In the first place, it has been said that there is a genetic susceptibility to seizures. If one examines the families of the individuals who have post-traumatic epilepsy or seizures in the siblings and parents, the incidence of seizures is not particularly higher than in the families of individuals who have uncomplicated head injuries. Only in the group of post-traumatic epileptics who have generalised attacks, and generalised abnormalities in the electroencephalogram is there a significantly higher incidence of epilepsy in the family. The likelihood of genetic factors is real but apparently does not play a very significant role in the determination of post-traumatic epilepsy.

There are a number of other important factors. These may be described under the general heading of severity of injury. In closed head injury, that is those cases in which there is no violation of the continuity of the duramater, the likelihood of a seizure occuring is relatively low and correlates with the period of unconsciousness which accompanies the injury. The longer the unconsciousness the greater the chance of epilepsy. The time of the follow-up examination is another factor. In closed head injury within the first month there is a relatively high chance of seizures developing (1-5%), but after that the risk of an epilepsy developing is slight. In fact, if an individual has no evidence clinically of neurological impairment after a head injury and has a normal electroencephalogram, one may say with reasonable medical certainty that if an attack has not occurred by one month, that individual has no more chance than any one in the general population of developing a seizure. The prognosis after an open head injury in which there has been a violation of the duramater, is quite different. There is the same high incidence, initially, of post-traumatic epilepsy and as I mentioned, in the first week there are a considerable number of patients who will develop seizures — somewhere around 20-25%. At the end of 3 months the risk has considerably decreased; after six months it has decreased still further and at the end of two years is about 5 to 10%; over the next eight years there is a diminishing likelihood of approximately 1% per year of the individual developing a seizure. This means that in the event of a closed head injury, the likelihood of post-traumatic epilepsy is very slight after the first month, but with an open head injury with damage to the cortex the risk is relatively high for two years and then it gradually decreases.

There are a number of other factors which will increase the risk of post-traumatic epilepsy. One is the location of the injury. Wounds which involve the poles of the brain—for example the frontal and occipital regions and even the temporal region have an overall incidence of post-traumatic epilepsy of about 15-20%. But those wounds that involve the central region have a 50% risk of fits developing. This relates to the fact, which may be demonstrated experimentally, that the epileptogenicity of various parts of the brain is different. It is relatively low in the occipital and frontal regions and very high in the central region. Thus we could expect that there would be more epilepsy with central wounds. Why don't we see more epilepsy with temporal wounds? The reason is that the medial portion of the temporal lobe is not primarily damaged. The traumatic lesions are at the tip and along the lateral surface of the temporal lobe with sparing of the medial parts, or if they are damaged, the wound is fatal. I think this is a very important consideration to keep in mind.

In addition to the location, the depth of the wound is important. Wounds of the head that involve the scalp carry a relatively low risk of an epilepsy developing. It varies in different series from 2 to 15%. If the wound however involves the duramater the incidence increases to the range of 20% and if it involves the brain substance—the cortex, the incidence is 40% or over. This merely reflects the fact that wounds of the cranium do not cause much damage to the underlying brain, whereas when a missile or a blunt object penetrates the dura, the brain is extensively involved. As a secondary factor, infection will increase the likelihood of epilepsy developing.

Whereas in closed head injury the likelihood of epilepsy is greatly increased by increasing length of unconsciousness, in open head injury, the period of unconsciousness is not a good guide to the likelihood of post-traumatic epilepsy. Many patients with penetrating wounds of the brain have no loss of consciousness whatsoever, and walk in with a gaping hole in the head. These individuals have a very real risk of post-traumatic epilepsy by reason of the location of the wound and the fact that the brain has been injured.

