt, they dry up, they scar over. *I do not think there is any in Namibia, have you seen any in Namibia?* Have you seen many?

R. Kock: How quickly *do they dry up*?

M. Kock: Well, it is kind of hard to ask a rhino that. I do not think we have had an opportunity to knock a rhino down a few weeks after we have moved it, but *in the boma it is pretty quick.*

Montali: Is there relief from fly biting? Do you have flies in the high veldt? I mean there is one difference if you do not have the density of flies.

N. Kock: But, we do not really know exactly what the fly is. We may just be moving them out of the area where 019.

du Toit: Nancy, you will see clusters of flies around the lesions 021 humid areas of the Zambezi Valley. You see those types of flies. You see them 022.

M. Kock: 022 *classic* because of these rhino, with just masses of flies around these lesions.

Morkel: It would be interesting just in a fly area, animals set in a boma in a fly area, just to treat the lesion with something to keep the flies away or knock off the flies and see how quickly it heals.

du Toit: The medicine does it very well. And in fact, Daphne Sheldrick uses it on her 025 uses *it actually* on the wound, and what is interesting I guess is that it does prevent the fly.

R. Kock: It is sulfanilamide powder with a potent 027.

Montali: Is there any attempt to treat the parasites just systemically? To treat the filarial parasite through the skin or is that not...

R. Kock: We have never tried it actually.

Montali: With Ivermectin, or...?

030-032? MIXED INAUDIBLE COMMENTS

N. Kock: OK, this is another condition that I was struck with in two black rhinos. The first one was actually not my discovery, but was something that was discovered before I went to Zimbabwe It was thought to be some 035 condition. And then the second one came along, actually one day 036 an animal. Thank goodness he was there, because he made sure that the entire part got back to me. Then I went back and looked at the other part which was still at our *membrane* research lab, and found that in fact that this was now two cases of exactly the same thing, and that is coronary artery aneurysm. That is a fairly uncommon finding in most species. Turkey get it, but in most other species you do not really find aneurysms. Predisposing factors in humans is probably *arteriosclerosis* more than anything else, and arteritis as well. In the horse and dog, this can go secondary to nematode parasites; in the horse *Strongylus vulgaris*. and in the dog 044. And in the black rhino, it may be parasitic as well. I had one acute case and one chronic case. When the acute case died we did lots of serial sections hoping to find some evidence of parasites. but I unfortunately was not able to find one.

The first case was actually the chronic case, this was not 049 myself. And this animal had a history that it had been put in a boma, was showing just some general signs of 051 and probably resulted in heart failure, although that post was not done by me. The heart was just basically chopped out and sent out to the Vet Research Lab; unfortunately, without other tissues that one would have to cooberate heart failure 053.

The second case , which was the one that occurred while Mike [Kock] and *Dave* [?] were *translocating an animal into* a boma and it died. And when they looked, they found the animal had 056 because this aneurysm ruptured 057.

R. Kock: What age were they?

N. Kock: I do not know what the age of the first one was, Raoul, you might know.

du Toit: It was not that old.

N. Kock: No, that is what my feeling is. Neither of them were very old animals in age. The second one was a pregnant female with a young calf, unfortunately a near term young calf. So it was not that old, definitely in reproductive age.

This is the coronary artery being opened up--with this very large basically blood clot or thrombus sitting inside of it. You can see it there, it is enormous. It is an unusual lesion, I have looked for it again, obviously since I have found these two cases 066. Histologically, this is the active case. A large thrombus in the center of this very ulcerated 068 surface. It is very inflamed, you can not really appreciate it to look at this. There are eosinophils in here which lead me *to believe it possibly was a parasite*. In the horse you might get this little *granular* 071. Usually it is more vasculitis and arteritis, as this actually is. And in the dog it often occurs in the aorta. The coronary artery is an unusual location for either of these two parasites 072.

In the chronic case, we had a rather huge lesion with a bit of *osseous hemoplasia*, deeper, below the 075. These were the 075 lesion, you can actually see one 076, or feel it actually as you 076.

Montali: Is *Strongylus vulgaris* an indigenous parasite in free-ranging rhinos?

