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UNION CARBIDE CORPORATION OLD RIDGEBURY ROAD, DANBURY, CT 06817

To (Name)	Medical Directors	Date	August 8, 1983
Division	Contract Physicians		
Location		Originating Dept.	Corporate Medical/HS&EA
Area			
		Area	Danbury, P2594
Copy to		Subject	Asbestos Related Diseases

PLAINTIFF'S EXHIBIT

UC-3619

Because asbestos related diseases both with and without asbestosis are a cause of considerable concern and confusion, I have attached an execellent article written by D. Davies that appeared in the July 16, 1983 issue of the BRITISH MEDICAL JOURNAL. Davies has distilled a great deal of information into a succinct description of when compensability may be indicated. His comments on lung cancer are especially interesting.

The fact that only a small percentage of pleural plaques (15% reported by Hourihane) are detectable during a lifetime by usual chest radiographs gives one concern. If our chest x-ray examinations are of poor quality and are poorly interpreted, we could be accused of exercising inadequate "reasonable" care in our detection efforts. Hilton has been working with you to try to upgrade our chest x-ray examinations and interpretations.

I appreciate that Dr. Hilton Lewinsohn called this article to my attention. Hilton is our resident expert on asbestosis and asbestos related diseases so if you have any questions or problems give him a call on extension 8-421-5214.

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enclosure

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value and importance? Once the questions are identified it may be easier to choose the forum in which to answer them.

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Deputy Editor,
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[3] British Medical Association. *The medical effects of nuclear war. The report of the British Medical Association's Board of Science and Education.* Chichester: John Wiley and Sons, 1983.

Asbestos related diseases without asbestosis

Asbestosis and mesothelioma are prescribed diseases under the Industrial Injuries Scheme and entitle the patient or his dependants to compensation. The term asbestosis should be used only to denote fibrosis within the lungs. The three other non-malignant conditions associated with exposure to asbestos are pleural plaque, pleural effusion, and diffuse pleural thickening. The Industrial Injuries Council now recommends that, when it reaches a certain extent, the last of these should also be prescribed.[1]

Pleural plaques develop in the parietal pleura, seldom cause disability, and are not to be prescribed. Pleural effusions may be transient but some are chronic and may recur.[2] No prescription is proposed for these, but, under the new proposals, if they cause sufficient bilateral thickening of the pleura an application to a pneumoconiosis medical panel would lead to certification.

Diffuse pleural thickening associated with exposure to asbestos has become increasingly recognised.[3 4] Both pleural layers are affected and restrict the expansion of the lung. Histological examination shows only non-specific fibrosis, and, as with effusions, the diagnosis depends on excluding other causes such as tuberculosis and other infections, collagen diseases, trauma, chronic uraemia, and drug induced fibrosis.[5] With such exclusions, the Industrial Injuries Council is satisfied that bilateral diffuse pleural thickening in asbestos workers is likely to be industrial and recommends that disablement benefit should be awarded when the thickening is bilateral, is over 5 mm thick, and extends over more than a quarter of the chest wall.

The prescription of this condition will be welcomed, though some may be dissatisfied that the disease has to be so extensive to allow certification. Nevertheless, the committee suggests that this requirement should be reviewed in the light of experience.

The report also considers cancer of the lung in asbestos workers. At present this condition is not prescribed, but when it occurs in association with asbestosis it is considered to be a sequel and benefit is payable. Claims which have been rejected during life may be successful after death because necropsy may disclose previously undiagnosable mild asbestosis. The council now recommends prescription of lung cancer in patients with asbestosis and in certain other circumstances. In doing so it had to consider how to take account of smoking habits and the amount of exposure which would justify certification. The problem of the contribution of smoking to lung cancer in asbestos workers has been disposed of neatly. Studies in the United States in insulation workers have shown that the relative risks of developing lung cancer are: non-exposed non-smokers 1; asbestos exposed non-smokers 5; non-exposed smokers 11; asbestos exposed smokers 53.[6] Because exposure increases the risk by a factor of five in both smokers and non-smokers the council recommends that smoking should be disregarded.

Cancer of the lung in asbestos workers may be divided into two broad types. Firstly, peripheral adenocarcinoma, which is unrelated to smoking and accepted as being due to asbestos exposure. The second is squamous, undifferentiated, and small-cell cancer arising in proximal airways, certainly related to smoking but only doubtfully related to asbestos. Perhaps wisely, the council makes no attempt to distinguish between these and recommends prescription irrespective of type. Most authorities accept that the risk of lung cancer is increased only when there has been heavy exposure to dust. Reasonable estimates of exposure may be made for people working in circumscribed places such as asbestos factories, but this may be impossible for, say, shipyard workers or engineers with intermittent exposure in various jobs, particularly in the distant past.

Faced by this difficulty the council has made an ingenious proposal that the presence of other indicators of asbestos exposure should justify certification. It recommends prescription when primary cancer of the lung is accompanied by one or more of the following features: asbestosis, bilateral diffuse pleural thickening, and bilateral pleural plaques.

Asbestosis provides clear evidence of heavy exposure, but, though bilateral diffuse pleural thickening and bilateral pleural plaques are linked with exposure to asbestos, the relation is less definite and more capricious.[7] Moreover, pleural disease and asbestosis are also poorly correlated, for the former may appear in lightly exposed people such as the wives of asbestos workers and residents in the vicinity of asbestos mines and factories. Given a minimum exposure to dust, the development of pleural calcification, which facilitates the diagnosis of plaques, is probably mainly determined by age, usually taking over 20 years from the first exposure.[8 9] Only some 15% of plaques are detectable during life,[10] but this proportion might be higher if oblique *x* ray views were used in addition to the routine ones.[11] Hence probably the people who are certified during life will not be all those who have plaques but only the minority in whom they are demonstrable. The case for necropsies in people who have had industrial exposure to asbestos and die of lung cancer is clear.