I would like to say something about the overall course of patients who do develop post-traumatic

epilepsy. The fact that an individual has a seizure after a head injury does not mean that he is going to have seizures for the rest of his life. Quite a few patients will have seizures only in the first week or two after head injury and may have no more attacks; even those individuals who have seizures six months or year after the injury have quite a good chance of epilepsy ceasing. In fact, if we look at a large group of patients who have post-traumatic epilepsy and follow them up over a period of 10 years, we find that there is a large number that have seizures in the first year or so. As time goes on the likelihood of a major seizure decreases, so that after about 10 years it is only about 10%. The likelihood of a focal attack occurring remains at a somewhat higher level, perhaps around 35%. But of this entire group, all of whom had an attack at one time or another, there is a larger number who have had no attack for 2, 3, 4, 5 and 10 years. In fact, approximately one-half of the patients after a period of 5 years will be free of attacks irrespective of whether they are or are not taking anticonvulsant medication. The other point of interest is that there is a greater tendency for the major attacks to cease than the focal attacks. However, the focal attacks usually do not render the individuals incapable of taking care of themselves. I have patients now followed up for 25 years, who occasionally have a little twitching of the thumb which they recognise as a manifestation of an epilepsy, which in the early stages of their head-wound would have spread to their arm and face and perhaps become generalised with loss of consciousness. But the only manifestation now is this little twitching of the thumb. Other patients may have episodes of numbness of the fingers which were the initial manifestations of their generalised post-traumatic epilepsy years ago. This fact is of considerable importance to the injured patient who is very worried about what he maybe able to do later in life. As a matter of fact, the disability is not so much related to the occasional seizures as it is to two other factors. One is native intelligence. Those individuals with high IQ are able to go back to work and function quite well even though they have some attacks. Second the neurological deficit—an individual who has hemiplegia, aphasia or hemianopsia is somewhat incapacitated in his work. But the attacks themselves are very rarely the cause of the individual not being able to work after the initial period of 4 or 5 years.

I would like to comment briefly upon the treatment and prophylaxis of these attacks. We ordinarily give phenobarbital and dilantin once a person has had an attack with the idea that we are going to prevent future attacks. The hypothesis is that this will relieve the attacks in about 65% of cases. It has been suggested that if one gave phenobarbital immediately after the head injury one could decrease the likelihood of attacks occurring. This is based upon several premises. One is that the anticonvulsant medication would pretreat the epilepsy and cut down the likelihood of attacks by 65%. The second is by giving the anticonvulsant medication one can prevent the development of an epileptogenic focus i.e. the firing area in the brain which generates the neuronal activity that spreads to give rise to the fit. We know that foci take a period of time to develop somewhere between 3 and 12 months so that if one gave phenobarbital or dilantin one might prevent the maturation of the focus. In the third place by giving medication one might prevent the likelihood of a secondary focus developing. If we look at the electroencephalograms of patients who have head injury, we find, in a fairly high percentage of patients, spiking at the site of injury in the first few months after the injury. This, usually gradually decreases and within six months most of the spiking has subsided whether or not the patient develops seizures. It is only a manifestation of damage to the brain. This means that in a certain number of patients who have post-traumatic cortical spiking, by some means the neurones are able to inhibit the development of the focus.

Now let us see how one might apply this knowledge to the prophylactic therapy of post-traumatic epilepsy. If one wishes to pretreat the epilepsy, one should give anticonvulsant medication in therapeutic doses, so that the level of phenobarbital or dilantin in the blood is the same as that in patients who are being treated for an established epilepsy. Hence the head injured person should be given 60 mg of phenobarbital once or twice a day or 100 mg. hydantion two or three times a day. Then the incidence curve should be lowered to about 1/3 of that otherwise. We would then have a curve for the likelihood of seizures following open head injuries beginning about 5-10% and over a period of a couple of years coming down to about 2%. To determine the duration of medication one might ask at what level are we willing to accept the likelihood of seizures, 2.1 or 0.1%? The cut off