N. Kock: I am going to get to some information on the parasites we have taken off 080 later on. We do get strongyles, but I do not know if we have been able to identify what exactly it is. Since that time, I have been looking in arteries to see if I can find...

Montali: You have not seen any mesenteric, any old healed...

N. Kock: No I have not. But, as I say, you often do not get the whole carcass if it is a large animal and they have to transport it out. But when I have been able to, I have looked for it. This opportune ulceration has been another finding that has been quite common. It is a little bit unusual because it is midesophageal, and they are really sort of like button ulcers that you might see in the intestine of rhino 087. But they are not really necrotic, they are quite glandular, they are obviously ulcers. If this were some caustic agent that was causing this... I mean I also think it can occur for a few different reasons. It can either occur from the inside out, whether something damages the surface and 091, or you can have a 091 lesion where there is 091 infarction and ulceration 092. If you had some caustic agents that then were ingested, you would expect an 093 lesion 093, and this is not the case. If this were a stress related lesion, 094 lesion, you would expect to have gastric ulcers, and also you might expect to find more 096-cardia rather than esophageal lesions.

Histologically, these are extremely bland lesions. They do not have a vascular base. And this is what they look like. This is one edge of one of these ulcers. There is not really very much 100. There is not vasculitis, there is not any thrombosis. It does look like something has just damaged that to cause the ulceration. 102 here and here there is a little bit of inflammation, but not very much though, very little. One thing I thought it might be, after having looked at a few, is that rhinos do get bot flies in their stomachs,

similar to horses, and sometimes you will find them in 106. But, in all the cases I have seen, I have never really found bots in those animals, but I suppose that is a possibility 107.

Bolin: Is there something about the midesophagus of the rhino? Is that at the thoracic inlet or something where you could postulate ingesta coming down and suddenly gets constricted there, and just physical damage? Is there something about this location?

N. Kock: Well, I would not say it is just one spot, it is sort of not up here and it is not down there, you know, it is somewhere in-between.

Montali: You do not see much inflammation in the base, and there is not really any 112. So this is a fairly recent occurrence? Because it is traumatic, some sort of a passage...

N. Kock: I suppose it could be. But, it is unusual because they are all sort of oval to round and they are not...

Montali: So they are multiple, they are multiple small...

N. Kock: No, they are not that small actually, they might be...

Montali: Linear?

N. Kock: No, not linear, kind of oval...

R. Kock: Are they dry season or wet season?

N. Kock: When ever I get them!

Montali: What is the incidence, are you seeing a lot of these, or just...

N. Kock: I have seen enough of them to make me look for them. Again, some *of the postmortems in the field* 119.

Montali: They do not open the esophagus too.

N. Kock: And they *often do not open the gut, just pull it out* 120.

Jessup: Because these animals usually die from other causes than the lesions.

N. Kock: This is something I have seen, and I will talk about this later. I have seen with animals that have 122. It could be the reason that there is not very much inflammation is because perhaps 124 the animal is stressed and there is not much of a *blood supply.*

du Toit: Specifically, which animals had this, animals only in the bomas, adults, animals 126?

N. Kock: The animals from 127 had this, that is some of them. The animal that died in the low veldt, and was close to 129.

du Toit: Was it recent?

N. Kock: No, subsequently he has died 131. I would not say it is every animal, certainly not, just enough to make 133.

Harvey: What is the location of the... There are steroid induced ulcers in some of 133. Where do they show up?

Montali: They are usually gastric and usually towards the pylorus.

Paglia: Have these ever been seen in any of the captive animals?

Miller: I am not aware of it, but I know as you were talking, I am not sure the esophagus has always been fully opened up.

Bolin: You just insulted every pathologist here and veterinarian! Do you want to take another stab at that?

Miller: Certainly we have seen in animals stressed and getting agonal disease, different from what you are describing, but gastric ulcers. We do see what we presume are stress induced ulcers. Enough that on any animal that we are treating we are just recommending 144 or some coating agent. But we are not aware of any esophageal ulcers that are reported.

N. Kock: I just think they are unusual.

Paglia: None of these has a granulation tissue base, so there is no 146.