The council's aim has been to identify people with sufficient exposure to dust to justify certification if they develop lung cancer. Workable regulations are difficult to devise, but the proposals favour those with pleural *x* ray shadows which may be the result of light exposure in the distant past and discriminate against those with much heavier exposure who, for unknown reasons, do not develop pleural lesions or have not had time to do so. The council has decided against specifying a duration and degree of exposure as a qualifying condition because it considers this to be arbitrary and lacking in scientific validity. Even so, the criteria of pleural lesions that it proposes are probably a less than satisfactory index and, though the extension of prescription will be welcomed—even if it requires legislation—probably many will find that the details of the proposals which are now before the Secretary of State for Health are unsatisfactory.

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Union Carbide trial exhibit UC-3619: Thomas A. Lincoln to Medical Directors and Contract Physicians on Asbestos... Published by AsbestosClaims.Law
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Difficulties with knees

Only a few years ago internal derangement of the knee was an accepted diagnosis which led to arthrotomy on the painful side of the joint with, almost inevitably, the excision of the cartilage: "better to remove a normal meniscus than miss the torn posterior horn." Those days have passed; the meniscus is now preserved at all costs, and in some centres it may even be repaired. One of the main reasons for this change is that successive reviews of the late results of meniscectomy have shown that the operation is far from being benign—and that it is certainly not the cure for all problems of the knees. Meniscectomy may, indeed, increase the problems in a knee which is unstable from damage to the ligaments.

For the knee still remains an enigma, despite a wealth of new tests and diagnostic aids such as arthroscopy and arthrography. Among the most taxing of the problems within the knee are those of rupture of the cruciate ligaments. Rupture of the posterior cruciate ligament is the easier to manage. On examination the tibia is found to hang back when the knee is flexed to 90° with the foot on the examination couch. The anterior draw sign is positive—the tibia can be drawn forward when the leg is in the position described—but in reality it is moved to the "normal" position only relative to the femoral condyles. Radiographs may show an avulsed fragment from the posterior aspect of the tibia. Treatment is conservative or operative. The fragment may be screwed back into position, or the ligament may be repaired directly or replaced by nearby tendons or muscles. This injury is frequently associated with major disruptions of the knee in which serious neurovascular injuries may occur requiring urgent attention. Longstanding injuries require careful assessment before surgery, which should aim at controlling any straight or rotatory instability. The exact nature of the surgery will depend on the instability and is likely to be complicated. Surgery may not be needed, however; where possible the management should consist of quadriceps and hamstring exercises to control the knee, with advice to the patient to reduce his demands on the joint. Dandy and Pusey[1] found that in a group of 20 patients treated by this conservative approach the functional results bore no relation to the degree of laxity of the ligament—and 18 of the patients eventually decided that their symptoms were not severe enough to justify surgical reconstruction.

The greater problem is the anterior cruciate. After years of discussion authorities still cannot agree. This difference of opinion was highlighted again in the *Journal of Bone and Joint Surgery* in February of this year. Hughston and Barrett reported that rupture of the anterior cruciate did not in itself contribute to instability of the knee.[2] They believe that the instability which occurs in knees with this lesion is related to damage to the capsule, other ligaments and muscles, and the medial meniscus. In their hands careful evaluation and repair of the damaged structures produced knees stable enough to withstand vigorous sport, whether or not the anterior cruciate was ruptured. They emphasised the prime importance of the medial meniscus as a stabilising structure.

By contrast, Noyes *et al* described the disability in 103 patients with chronic laxity of the anterior cruciate ligament uncomplicated by other major deficiencies or previous reconstructive procedures.[3] They showed that damage to this ligament was detected in only seven of the 103 patients initially, including those examined by orthopaedic surgeons. They found that if untreated an isolated anterior cruciate lesion would lead initially to a lower sporting achievement and later to reinjury, damage to the meniscus, and eventually to osteoarthrosis.

The average orthopaedic surgeon finds such a wide divergence of opinion difficult to understand. He should have found comfort in the sensible editorial in the same journal, though the author, Robert Larson, could not reconcile the differences.[4] He did, however, attempt to give some guidelines on management based on his belief that all the structures play a part in the stability of the joint; none is all important, all depend on each other. Thus to re-establish stability all damaged structures should be identified, repaired, or augmented. Larson argues that repair should be undertaken urgently after injury, but acknowledges that this approach requires accurate and early diagnosis. The anterior cruciate itself, he believes, should not be repaired but augmented, for experience has shown that repair is difficult and produces poor results. He thinks that repair of the knee with chronic injuries should be approached with caution, for judicious advice to the patient may obviate surgery, especially if he is taught to develop his hamstrings and quadriceps and lower his athletic requirements. In those patients whose knees are so unstable as to interfere with normal life—or those who want to engage in sporting activities at a high standard—the joint should be carefully assessed and the appropriate repairs and augmentation undertaken.

In the light of all this conflicting evidence, what is the key? Perhaps the answer is a superspecialist service for the few patients who are crippled by the damage to their knee, or perhaps a better diagnostic service for the acutely damaged knee, or perhaps both.

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