oint for this medication would then have to be determined. If you are going to accept a 2% risk, you can stop your medication at 2 years and the chances are only 2% of the individuals will develop subsequent seizures. If you want 0.1% you have to continue the medication for at least, 5 years, may be 10 years. On the other hand, in the case of closed head injury in which, if seizures are going to occur, they do so within the first month itself, prophylactic anticonvulsant medication would only have to be given for 3 months. In Czechoslovakia, Austria and the United States of America, anticonvulsant medication is being administered to head injured patients to test this hypothesis. There is an obvious disadvantage in that therapeutic doses may have certain undesirable side effects such as drowsiness. However, if the medication is given with the idea of preventing the development of a brain focus, it may be prescribed in a much smaller dose, because the evidence from experimental animals is that the spiking of a focus may be lessened by a very small amount of anticpileptic medication. Hence instead of giving phenobarbital 60 mg probably 15 mg twice a day might be adequate or 30 mg at night. Over a period of time one could probably maintain a level sufficient to inhibit the development of an epileptogenic focus. If the therapy is based on the second premise, the medication should be continued for the time it takes for a focus to develop which would be somewhere about six months to 2 years. Neurosurgeons for a long time have given their high risk cases anticonvulsant medication. Those are the cases with wounds of the central region that have a 50% chance of developing a seizure. But in the past, we did not apply this hypothesis to closed head injuries or in a systematic way to open head injuries.

Following the lecture, Dr. Walker answered questions from members of the audience :

Gp. Capt. Bajwa : Did you come across any type of psychomotor epilepsy following head trauma?

Dr. Walker : Immediately after head injury, psychomotor epilepsy is quite uncommon. It is generally due to damage to the medial temporal structures which, as I mentioned, are not commonly involved by the primary injury. They tend to be involved somewhat later as a result of oedema, intracranial hypertension and so forth. Hence, the incidence of psychomotor seizures following head injury is not very high. It is somewhere in the vicinity of 16%.

Gp. Capt. Murty : How to exclude that the injury itself is not due to the first attack of epilepsy ?

Dr. Walker : The problem is one that has been debated a great deal, viz , that when an individual has a seizure, and falls down, is the injury which may be sustained to the brain as a result, responsible for continuing seizures, particularly in young children. In individuals, for example who have febrile fits, it has been said that the damage to the brain which results from the engorgement of the brain during the generalised seizure is responsible for small areas of softening within the medial temporal structures, because these are pushed into the incisura as the result of a transtentorial herniation. I do not think we have a satisfactory answer to this problem at the present time. There is no doubt that patients who have generalised seizures do have an engorgement of the intracranial structures and that there may be, if the tentorial notch is fairly wide, a transtentorial herniation with damage to the medial temporal structures. Some time later, and it may take 10 or 15 years for these epileptongenic lesions to mature, seizures may occur. The period of maturation of the foci in the temporal lobes is considerably longer than that in the central region, that do so within a period of about six months to an year. In monkeys, for example, when we put alumina cream in the medial temporal structures, it takes 6 to 9 months for electroencephalographic changes to develop and even longer for clinical manifestations. In the same monkeys, alumina cream in the central regions will produce fits within six weeks.

Lt. Col. Suri : There are certain problems which again crop up in my mind. For example in a closed head injury, the maximum risk is within the first month. After that the chances of seizures are hardly any. So, does that mean that if in a pilot after a month we find no change in the electroencephalogram, we can allow him to fly ? Or if we find only mild slowing, say in the theta range between 5–7 Hz and knowing that he had a closed head injury the chances of his getting seizures are hardly any especially if we do not find any other neurological or psychiatric deficits, can we then allow him to fly ?