N. Kock: A little bit, I mean there probably is a little bit of 147 starting in here. But basically they are fairly recent. They are not 147 they are not *teeny weenie*.

Paglia: I just recall seeing this picture in one of the National Geographic articles of a rhino delicately eating this very thorny piece of vegetation. I wonder if...

N. Kock: Yeah, the only thing that bothers me about that they are sort of all the same size. They are sort of round to oval. They do look to me like bot flies maybe hatched there. They do look to me like that.

Montali: Yes, that sounds like 152.

N. Kock: But then I look and I can not find them. And it may be, I mean I am not really sure, I know if you have been treating animals for parasites at some stage... And it may be that these animals have been treated and then 154 and then have gotten them. Sometimes I do not have the best history, in terms of complete medical history.

Stover: So these animals 156 free-ranging, captive?

N. Kock: Well, some had been translocated for some time. Not from bomas, they had been in bomas, but then they had been moved out. One in particular had been translocated for more than three years.

M. Kock: I think Nancy, one common theme there is 158-162.

Montali: You think they are incidental findings basically.

N. Kock: I think they are incidental findings, definitely compared to 163. Here is another incidental finding, it is just one animal, but I thought it was quite interesting. We did vaginal cytology on this one 166 translocated female. And one of them had quite a dramatic *colitis* also some 168. This sort of looked to me almost like *a viral lesion* 168 a bit vasicular. But I do not think 170 *more chronic than it appears to be* 170.

Munson: Was it at the mucocutaneous junction or was it inside the vulva?

N Kock: I will show you. This is a normal animal with some *ticks*. If you opened that up, just to show you the normal mucosa. You can see here a bit of ulceration and actually that does not show it as well as I thought it might. But some of the areas actually look like their vascular regions are more 181.

R. Kock: Where is the tail?

N. Kock: I do not know!

M. Kock: This one did not have a tail.

N. Kock: That is right, there is a lot of animals in Zimbabwe without tails though.

R. Kock: You know without a tail there, it is very exposed.

N. Kock: That is true, except that it does fold right in. It is still quite protected. Now this next condition is something that I do not really think is probably as incidental as I first thought it was. At first there was only one animal, then two, then three. At this point there were only two, but there are three. Two adults, not particularly old animals, had striking fibrosis around the Purkinje fibers of the heart. In neither of these cases, I know the histories on both of these cases, was it approximately or directly related to death. But Raoul [du Toit] has talked about some die-offs, and I have tried to look into what might cause them. There is an extremely small amount of literature on these kind of lesions. But, half of them are exposed to 197 and have this lesion in part. I think that is a bit unlikely where we are. There is also a plant, called *Digari asparagus* in Zimbabwe that has been associated with similar type lesions in skeletal muscles in cattle. It maybe that this lesion is possibly related to plant toxin.

Munson: Have they identified the toxin?

N. Kock: Yes they have. Unfortunately I did not bring much with me. But there is a very good South African book called Plant Toxicology and it is written up in there. It is not described as a cardiac toxin, but it is striated muscle, it is skeletal muscle. One of the toxicologists at the university feels that perhaps it could also cause a similar lesion in the heart.

Montali: Also, even copper deficiency in cattle can cause cardiomyopathy with fibrosis and cardiofibrosis.

N. Kock: These animals do not have endocardial fibrosis. I will show you come pictures of it, but it is striking and it is just *very* Purkinjeal. I think I have one acute case where you can actually see Purkinjeal degeneration or 213.

Q? 214: Which of these animals were 214?

N. Kock: I have the histo results, but I do not have them with me. But they were just an incidental finding, in addition to the 217 lesions. Now, this is a normal Purkinje fiber, it is just a modified cardiac fiber really, which is involved in impulse production and heart beat. As you can see they are usually quite closely opposed to myocardium and myocardial fibers. This is an early case. Here you can see the color change in this. This is just a hematoxylin-eosin stain. There is a little bit of vacuolation here as well, and a little bit of color change. When the tissues take up this eosin stain, it often indicates there is some

membrane damage and degeneration or necrosis of these fibers. I think this is quite an early change. Another indication here looks like this nucleus does not look particularly happy. The fiber is a bit shrunken and the nuclei are a bit small, a bit vacuolated, compared to these which obviously look a bit more happy. This fiber is probably not viable.