Dr. Walker: I think it depends upon what degree of risk you are willing to accept. There is a slight chance that seizures might develop after a closed head injury. We in the United States have taken a view that pilots should be practically perfect and if an individual had a closed head injury with loss of consciousness for a period of time, that individual should be grounded automatically for three months. He is examined initially and at the end of three months electroencephalographically and clinically. If he has no clinical and no electroencephalographic abnormality after three months, he would be re-examined after six months at which time if he still has these abnormalities, he will be examined at nine or twelve months. So, he will have a period of approximately six months to an year or more before he can go back to flying duty. If in that six months his clinical status, is perfectly normal, and, if the electroencephalograms are normal then we would accept the slight risk of post-traumatic epilepsy, which probably is not more than one chance in 200, that over a period of years he might have a seizure

Lt. Col. Suri: How long do you take an EEG record in a pilot? Do you take a sleep record and attach importance to any asymmetry found in it?

Dr. Walker: The answer to the first question varies a little bit from center to center depending upon the electroencephalographer. But in general, the electroencephalographic records made in the United States are approximately for 30 mintues using the standard montages. We routinely use sleep and we routinely use Hyperventilation and Photicstimulation in these cases. Now, as to asymmetry in sleep. I am not quite sure what the general feeling of electroencephalographers is, in the United States. My personal impression is that, if there is only an asymmetry that is not particularly significant.

Lt. Col. Suri: There is one specific problem that we face. There are some pilots with us for whom we do not have a baseline EEG. When they go from subsonic to supersonic aircraft, an EEG is done and an abnormality is then found. Now my question is while we should not let them go into supersonic aircraft, can we allow them to continue flying as they have been doing before?

Dr. Walker: This is centainly a very difficult question to answer. Their performance previously might have been excellent, and yet they may have abnormalities in their EEG. Again I think this is a question of what risk you are willing to accept. It has been found in a number of studies, perhaps the best one carried out so far is by Sem Jacobsen in Norway for the American Air Force, that individuals who have an abnormal electroencephalogram, when they are in a plane and pull out of a rapid dive, tend to black out. They do so much more frequently than individuals who have a perfectly normal record. So, if you are willing to accept a certain risk these individuals might be allowed to fly their planes. I would say that an individual who has an abnormal EEG should not pilot a commercial flight. I think the added risk to the passengers on the flight is greater than when their records are perfectly normal.

Lt. Col. Suri: I think in 1970 you told me that even a single well-documented epileptic seizure is a ban for flying for life and irrespective of the cause, e.g. somebody had a bout of alcohol or was at a high altitude when he had a fit. Does it still hold good?

Dr Walker: I would concur. I think the person who is flying a plane that not only costs perhaps half a million dollars or more or a few million rupees, but also has the lives of other people in his care, should not be permitted to fly if he has had a seizure. Although the chances are very slight that he will have another seizure, there is a risk and I do not think that it should be accepted.

Dr. Mani: The time of epilepsy after a closed head injury has been variously termed as immediate, early or late. How immediate is immediate, how early is early and what is late? What are the cut-off points?

Dr. Walker: The immediate ones are those which occur within a matter of few seconds or few minutes of the injury. The early attacks are variously defined as those which occur within the first week or the first month of the head injury and it depends upon the author where he puts that cut-off point. In the case of closed head injury I think you probably could use a cut-off point at approximately one month, because you will have all the high risk cases within that period of time. In the case of open head injury it is not so clear as to where the cut-off point should be. For some people, the cut-off point is at three months. There

are some neurologists who say that the individual who has a seizure within that first three-month period does not have post-traumatic epilepsy. They base this upon the fact that such individuals who have an early seizure have only a slight chance of developing an attack later and that these attacks are due to the effects of trauma, lacerations, contusions, haemorrhage and oedema of the brain and once these have been repaired (generally within a month or so), these factors are no longer operative. They also contend that if the individual develops an attack later, it is not related to the first one, but is an independent post-traumatic epilepsy. I think this is a matter of semantics and whether you draw the line at a week, a month or later makes little difference. The fact remains that an individual who has an attack in this first period has about a 50% chance of having no more attacks but if you are a pessimist he has a 50% chance of having a fit.

Dr. Mani : Arising out of this, in closed head wounds, you have mentioned the period of unconsciousness as one of the criteria for determining the severity of the head injury. What would be the period of unconsciousness which you would—require five minutes, half an hour, before thinking of prophylactic anticonvulsant medication ?

Dr. Walker : The period of unconsciousness would be one hour. I think an individual who has only a period of 2, 3 or 5 minutes of unconsciousness has a very mild head injury and if he does not have an attack immediately you might almost say before you see him, the chances are that he won't. If the individual is unconscious for more than an hour, he has a much higher risk of epilepsy.

Dr. Mani : That means that in the less risk group you would not consider prophylactic anticonvulsant drugs therapy. Am I right ?

Dr. Walker : No, I would weigh the disadvantages, viz., the side effects of anti-convulsant medication, the cost of the drug and number of other things that may make taking medicines painful to the patient, against the risk of epilepsy. It would turn out that those individuals with a short period of unconsciousness would be given no medication.

Dr. Mani : What about post-traumatic amnesia ? Would that come in determining the severity of head injury ?

Dr. Walker : Post-traumatic amnesia ordinarily corresponds to the period of unconsciousness. Some people use post-traumatic amnesia as a criterion because they say they can measure that better than the period of unconsciousness. But the two are very closely related.

Dr. Mani : If I understood correctly when you say unconsciousness, you tend to include post-traumatic amnesia also in it.

Dr. Walker : No, I would not include the period of post-traumatic amnesia as a part of the time of unconsciousness.

Dr. Mani : What about depressed fracture of the skull with or without dural tear ?

Dr. Walker : The presence of any complications of a head injury increases the likelihood of seizures. Skull fractures are one of those. How much it increases depends upon a number of factors. One is the depth of depression. There are cases in which the depression is only a few mm. I doubt whether that has any significance. But there are cases where the depression is greater or close to a vessel, so that there is an extra-dural collection of blood and here the likelihood of brain damage is increased and also of seizures. But, in general, if there is a depressed skull fracture, you have to add about 10% to the ordinary risk of post-traumatic epilepsy.

Dr. Mani : Similarly, any neurological deficit would also increase the risk rate ?

Dr. Walker : That is right. One of the factors is the severity of injury.

Dr. Mani : Even for a closed head injury, not necessarily open.

Dr. Walker : Both closed and open.

Dr. Mani : One of our problems is that most of our patients are highly irregular in taking their drugs, even when they have seizures. If we, are going to give one of these drugs as a prophylactic medication, I very much suspect that

they would be at least as irregular, if not more so. In such instances, what are the risks of their getting seizures because of irregularity in medication?

Dr. Walker: Let me make a statement first and then I will come back to answer your question. It is common knowledge, that when you are treating pharmacologically a person with epilepsy, if you suddenly stop that medication, there is a chance that within the next few days, he will have a seizure. Perhaps this risk is greater than before he started taking medication. The question which you are posing now is, if you give prophylactic medication and you assume that you are suppressing attacks and then the patient stops the medication, will you have the withdrawal effect? Well, this depends upon a number of factors. One, is the type of medication which you are giving. The half-life of some of the anticonvulsant medications such as phenobarbital is very long so that if a dose or two, is missed it does not matter. Only after ten days with phenobarbital, will the drug be washed out completely. An effective anti-convulsant dose will be maintained for five or six days so that such individuals probably will not have any ill effects if they just miss an occasional dose. On the other hand, if you are giving diphynyl-hydantoin which has a considerably shorter half-life and the medication is stopped the drugs would be washed out quickly and the blood concentration fall below the effective anti-convulsant level so that there would be a greater danger of epilepsy developing. This is a possibility but at the moment we do not know how great the risk is.

Dr. Mani: Regarding EEG, what type of abnormality are we talking – abnormalities in the background or paroxysmal discharges?

Dr. Walker: I would say of both background abnormalities and the paroxysmal discharges.

Dr. Mani: When you say pilot should not show any abnormality, you mean it must be a completely normal EEG?

Dr. Walker: Yes, what we would call as a normal EEG?