R. Kock: In humans with 231 circulation problems, do they develop any 232 changes?

N. Kock: No, that affects usually just myocardium and areas of the myocardium, basically the coronary arteries and *endocrine* system. So, if it is blocked, the tissue beyond that dies. And it is everything that dies, it is not just Purkinje fibers. This is quite an interesting lesion. And here is a later lesion. As you can see here now, rather than these Purkinje fibers being directly opposed to the myocardial fibers, there is all this fibrous tissue in-between. And just to ensure it, this is a trichrome stain, which stains for fibrous connective tissue; just showing that actually is fibrous connective tissue and that is even sequestered in the myocardial fibers as well.

Morkel: How would that affect the function of the heart?

N. Kock: Well, it is hard to say, it really depends on where it would be located. You do not have to have a very big lesion in the heart for it to cause the heart to stop beating. I mean, any lesion in the heart can be significant. In these particular cases, I do not think it was related to death. One of them was fighting. One of them actually was that bull that you talked about, that was definitely a trauma related death. I can not remember, but I have seen this now, and I think all of these animals did come from the Zambezi Valley, these are all the cases.

Munson: Nancy, is this in any way related to what you have seen with the elephant trunks? A neuropathy perhaps?

Paglia: There is no interstitial fibrosis or subangiocardiofibrosis?

N. Kock: There is no real inflammation and there is no myocarditis.

Paglia: It is just all 256.

N. Kock: No, it is just anywhere.

Paglia: Anywhere you find the Purkinje fibers?

N. Kock: Right

Citino: Encephalomyocarditis virus can sometimes selectively pick out the Purkinje fibers. I know in other hoofstock in particular, when there were very mild cases you could selectively just find Purkinje fibers taken out of animals acutely dying, just due to cardiac dysfunction of the Purkinje fiber. In the US here we have had a few cases of black rhinos that have died from encephalomyocarditis.

N. Kock: Well, there is another possibility. I know Raoul [du Toit] was talking about die-offs, and unfortunately I have never been around any die-offs. I think a lesion like this is certainly a possible *candidate*.

du Toit: How quickly does a thing like this develop?

N. Kock: Well, fibrosis is something that... you know, it depends on how severe the lesion is to begin with. But the fibrosis can start within a couple of weeks of there being some damage. It would be very hard to say.

Paglia: Those look pretty old. 274 have very small nuclei. It looks like it is beyond the healing stage. This is not...

N. Kock: No, I think this is a healed lesion now, but I am saying the fibroplasia could start quite soon after the damage done to the fibers. Then again, you do not know, an animal could live quite happily with this too. It just depends on where it is in terms of the production of the heart.

Montali: I think clinically you would expect dysrhythmia, as you would expect... which would eventuate in a sudden death situation.

N. Kock: Yeah, but the thing I did not find in these animals though, is any evidence of heart failure, congestive heart failure.

Montali: Well, it would not necessarily be heart failure.

N. Kock: No, that is right, it would not. So I think this is, at this point at least, not leading to heart failure. But it is not to say that it could not result in acute heart failure at any given time.

Another thing that I did... We collected feces from 290 animals, and just had a look to see what they were carrying. I do not think these are 291. This is what is seen and they can vary quite a lot in a clinically normal animal. About 80% have some type of nematode, things like 294.

I will talk a little bit about *parkwater*. I do not know if *parkwater* is a clinical disease problem in black rhino at all. But, I think they may get exposed to it. I tested both black and white rhino for presence of antibodies to 300 to *parkwater*. I am using quite a specific test, so I am fairly satisfied with the results, as well as cross reaction between 303. But this test which we run 304 is fairly reliable in other species. And I found about half of them from the lower Zambezi Valley were positive, but only one from another area. I will show you on the map. The ones that were positive came from this area of the Zambezi Valley, and the ones that were largely negative were from this area in here.

This is a tick transmitted disease, by *Amblyomma* ticks. And there are two main *Amblyommas*, 312. And it is interesting that both of those ticks were found on the animals in this area, where there was positive titers to *parkwater*; and only sparsely down in this area. So it may have something to do with the *transmission of the* disease and distribution. It is interesting also that white rhino are about 80% 319 tested positive for this area here, Hwange National Park, and those are tested quite often. 321 transmitted this disease to cattle. I think this is important, because it is beginning to be felt that carrier stages of this disease is probably more likely the rule than the exception. And if we can actually isolate the organism from this 329 important in this area 329 translocation 329. Particularly 330 area where there might either be a tick that is capable of transmitting this disease, or if ticks happen to go with the animals and are then able to 333.

Now, I am going to just talk quickly about something that is not a problem in free-ranging animals, and certainly appears to be normal activity. I did a retrospective study on about thirty animals from Zimbabwe, and looked for the lesion hemosiderosis as an indication of hemolysis. What I found were that free-ranging black rhinos, and these were animals that had died say within two days of capture... One animal died 341. Another one had degenerative horn disease, and was unable to be managed in the boma. These animals I used as reference levels for degrees of hemosiderosis. The one animal with degenerative joint disease was quite old 346. I used that as a reference level 348. What I found was that these animals that were free-ranging do not accumulate this, and so then I extrapolated that to say that they probably do not get *hemolytic* anemia or chronic hemolytic anemia in their natural setting. Then about 73% of translocated animals have excessive and often widespread hemosiderosis, and this seemed to worsen as time went on. 356 I did find that some habitats seem to be less affected than others, and I thought that that was probably related to nutrition. That was just my feeling, I have no real basis to base that on. Because some hemolytic hemolysis can be triggered by stress, I have a feeling 363.

I will show you a few slides of that. This is a spleen 366 position; and this is a normal spleen in an old animal, the animal that died of degenerative joint disease. And you can see there is relatively very little 370 *lesion*. This is lung, this is a Prussian blue stain just to show the blue stain of hemosiderin. This is liver, and quite a lot of hemosiderin 380. In the intestinal tract often you find a lot, it is widespread, as I said. Again Prussian blue on some of the intestinal villi. This is an adrenal gland.

And then I went back and looked to see if I could find other things that went along with this. One thing that seemed quite obvious was lymphoid atrophy in spleen, and lymph nodes and 390 and gut associated with lymphoid tissue. Two fetuses had what I would define as hyperplasia. Poor body condition also seemed to be quite common in these animals, and I ended up thinking it probably had a lot to do with stress.

Smith: Did you say you saw it in fetuses?

N. Kock: In two fetuses. And this is a lymph node from an animal with hemosiderosis. There is a few what looks to me like 398. This is the spleen, again a very prominent spleen muscle framework because there is relatively very little 400 in there, as opposed to a normal spleen 401. This is not a particularly good slide, this animal has been autolyzed; this is the thymus of an animal, a neonate. There is not very much lymphoid tissue left. I also find a lot of gastric ulcers sometimes and gastric 408.

Septicemia, again, this is not something we have found in free-ranging animals, but it has been a cause of death in animals that have been caught. In one case we had an *occasional* septicemia as a result of *Streptococcus equisimilis*. In several other cases we have isolated the same organism from dart wounds. The first animal, this was before the use of these long-acting tranquilizers, had bashed its horn in and had quite a severe infection of the nasal bones which tracked all the way up basically to the cranium, it did not go and pass through. But, in this animal I recovered this organism with heavy growth from the nasal

discharge, from the tonsils, from the liver, from the mesenteric lymph nodes, and from 432. This is an organism that is specifically transmitted from horses *which may not be too surprising, because they are both* 433. And this was again recovered from infected dart wounds in several different animals.This is not the animal unfortunately, I could not find the animal that had the fractured 436, but just to show when these animals knock their horns off, how damaged the tissue can become. This is some hemorrhages on the animal that died of the septicemia. And a dart wound from which we *recovered* 445.

The last thing I wanted to talk about is the possible creosote toxic issue. It has been quite a controversial one. Because, as it had been pointed out, for political reasons we had animals that we moved into creosote treated bomas and unfortunately they had to stay there for quite a while. I am not exactly sure how long, but after some time, two of these animals developed jaundice, subcutaneous swellings of the 452, just on one side or on both sides and anemia after they had been housed in these bomas. Both declined in condition and the one died and the other one was euthanized 456. Both of these animals were anemic with hematocrits of 18 and 25. Both had increased liver enzymes, both had extremely high *albumin* which were a combination of unconjugated and conjugated.

M. Kock: Nancy, can I just say that the phosphorus of these animals was extremely low.

N. Kock: They were extremely low, but it seems to me there was some problem at first. I did not mention it because I thought there might be some *problem with those*.

Jessup: What, laboratory analysis problems?

N. Kock: Yeah.

Blumer: When we get to the other ones, you will see we have consistent findings in a lot of these animals.

N. Kock: Very well. Grossly, the most dramatic finding is an incredibly bright green 470 just quite large livers and just absolutely diffuse with bright green. They had hemorrhages, swellings in the arms were result of hemorrhage and the hematoma, into the lungs, into the wall of the bladder, scattered across the serosal surfaces. The gums basically had bleeding ulcers and were 477 in both animals. One animal had been passing dark brown red urine and we suggested that from the basis of hemoglobin vs. 480 hemoglobin. There was not any ulceration of the bladder, so I did not have any *blood in the bladder*. There was not a lot of 483 bladder wall.

This is just to show a normal rhino liver--it is usually quite blue, quite a lot of 488 *lymphatics* on the surface. And this is the crummy looking liver from the animals that died. This is the cut edges, actually they look a bit blue to you, but they were extremely green. Hemorrhages into the lungs were quite extensive in some areas. And these are the hemorrhages and also showing jaundice in subcutaneous tissues into the forelimbs and shoulders. Again, more hemorrhage into the same muscle masses of the forelimbs. Serosal hemorrhages were scattered everywhere. And then microscopically, I described the lesions as 500 of hepatocytes, and this was due to the accumulation of bilirubin. We did several sets of special stains on

this and this was... You can use special stains to differentiate between bile and to *remove bilirubin*. While there was certainly bile in the hepatocytes and 506 lots of bilirubin and a small amount of iron, but nothing like hemosiderin. The animals also had bone marrow hyperplasia which goes along with the hemolytic response of anemia. I suspected that they had hepatopathy and also 512 as well. Then afterwards we found that the animals were mistaken and sent from 514 to Australia 515 lesions and died.

du Toit: Plus, Nancy as well, four of the five rhinos sent from *Boldin* from the Soil Conservancy to the *conservancies* were lost, died.

Jessup: With the same lesions?

du Toit: Yeah.

N. Kock: I think there was only one postmortem done.

du Toit: 521-523

Morkel: Nancy, that early report in the Journal of Zoo and Wildlife Medicine, was that *tissue* involved in that? I can not remember.

N. Kock: In?

Morkel: Creosote poisoning in the black rhino

N. Kock: No, it was a suspected form.

Morkel: OK, so there is no imperil.

Montali: Yes, *Schmidt's* article. There were two cases, they did not see any tissues. They could never recover the tissues, fresh tissues.

Morkel: Where the slides the similar to these?

Montali: I have seen the slides--they are exactly the same.

N. Kock: You do not mean the old one, the one 536.

Montali: 1981 I think. Two black rhinos that had been at the San Antonio Zoo for about 10, 15 or 20 years. Is that the article you are talking about Nancy?

N. Kock: No.

Montali: I would like to hear about the other one then.

N. Kock: The other one was just a suspected case. And this is one of those special stains showing just tremendous looking 542 hematocytes. Creosote is not a single compound, it is a combination of 546 basically. It is quite toxic to pigs, and causes actually the same lesions and hepatopathy and anemia. It also has been reported to result in reproductive problems with stillbirths. It would be interesting to see if any of the surviving animals have this lesion. It has been mentioned here why some animals and not others. I think a lot of that might have to do with the age of the animals, some animals rapidly can get it by rubbing it and get through abrasions in the skin. And a couple of these animals had some abrasions. It can be inhaled, can be ingested. I think it is possible that the differences in the different bomas could have resulted in some animals just being more exposed than others.

I was just going to say that all of these features and interesting things I found probably really do not have very much to do with the real situation with rhino. Poaching is probably the worst problem in free-ranging black rhinos. And I would just like to thank the organizers of this conference for getting us all together to try and solve the problem.

Montali: I would like to just ask one question--how long were those two in the bomas, what was the period of time they were in the bomas possibly?

M. Kock: Three or four months. They were animals that we moved in, Evan [Blumer] was there. We moved them into the bomas at *Boldin*. They were going to be in the bomas for approximately two to three weeks I think and then they were going to be shipped to the United States. But, then getting close to Christmas, all the planes had been booked from 580, so the rhinos stayed until I think the following June, July?

Blumer: April.

M. Kock: And these animals... I mean this is something that when we are in the working groups we need to look at very carefully, because we followed these animals from the time we caught them to the bomas and they developed these 585 signs. And even the ones from Australia for example, no indication whatsoever in these animals was there ever a problem. For example, one bull that died on the 588 Islands was quite normal when we bled them. All the blood parameters are normal--I have them here. I think the stress of loading... It was a difficult animal to load, it would not cooperate, so a little stress at loading. Then 591 and then we noticed was quiet and suddenly it went yellow, and I do not know how long it survived there. But, there was no indication ahead of time. So they obviously maintain themselves *with the damage* there and then suddenly the signs develop. It is not like it is a gradual build up.

N. Kock: The other thing we asked, when we bled the two animals that were sick... we bled other animals too, and the others were slightly jaundice, but they then seemed to get better, because we followed them for a while.

Miller: I just wanted to say too about the analysis of the creosote, it often has other contaminants. We were able to get it analyzed at *Monsanto*. It appears that if you bought creosote, it was just creosote. It was relatively pure creosote. it did not have PCV's or Dioxin. And so often what are misdiagnosed for creosote poisoning... It is often a *contaminate quite a bit*. This does appear to be fairly pure creosote.

N. Kock: And not very toxic?

Miller: No, creosote itself can be toxic. It is just that now we know we were not dealing with Dioxin toxicity.

Jessup: Now we are only down to 15 compounds instead of...

Miller: So often creosote can just be... Well, we know what dioxin does in horses. And so we are able to take it back up and just looking pretty much at creosote, the "mish" that it is.

N. Kock: Just to finish, I would just like to thank the University of Zimbabwe Research Forum and help in funding while we investigate these things and Department of National Parks and Wildlife to permit us to carry out these studies, and National Wildlife Veterinary Services who always support us financially, as well as spiritually. And also all of our co-authors 624. Thanks very much.

END TAPE 4A 626

ADDITIONAL COMMENTS:

Paglia: When we sent out the note to indicate that the *compound should* be avoided, because we were concerned that related compounds at this stage that the hemolytic tracer was in G-6-PD deficient people. That was in February, right during that period of time. I recall when you got that note and you went down and looked at those animals, that five of them had signs of jaundice. Is that correct? You looked at their mucosal surfaces or conjunctiva, that there were some signs that they were...

du Toit: Yeah, Mike and Nancy were away at that time and Chris 640 and the other vet were becoming concerned about these animals. And I took it 642 till you have seen some of those animals 644.

Paglia: They had no other signs than that, but they were showing some indication 647 were up, on what ever basis. We were not thinking of creosote toxicity as the distinct enemy 649, only because those *aromatic hydrocarbons were among* 650.

Bolin: What year was that?

Paglia: This was the year the animals were here. A group of 20 was captured 653.

du Toit: Just before we move off the things that have been seen in animals in Africa, I think we may have given a slightly misleading impression of the occurrence of skin ulceration. There have actually been a fair number of cases of skin ulceration other then the filarial lesions that have been reported. I can think of four or five in Zimbabwe, mainly in boma situations where nutrition is poor. In one case, where an animal had died, he had also shown a regular and strong suggestion of a hemolytic crisis. But, *very severe* ulceration, and I actually sent *Linda [Munson] the information* I had on that some time ago. Since then, there has been another case reported in South Africa, animals moved up *to our Wilderness* Area. The report on that introduction commented on these raised ulcers that appeared on the skin of white rhino. So it is something that has appeared in a 678.

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